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

the midline then he can be referred for the more expensive CAT scan.

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Aetiology of appendicitis

SIR,—We read with interest the repeated assertion by Mr D P Burkitt and others that "the geographical distribution of appendicitis indicates that it is a disease associated with modern Western culture" (3 March, p 620).

Reports, which often comprised moderately large series of cases of appendicitis, have recently been published from Africa,¹⁻³ South America,⁴ the Middle East,⁵⁻⁷ Malaysia,⁸ Indonesia,⁹ China,¹⁰ and even the Arctic.¹¹ The World Organisation of Gastroenterology research committee is conducting a large-scale multinational survey into the presentation of abdominal pain around the world. In every single centre, including Mexico City and Khon Kaen, Thailand, in large series of several hundred cases per centre, the commonest cause of admission to hospital with acute abdominal pain is acute appendicitis.

It is not helpful therefore at the present time to equate appendicitis with modern Western culture. The relation between the two—if it exists at all—is undoubtedly far more complex than Mr Burkitt's simplistic assertion.

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¹ Bonino, C A, *et al*, *Chirurgia Italiana*, 1977, **27**, 594.
² Omigbo, W L, *South African Journal of Surgery*, 1977, **15**, 67.

³ Watson, W C, *Journal of Tropical Medicine and Hygiene*, 1968, **71**, 229.

⁴ Dolz, A, *Revista Chilena de Pediatría*, 1976, **47**, 121.

⁵ Keeley, E P, *Journal of the American Medical Association*, 1968, **206**, 647.

⁶ Osman, A A, *International Surgery*, 1974, **59**, 218.

⁷ Shamoun, S E, and Al-Khaddar, M A, *Journal of the Royal College of Surgeons of Edinburgh*, 1978, **23**, 369.

⁸ Sall, H B D, *Medical Journal of Malaysia*, 1972, **27**, 43-4.

⁹ Pancla, H O, *Tropical and Geographical Medicine*, 1975, **27**, 354.

¹⁰ *Chinese Medical Journal*, 1977, **3**, 266.

¹¹ Lifliandskii, D B, *Sovetskaya Meditsina*, 1967, **30**, 134.

SIR,—May I please be allowed to comment on the letter by Mr D P Burkitt and others (3 March, p 620)? In my work *The Saccharine Disease*,¹ helped especially by careful graphs drawn by the late Mr A Elliot Smith and Dr Walter Yellowlees, I have shown that the rising incidence of appendicitis can be closely related to the enormous rise in consumption of refined carbohydrates and especially of refined sugar since 1815 (about 7 kg of sugar per head per year then against over 45 kg per head per year now). I have also shown that this great rise in consumption is accompanied by much intestinal fermentation, which basically accounts for the evil-smelling motions so frequently occurring in civilised man today, and frequently effecting tumour formation in the colon. (Motions from natural foods, which never contain this amount of sugar, in nature, are never evil smelling.)

I am not dreaming of suggesting that the presence of faecoliths is not of crucial importance in the aetiology of appendicitis and I have discussed this matter closely with Mr Maurice Frohn himself, who knows far more of this

subject than I could ever hope to do, but I submit that right at the bottom of this important evidence there does lie consumption of refined sugar as a causative factor, as my book tries to show.

For 40 years now I have been collecting natural history specimens that bear directly on this subject, which I would gladly show to any reader interested in the matter.

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¹ Cleave, T L, *The Saccharine Disease*. Bristol, Wright, 1974.

Trends in duodenal ulcer

SIR,—Surgeon-Captain Cleave's theory about the relation between over-refined foodstuffs and diseases of the large bowel and even coronary artery disease¹ is gradually becoming known and more gradually accepted. But for some reason his even more thoroughly worked-out theory of the causation of peptic ulcer² seems to be completely ignored. Your leading article on duodenal ulcer (10 March, p 641) makes no mention of it and speaks of the late Victorian epidemic of gastric ulcer in young women as though it were beyond explanation. Cleave's theory explains very neatly the changing incidence of peptic ulceration. Explanation, of course, is not proof; but in the absence of any other plausible theory it is rational to act on the best we have, at least until it has been disproved.

In brief, what Cleave says is that anything which tends to prevent the buffering of gastric acid while not delaying gastric emptying predisposes to duodenal ulcer. Anything which excessively delays gastric emptying, especially when the contents are inadequately buffered, predisposes to gastric ulcer. The principle cause of inadequate buffering is the consumption of refined carbohydrate unaccompanied by protein. This is a twentieth-century custom and has led to a twentieth-century disease. Causes of delayed stomach emptying include inadequate mastication (as a result of an absence of teeth, for example), consumption of indigestible or fried food, pyloric stenosis, and excessively tight clothing. The Victorian gastric ulcers, in Cleave's view, are attributable to the fashion for tightly laced corsets.

Thus the theory, which Cleave supported with wide-ranging epidemiological evidence, encompasses not just diet but the way in which foodstuffs are refined, combined, and consumed.

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¹ Cleave, T L, *The Saccharine Disease*. John Wright, Bristol, 1974.

² Cleave, T L, *Peptic Ulcer*. John Wright, Bristol, 1962.

Sugar and diabetes mellitus

SIR,—Professor Harry Keen and his colleagues (10 March, p 655) say that the evidence for the suggestion that dietary sugar (sucrose) is a cause of diabetes mellitus "is largely circumstantial." If this means that the evidence, like that in the work they report in their own paper, is based largely on epidemiology, then they are wrong; for they entirely ignore the very large amount of evidence from experiments both in laboratory animals and in human subjects. Some of the features of diabetes that are produced by diets that

contain sucrose are: impaired glucose tolerance,¹ abnormal insulin response to a glucose load,² insulin resistance of the tissues,^{3,4} increased blood concentration of triglyceride,² retinopathy,⁵ and nephropathy.⁶

In regard to nephropathy, Professor Keen and his collaborator Dr J J Jarrett in the book *Complications of Diabetes*,⁷ which they edited, have many pages dealing with the importance of the changes in the glomerular basement membrane (GBM) in diabetes; we have recently shown that rats fed sugar-containing diets develop precisely these changes (report to be published). Electron microscopy revealed the characteristic thickening of the GBM; biochemical analysis showed that the composition of the GBM, especially in relation to its content of amino-acids, amino-sugars, and the enzyme glucosyltransferase, was comparable to the composition of the GBM from rats with streptozotocin-induced diabetes. It seems to me that, circumstantial or not, the experimental evidence makes it difficult to absolve sugar from having a causative role in diabetes.

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¹ Cohen, A M, *et al*, *American Journal of Clinical Nutrition*, 1966, **19**, 59.

² Szanto, S, and Yudkin, J, *Postgraduate Medical Journal*, 1969, **45**, 602.

³ Vraná, A, *et al*, *Nutrition Reports International*, 1971, **3**, 31.

⁴ Bruckdorfer, K R, Kang, S S, and Yudkin, J, *Proceedings of the Nutrition Society*, 1974, **33**, 3A.

⁵ Papachristodoulou, D, Heath, H, and Kang, S S, *Diabetologia*, 1976, **12**, 367.

⁶ Kang, S S, *et al*, *Biochemical Society Transactions*, 1977, **5**, 235.

⁷ Keen, H, and Jarrett, J J (editors), *Complications of Diabetes*. London, Edward Arnold, 1975.

Alopecia areata

SIR,—I read with interest your leading article on alopecia areata (24 February, p 505). Since the report by Happle and Echternacht¹ I have been treating a group of patients with dinitrochlorobenzene (DNCB) sensitisation using the same protocol. Four patients with alopecia totalis, and four patients with patchy scalp loss have been treated for periods ranging from one to 15 months. In one case vellus hair developed; no patients grew normal terminal hair.

Although the immunological hypothesis is attractive I feel that DNCB is no more specific than previous therapies utilising irritants which caused non-specific inflammation.

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¹ Happle, R, and Echternacht, K, *Lancet*, 1977, **2**, 1002.

Minocycline-induced interstitial nephritis

SIR,—We wish to comment on the article by Dr R G Walker and others (24 February, p 524), which cites minocycline as being the causative agent in a case of acute interstitial nephritis.

It should first be pointed out that the dose of minocycline of 250 mg four times a day is five times that recommended in the UK. In addition, it is not uncommon for respiratory symptoms to be associated with glomerular nephritis independent of drug treatment. Secondly, all tetracycline analogues, without exception, possess antianabolic activity but