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 Lifitsandskii, 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