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 Pediatrica*, 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, *Sovetskaia 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 disease[1] is gradually becoming known and more gradually accepted. But for some reason his even more thoroughly worked-out theory of the causation of peptic ulcer[2] 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 Echternacht[1] 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

this is rarely of clinical significance except in malnourished or geriatric patients. Unlike earlier analogues, and owing to a mainly enterohepatic excretion, the plasma clearance of minocycline is largely independent of renal function. Recent papers[1 2] indicate that uraemia is not exacerbated if minocycline is used in cases of renal insufficiency.

Finally, we are not aware of anything in the published literature to support the theory that this case of interstitial nephritis had anything but a temporal association with minocycline.

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[1] Sklenar, I, *Agent Actions*, 1977, **7**, 369.
[2] Heaney, D, and Eknoyan, G, *Clinical Pharmacology and Therapeutics*, 1978, **24**, 233.

General medicine and visual side effects

SIR,—While I have admired Mr P A Gardiner's attempts to present a simple and lucid account of basic ophthalmology for non-specialist practitioners, I feel I must write to question his remarks on iatrogenic disorders (17 February, p 461).

The drug of choice for pupillary dilatation for ophthalmoscopy is tropicamide (Mydriacyl) 0·5% on account of its rapid onset, producing a mydriasis of short duration with minimal effect on accommodation in this strength.[1] However, if cyclopentolate (Mydrilate) is chosen for pupillary mydriasis for ophthalmoscopy the 0·1% solution should be used, not the 0·5% solution, which has a prolonged and undoubted effect on accommodation. It has, moreover, been shown that attempts at reversing with pilocarpine the mydriasis produced by parasympatholytic agents, such as cyclopentolate, is not effective.[2] It should be added that the use of a drug such as cyclopentolate rather than atropine is much more likely to cause central nervous system disturbances in young children.[3-5]

I think Mr Gardiner should have clarified the difference in systemic drug effects in patients with chronic simple glaucoma and closed-angle glaucoma. It must be emphasised that patients in whom the diagnosis of chronic simple glaucoma has been made and who are under treatment are not at risk with the drugs he suggests, for the mild anticholinergic effect of such drugs does not jeopardise the control of intraocular pressure established with anti-glaucoma agents.[6] As regards narrow-angle glaucoma, patients in whom a peripheral iridectomy has been performed or who are using pilocarpine to prevent angle closure developing are at minimal risk, and it is only in the patients in whom a diagnosis has not been made (or who have not been treated) that the systemic drugs may produce pupillary dilatation and thus close the angle. The drugs mentioned are not contraindicated in patients having treatment for closed-angle or chronic simple glaucoma and no glaucoma patient should be denied appropriate systemic therapy with the drugs listed.[7]

Probably the most important point of all is the statement that "the clinical evidence that long-term treatment with systemic steroids causes cataracts is tenuous." Although there has been contention, the weight of evidence of many studies has shown that steroid-induced cataracts are directly related to the dosage and duration of treatment,[8 10] and only a maintenance dose of 7·5 to 10 mg of prednisone (or equivalent dosage of other steroid preparation) is safe and will not lead to posterior subcapsular lenticular opacities. Indeed, once these opacities have developed they will not regress and may well progress despite withdrawal of systemic corticosteroids; so his suggestion of ophthalmological surveillance when such opacities develop is, of course, only observational and can in no way effect the course of events.

The statement that chloroquine and similar drugs used for malaria are seldom used long enough for visual problems to arise is generally true but this drug is a cumulative toxin and there have been reports of airline pilots developing problems after having used chloroquine over a prolonged period in prophylactic therapy. I myself just six weeks ago have seen a West African who has typical chloroquine retinopathy, with resultant gross field loss, from using chloroquine in moderate dosage for short periods intermittently over a period of 20 years to control acute attacks of malaria.

Ethambutol produces visual disturbance not by toxic effects on the retina but by an optic neuritis (toxic optic neuropathy). This must be emphasised, for the visual loss has an acute onset and the drug should be withdrawn immediately.[11-13]

Without wishing to prolong my comments unduly I would finally like to remark that in the appendix some doubt must be cast on the effects attributed to various drugs listed. I would hope that readers of this part of the article would refer to established texts on ocular toxicology before accepting these observations as established dogma.

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[1] Davidson, S I, *Transactions of the Ophthalmological Societies of the United Kingdom*, 1976, **96**, 327.
[2] Anastasi, L M, Ogle, K N, and Kearns, T P, *Archives of Ophthalmology*, 1968, **79**, 710.
[3] Praeger, D L, and Miller, S N, *American Journal of Ophthalmology*, 1964, **58**, 1060.
[4] Binkhorst, R D, *et al*, *American Journal of Ophthalmology*, 1963, **55**, 1243.
[5] Wang, M K, and Tatane, J R, *British Medical Journal*, 1974, **1**, 453.
[6] Davidson, S I, in *Recent Advances in Ophthalmology*, ed P D Trevor-Roper, p 278. Edinburgh, Churchill Livingstone, 1975.
[7] *Drug and Therapeutics Bulletin*, 1975, **13**, 7.
[8] Crews, S J, *British Medical Journal*, 1963, **1**, 1644.
[9] Williamson, J, *et al*, *British Journal of Ophthalmology*, 1969, **53**, 361.
[10] Spaeth, G L, and von Sallmann, L, *International Ophthalmological Clinics*, 1966, **6**, 915.
[11] Carr, R E, and Henkind, P L, *Archives of Ophthalmology*, 1962, **67**, 566.
[12] Leibold, J E, *Annals of the New York Academy of Science*, 1966, **135**, 904.
[13] Barron, G J, Tepper, L, and Iovine, G, *American Journal of Ophthalmology*, 1974, **77**, 256.

Chiropractors and the AMA

SIR,—The "closed-shop" attitude of the American Medical Association towards chiropractic is exposed in Barbara Culliton and Wallace Waterfall's article (17 February, p 467). The GMC, on the other hand, accepts that doctors may refer patients to non-medical chiropractors if they consider them to have the necessary skill, on condition that the referring doctor retains ultimate responsibility for the patient. This appears to be a far more reasonable attitude.

What purpose does it serve to attack chiropractors by misrepresenting the facts? Is chiropractic a cult? The founder, D D Palmer, used such terms as "innate intelligence" to describe the body's healing power; but he was simply stating that a therapist does not heal but merely stimulates the body's own healing mechanism. In no way was he founding a religion. It is of course true that there is a tendency for Americans once convinced of something to "sell" it with a quasi-religious fervour.

Is chiropractic unscientific? I am quite certain that I have a far better scientific explanation of the way in which mechanical derangement of the vertebral column causes symptoms and "adjustment" restores normal neuromuscular co-ordination than I have for the actions of many drugs, physical therapies, and even surgical procedures.

Is chiropractic a health hazard? It is in fact one of the safest forms of treatment, accidents being incredibly rare. If chiropractors delay the referral of patients for more appropriate treatment, such a situation can only be encouraged by physicians and surgeons who refuse to take referrals from chiropractors.

Medicare requires that a subluxation be demonstrable by *x* ray. Many are not as they consist of fixation of a joint within its normal range of movement. They can therefore only be diagnosed by motion palpation. Medicare's requirement encourages excessive use of radiographs.

In order to understand common pain syndromes and neurophysiological effects relating to disorders of muscles and joints, particularly of the spine, and to learn effective manipulative procedures to treat them, I found it necessary to go outside the medical profession to a chiropractic college. What a pity so few doctors have done this.

Let us hope that we in Britain will not follow the example of our American colleagues. In our relations with such a potentially useful body of people as the chiropractors surely co-operation is better than confrontation.

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Homoeopathic medicine

SIR,—As many doctors and patients are aware, there is at present a tremendous boom in the teaching and practice of all varieties of healing outside the conventional medical establishment. This has arisen from the mechanistic and specialised approach in much of modern medicine and the increasing concern of the public, and indeed of many doctors too, about the side effects, toxicity, and allergic reactions of many modern drugs. This has resulted in the setting up of various "health clinics" around the country and many lay unqualified persons advertising as consultant homoeopaths, acupuncturists, herbalists, etc.

Some of these do undoubtedly help patients, but the dangers of practitioners treating conditions which require surgery, replacement therapy, or expert advice are all too obvious to the trained physician, and bring into disrepute those qualified doctors who are trying to broaden their therapeutic skill by using homoeopathy along with orthodox medicine. There seems to be no way in which such practitioners can be prosecuted by law, and the only way the public can be safeguarded is for doctors and patients to be made aware of who is properly trained and who is not.

The only official homoeopathic medical body is the Faculty of Homoeopathy, registered by Act of Parliament and recognised