effective, but not all families are willing to tolerate the consequent nocturnal disruption. I disagree that children under 7 or 8 are indifferent to their wet beds; facts elicited by a more than superficial inquiry suggest otherwise.

To claim, as the authors do, that the use of these "dangerous drugs" for children under 8 "cannot be justified" is not correct. There is practically no danger with proper dosage, and proper dosage can easily be ensured if we prescribe unattractive tablets rather than tasty syrup.

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

Fats and atheroma

SIR,—May I reply to a number of comments which appeared in your correspondence columns following the publication of my contribution to the "Fats and atheroma" debate (17 March, p 732).

Dr D P St George (31 March, p 890) attributes to me the statement that "the decline in death rate from ischaemic heart disease in the USA, paralleled by changes in diet, at last gives us overwhelming evidence for a nutritional aetiology of ischaemic heart disease." I regard this phenomenon as only one of a substantial number of clues and this is made clear in my review. Sir John McMichael (p 890) once again criticises the published trials of primary prevention by dietary means. I pointed out that there were indeed a number of faults associated with each but that an impressive feature was that each pointed in the same beneficial direction. He now describes three further "negative" studies—one is published in a National Dairy Council publication, another in *Doctor*, and the third in a publication which thus far Blackwells have been unable to trace for me. Once these have been published in more accessible places it may be possible to evaluate them critically.

"Primary prevention trials of cholesterol reduction by clofibrate have failed to produce convincing evidence of benefit," claims McMichael. This may well be true if the benefit being sought was "community protection" against ischaemic heart disease. Probably the best conducted clinical trial of primary prevention to date, the WHO-sponsored clofibrate study,¹ provided convincing confirmation of the importance of cholesterol (or some other factor for which cholesterol is a fairly accurate marker in the aetiology of ischaemic heart disease). Cholesterol reduction was closely associated with a reduction in ischaemic events, the most striking changes being apparent in those at greatest risk (because of other risk factors or because of particularly high cholesterol levels). The fact that total mortality was not reduced in the overall analysis comparing clofibrate and control groups may raise questions concerning the precise clinical role of this drug, but with regard to the present debate the correlation between cholesterol reduction and reduction of ischaemic episodes is particularly important. (Final interpretation of the clinical relevance of this drug must await the publication of the analysis concerning benefits and risks in the high-risk group rather than the whole treated population, since it is among them that a beneficial effect is most likely to be seen.)

Sir John McMichael again offers the Blumgart report on induced myxoedema as "priority" evidence against the cholesterol hypothesis because after "many years of the induced hypercholesterolaemia there were no coronary occlusions." Can 1-13 years (in eight individuals) really be seriously regarded as a long period of time in the genesis of atheroma?

Dr H M Sinclair (7 April, p 952) implies that I

am actually recommending *trans* polyunsaturated fatty acids. I merely stated that the evidence suggesting that they were harmful was not strong—and indeed perhaps I might point out again that the dietary advice I offered would provide perhaps only 10% of energy from polyunsaturated fat, and consequently only a very small proportion of *trans* polyunsaturated fatty acids.

I agree entirely with Dr Sinclair, Professor M F Oliver (31 March, p 890), and Sir John McMichael that pursuit of Dr John Vane's approach (17 February, p 484) is a potentially exciting one. Certain fatty acids may well be shown to be precursors of antithrombotic substances. Nevertheless, until further information is available I continue to feel justified in making the recommendations I have offered and feel confident that no major recantation of faith will be necessary in the light of future discoveries.

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¹ Committee of Principal Investigators, *British Heart Journal*, 1978, 40, 1069.

Nutritional intake, adiposity, and diabetes

SIR,—May I be allowed, please, to comment on the letter by Dr D P Burkitt and the Revd H C Trowell (21 April, p 1083). Speaking of Africa in my book *The Saccharine Disease*,¹ I comment on the fatal effect of removing fibre from foods, especially from carbohydrate foods, as in those containing sugar, which I corrected in HMS *King George V* in the last war by bringing in raw unprocessed bran by the sackful straight from the mills, which was very popular with the ship's company—a project that was carefully recorded in the *BMJ*.²

In the same book I try to show that Dr G D Campbell and Dr E L Batchelor showed an almost unparalleled increase in diabetes in large numbers of the 400 000 Natal Indians in South Africa, working in the sugar plantations, who were also given a ration of refined sugar. This incidence of diabetes in these people is, still, I believe about the highest in the world, and obesity is also common in them. Dr Campbell also showed that the increase of coronary disease in the Natal Indians is almost as great as the incidence of diabetes, and indeed coronary disease in diabetes generally is well known to be so high that it is often the commonest cause of death in this disease.

More important than any medical opinion, I submit, is Nature herself, which is what my book seeks to establish, especially in its diet sheet, and I hope that the book will have a prophylactic effect in both diseases.

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

² Cleave, T L, *British Medical Journal*, 1941, 1, 461.

One man's schizophrenic illness

SIR,—I was much impressed by the article "One man's schizophrenic illness" by Peter Wescott (14 April, p 989). In particular, I value the opportunity such an article gives one to hear criticism of doctors by a long-suffering patient. Perhaps the excellent "If I had . . ." series could be followed by a series entitled "When I had . . ." written by real patients (preferably non-doctors) about their

own experiences within the NHS. It is not always comfortable to hear just criticism of oneself and one's fellows, but it is good.

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Another personal view of cardiac arrhythmia

SIR,—I always read with interest the articles that appear from time to time in the *British Medical Journal* under the heading of Personal View. In these articles some of our colleagues tell of their experience with illness in themselves. The article by Dr I W B Grant (14 April, p 1012) prompts me to write of my own experience with cardiac arrhythmia (extrasystoles). It may help to reassure him and his attitude to irregular heart beats.

I am a retired orthopaedic surgeon, now aged 77 years. I know precious little about cardiac disorders, and have never managed to read and understand electrocardiographic recordings. At the end of the war I noticed that after drinking coffee I started having irregular heart beats. I consulted a friend who was a cardiologist at a London hospital and had ECG No 1. I was told that I had an "irritable heart," with extrasystoles. He showed my wife the technique of carotid compression. We did this periodically, but she was afraid of killing me, so we stopped doing it when I had a bad attack of palpitations. However, I took a quinidine tablet, which stopped the extrasystoles. Having been reassured, I no longer worried about them, even if I got them when operating. Some years later, when the attacks occurred more often, I saw my own hospital's cardiologist (ECG No 2). He told me not to worry if I developed an attack when operating. His advice—"Take a quinidine tablet." So it continued, and I did not bother to feel my pulse, as I suffered no inconvenience beyond knowing that I was palpitating; but I declined a cup of coffee after operating.

Shortly after retiring when I was about 70, having had about three days of very irregular pulse, normal rhythm resumed of its own accord. Some days later I suddenly found that my right arm appeared paralysed. Panicking, I considered that I had had a stroke, but after some five minutes or so full mobility of the arm returned. Thinking that I might have had an infarct, I consulted my general practitioner, who had another ECG (No 3) carried out. He confirmed that I had arrhythmia, and sent me to see a third cardiologist, who again took an ECG (No 4), and suggested that I should take lanoxin. When I reported back to my general practitioner for a prescription he gave me the opinion that I should take lanoxin to the end of my days, and suggested that I should "live from day to day."

That was probably six years ago, and in the interval I managed to nurse my paralysed late wife, having to get her up on to her feet whenever she fell, which she did several times a day. I never bothered about my own heart misbeating. Now occasionally, if I remember it, I do take once a day a lanoxin tablet 0.25 mg if palpitations disturb me more than usual. I think that it steadies the rhythm for some hours, then once again I start irregular cardiac beating. I no longer worry about my heart's function. I have had long anaesthetics for a prostatectomy and three retinal welding operations. I do gardening, home decorating, and smoke heavily.

So cheer up, Dr Grant. Don't worry too much over your pulse rhythm misbeating. Maybe you will live 30 years with it irregular as I have done since first my heart started misbeating—the exact pathology of which, as a surgeon, I never remember. Extrasystoles, fibrillation, or other conditions with

abbreviated letters, so long as I have no other symptoms than pulse irregularity, I ignore.

I gather that quinidine is "out" and lanoxin is "in" for these arrhythmias, but I note with interest Dr Grant's experience with disopyramide.

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Aetiology of appendicitis

SIR,—Mr F T deDombal and Dr A J Hedley (24 March, p 820) dispute the assertion by Mr D P Burkitt and others that "the geographical distribution of appendicitis indicates that it is a disease associated with Western culture" (3 March, p 620), and cite recent reports published from a number of non-Western countries attesting to the frequency of acute appendicitis, found to be the commonest cause of hospital admission for acute abdominal pain.

This is also our experience with Canadian Indians and Eskimos in recent years. "Epidemics" of acute appendicitis, the appendix often being ruptured by the time the patient reached hospital, were noted in northern Alberta Indians in the 1950s, sub-arctic Indians and Eskimos of the western Arctic in the 1960s, and eastern arctic Eskimos in the 1970s, corresponding in each population group to drastic changes in diet and life style. This contrasts sharply with our experience in the early 1950s, when I saw during two years caring for more than 3000 Eskimos and Indians in the western Arctic and lower Mackenzie River area only three cases of appendicitis—all in children at native residential schools—while none occurred in the majority of children and adults staying at home.

Similarly, not one case of appendicitis was observed from 1928 to 1958 in a mission hospital in Pangnirtung caring for 4000-5000 Eskimos in Canada's eastern Arctic. But in 1964, for example, in an eight-week journey aboard a supply ship visiting the same shores three emergency appendectomies had to be performed in addition to numerous cases shipped annually to the new Frobisher Bay Hospital, where appendicitis has become the most frequent operation and commonest cause of hospital admissions for acute abdominal pain just as a decade earlier in the western Arctic.

Our observations in northern Indians and Eskimos—spanning a generation—of a marked change in the epidemiology of appendicitis, linked in each population group to drastic changes in diet and life style, are in accord with the ideas of Mr D P Burkitt and others that differences in geographical distribution of a number of intestinal diseases—such as diverticulosis, cancer of the colon in the older, and appendicitis in the younger age groups—may be associated with diet and the way of living of modern man. There is, however, a noteworthy difference: northern hunting populations, especially the almost exclusively carnivorous Eskimos, ingested in their traditional diet no more fibre than modern Western man, in contrast to most non-Western population groups, which led Burkitt and others to their epidemiological deductions. We found that intestinal transit times were nevertheless much shorter in traditionally living Eskimos than in modern urbanised Eskimos owing both to dietary patterns (polyunsaturated fatty

acids in fish and game, especially sea mammals) and to activity patterns.

Considering the marked changes in the epidemiology of appendicitis as well as cholecystitis, diverticulosis, and cancer of the colon and rectum,² in Eskimos during the last generation, one must wonder if the relatively high incidence of appendicitis in non-Western countries cited from recent reports by Mr deDombal and Dr Hedley reflects new developments in the same way as in our northern native population groups.

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¹ Schaefer, O, et al, in *Proceedings of IV International Symposium on Circumpolar Health, Novosibirsk, October 1978*, in press.

² Schaefer, O, et al, *Canadian Medical Association Journal*, 1975, **112**, 1399.

SIR,—One aspect of the geographical distribution of appendicitis appears to be overlooked by Mr D P Burkitt and others (3 March, p 620). This is the undoubted decrease in the incidence of the disease in Britain and the West over the past few decades. I summarised the evidence for this decrease in 1976.¹ On the basis of published estimates of case-fatality rates in the British and North American literature it seems that the incidence of appendicitis in Britain increased from the late nineteenth century up to about 1940 (in accord with the dietary hypothesis), but the incidence has decreased since about 1940.

While part of the decrease may be due to changes in diagnostic criteria and nomenclature¹ it must be true that, as Mr F T de Dombal writes (24 March, p 820), the relation between appendicitis and modern Western culture is undoubtedly far more complex than any simplistic association.

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¹ Donnan, S P B, and Lambert, P M, *Population Trends*, 1976, **5**, 26.

Psoriasis and cancer

SIR,—I read with considerable interest the paper by Professor Sam Shuster and colleagues (7 April, p 941), and also the previous paper by Chapman et al.¹ The question has been raised² as to whether or not psoriatics are possibly relatively resistant to cancer in the same way as they seem to be less prone than average to solar keratosis³ and as some prokaryotes, for example, are less susceptible to radiation-induced mutational damage to the DNA. Naturally, considering only lesions induced in skin this, in a sense, could be a side effect, "trivial" but obviously useful—related perhaps to the enhanced skin cell turnover of psoriatics. On the other hand, if a more fundamental and possibly general cancer resistance were true, the relation of psoriasis to HLA haplotypes is of added interest and would need an explanation.² The observations on impaired aryl hydrocarbon hydroxylase activity in psoriatic skin¹ introduce a further special point in relation to HLA.

It would therefore be most interesting to know whether Professor Shuster and his colleagues have had an opportunity of HLA testing their psoriatic patients in whom they

have studied the aryl hydrocarbon hydroxylase. Even if numbers are small the data might be informative.

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¹ Chapman, P H, Rawlins, M D and Shuster, S, *Lancet*, 1979, **1**, 297.

² Polani, P E, in *Human Genetics: Possibilities and Realities* (discussion), Ciba Foundation Symposium No 66, ed R Porter and M O'Connor. Amsterdam, Excerpta Medica, in press.

³ Koscard, E, *Australian Journal of Dermatology*, 1976, **17**, 65.

* * * We sent a copy of this letter to the authors, whose reply is printed below.—ED, *BMJ*.

SIR,—We have done HLA studies on most of the patients with psoriasis in whom we showed a decrease in epidermal aryl hydrocarbon hydroxylase activity.¹ The results, which we will publish shortly, show an increase in BW17 frequency. In our paper we also suggested that the same abnormality of aryl hydrocarbon hydroxylase activity with its consequences for cancer protection is a possibility requiring testing in other organs; but, unlike Professor Polani, we would consider protection against cancer of the skin as major enough—if only because 2% of the population could be a little more optimistic about the future of photo-chemotherapy for psoriasis.²

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¹ Chapman, P H, Rawlins, M D, and Shuster, S, *Lancet*, 1979, **1**, 297.

² Rogers, S, et al, *Lancet*, 1979, **1**, 455.

Defects in copper winding of intrauterine devices

SIR,—During recent months we have been concerned at finding apparent breakage and loss of the copper winding from Gravigard intrauterine devices removed from patients attending family planning clinics. In each of our cases the intrauterine device had been inserted more than three years previously. The amount of copper winding lost has varied from a small deficiency to almost total loss.

We would be interested to know if others have found similar defects in the copper winding of devices left in situ for more than two years.

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Estimating gestational age

SIR,—I would like to support Professor C J Roberts and others in a plea for accurate obstetric dating (14 April, p 981). Dating is so important, not just for such screening programmes, but also in distinguishing "small-for-dates" gestation from mistaken dates and, of course, for reducing unnecessary induction of incorrectly diagnosed prolonged pregnancy. Most tests of fetal well-being likewise depend on an accurate knowledge of gestational age.