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