industry, membership of the board appears to consist almost entirely of biochemists and nutritionists with no cardiologist and little representation of epidemiology: much of the great body of epidemiological data was not considered. Perhaps the most striking aspect of the report is its inconsistency: the board considers it unwise to make specific recommendations in the absence of conclusive evidence and yet it has found itself able to make firm recommendations concerning a reduction in salt intake.

Sir John claims furthermore that "none of the best-conducted and statistically controlled trials of lipid-reducing polyunsaturated-fat diets or drugs have shown any preventive effects on the incidence or progress of coronary heart disease." There is no doubt that the recent WHO clofibrate trial showed a reduction in coronary heart disease incidence related to the cholesterol lowering property of this drug (the therapeutic implications of this study have been widely discussed, but this very finding has not been disputed).[4] We have reviewed in some detail the dietary studies of coronary heart disease prevention[4] and concluded that while absolute proof of a beneficial effect of dietary modification will probably never be produced in any single clinical trial, the results of the trials are encouraging. It is of interest that one of the two studies quoted by Sir John was indeed well conducted but certainly not "controlled" in the true statistical sense.[5]

It is difficult indeed to resolve thiis ssue in the correspondence columns of your journal, but we cannot help but wonder whether those who oppose dietary change may not at times be even more open to criticism than the polyunsaturated fat evangelists." We would not count ourselves among the latter since the dietary modification which we recommend does not represent a substantial increase in polyunsaturated fat but rather a reduction in saturated fat which marginally changes the polyunsaturated to saturated fat ratio.

J I MANN
JEAN W MARR

University of Oxford,
Royal Free Hospital Medical School,
London NW3 2PN

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[5] Mann JI, Marr JW. In: Miller NE, Lewis B. eds. *Lipoproteins, Atherosclerosis and Coronary Heart Disease.* Elsevier/North Holland. In press.
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SIR,—Professor Barry Lewis's article, "Dietary prevention of ischaemic heart disease—a policy for the '80s" (19 July, p 177), and Sir John McMichael's letter (16 August, p 517) both concentrate on the cardiovascular rather than the dietetic and alimentary aspects of this problem. We might reasonably ask ourselves what else has changed in Western diets since the coronary heart disease epidemic began.

Although there may have been some changes in the amounts of carbohydrate, fat, protein, and calories which we consume, the three major changes appear to have been loss of fibre, increase in sugar, and the use of substances which give food an "improved" texture—for example, emulsifying agents. Emulsifying agents are naturally present in a wide variety of foodstuffs, but present UK Government regulations list 57 permitted emulsifiers and stabilisers which may be added to food.[1]

Normally almost all ingested fat is absorbed after being emulsified by the bile salts; it is possible, however, that added hydrophilic emulsifiers are absorbed as are the bile salts in the enterohepatic circulation; hydrophilic detergents certainly are (unpublished data presented by J A McDermott, D H Hughes, and P M Quinlin of Proctor and Gamble, Miami Valley Laboratories, Cincinnati, Ohio to the Society of Toxicology meeting in March 1975). If this were the case, they could reduce the amount of bile salts required to maintain losses from the enterohepatic circulation. Even if this is not the case, added emulsifiers in the diet "pre-emulsify" lipids entering the gut which might allow the enterohepatic circulation of bile salts to become sluggish. Thus excretion of cholesterol, precursor of the bile salts, might be impeded.

Oral chenodeoxycholic acid in a dose of 750 mg daily will reduce the cholesterol saturation of bile in subjects who are on a reducing diet,[2] yet a level of 750 mg daily of this oral bile acid used in the treatment of gall stones is probably less, in terms of its surface tension lowering effect, than the amount of added emulsifier in some Western diets.

Thus added emulsifiers might cause changes in the enterohepatic circulation and in cholesterol levels. If high concentrations of low density lipoprotein cholesterol and low concentrations of high density lipoprotein cholesterol were found to be associated with diets containing substantial amounts of added emulsifiers, then they could be a risk factor in ischaemic heart disease.

Perhaps the role of emulsifying additives in our Western diet should be reconsidered before formulating a dietary policy for the '80s.

G H B MARTIN

Nuneaton,
Warks CV11 5TN

[1] *The Emulsifiers and Stabilisers in Food Regulations.* London: HMSO, 1978:8-9. (No 1486).
[2] Mok HYI, Von Bergman K, Crouse JR, Grundy SM. *Gastroenterology* 1979;**76**:556-87.

SIR,—In challenging Professor Barry Lewis's reference (19 July, p 177) to evidence of an increase in the incidence of myocardial infarction during this century, Sir John McMichael (16 August, p 517) suggests that physicians in the early decades simply did not recognise the disease and that Sir James McKenzie (who nowhere in his writings describes acute myocardial infarction) was unable to diagnose "characteristic attacks of myocardial infarction in himself."

If in McKenzie's time men in their 40s and 50s had been suffering infarctions on the same scale as today, it is inconceivable that he and his contemporaries would have failed to describe a disease which was causing such widespread havoc. McKenzie himself began to experience angina of effort aged 55[1] but his capacity for work thereafter seems to have been quite unimpaired. At 65 he was still able to play two rounds of golf,[2] and having run his three score years and ten he died aged 72. This relatively benign manifestation of coronary ischaemia can hardly be accurately described as "characteristic attacks of myocardial infarction."

Sudden infarction, often quite unheralded in a previously healthy subject, was first reported in this country in the 1920s[3] (personal communication from Dr Rae Gilchrist) and reached "epidemic" proportions in the two decades following the second world war. This history is of the utmost importance in understanding causes and seeking prevention. The wealthy Victorians and Edwardians attended by McKenzie and Lewis ate as much or more saturated fat than their present day counterparts[4]; their freedom from infarction cannot therefore be explained by a low intake of dietary fat, and they had never heard of polyunsaturated margarine. Nor has there been any increase in the consumption of saturated fats in the United Kingdom[5] which could possibly explain the upsurge in all classes of coronary deaths and disability.

As T L Cleave has repeatedly emphasised the most significant change in the nation's diet which preceded the appearance of widespread myocardial infarction and other associated diseases was the huge increase in the consumption of sugar which started in the latter half of the nineteenth century and continued apart from two world wars on to the 1950s.[6]

But laboratory-based obsession with the minutiae of lipid metabolism, a flight from commonsense, along with a sustained advertising campaign have succeeded in persuading many housewives and a surprising number of doctors to change their dietary fats. They have been persuaded to substitute highly unnatural processed foods such as margarine, corn oil, or some dreadful concoction of soya bean extract for natural fat of animal origin. There is no evidence at all that these substitutions have improved our health; indeed, it would seem that attempts to lower cholesterol levels by unnatural foods will do us nothing but harm.

W W YELLOWLEES

Aberfeldy,
Perthshire

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[3] Mead TW, Chahkrabarti R. *Lancet* 1972;ii: 913.
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[5] Trenchard T. *Health and Hygiene* 1977;**1**:77-82.
[6] Cleave TL. *The Saccharine Disease.* Bristol: John Wright, 1978.

SIR,—May I respond to those issues raised by Sir John McMichael (16 August, p 517) which were factual in nature? In January he and I amicably debated the role of diet in ischaemic heart disease. To my regret, he declined my suggestion that the proceedings be published, which would have afforded an opportunity to assess our arguments side by side. My Regular Review (19 July, p 177) summarised my contribution to this debate. Despite the time lapse Sir John has refuted none of my data. What does he offer instead?

(1) "None of the best-conducted [trials] have shown any preventive effects. . . ." I listed five trials in which reduced ischaemic heart disease incidence, and in some mortality, was observed. Trials in which the trial size and the extent of plasma cholesterol reduction are inadequate are not valid tests of the lipid theory.

(2) "Only about 50% of atherosclerotic plaques contain any visible cholesterol. . . ." Elsewhere Sir John omits "visible" and cites Osborn as his source.[1] Osborn, whose histological study does not mention selection criteria, grading units, or staining protocol, rarely saw coronary lipid deposits before age 5; but he noted lipid in 26 of 43 arteries (the table does not refer to plaques) at 16-20 years, 21 of 22 at 36-40, and 83% of those aged 41 and