extent or degree of coronary atheroma was found at necropsy in Glasgow men compared with London men,⁷ but mortality from ischaemic heart disease is much greater in Scotland, particularly around Glasgow, than in the south of England.⁸ Preliminary observations indicate that platelet aggregation to thrombin, collagen, and arachidonic acid is greater in farmers in the south-west of Scotland, where the mortality is high, than in farmers in the central east of Scotland, where the mortality is lower.⁹ Furthermore, the highest mortality from stroke in the UK is also in Scotland and particularly in the central-west and north-west regions.⁸

More data are needed to confirm or refute the longstanding views of an important association between a deficiency of EFAs and ischaemic heart disease. This may be mediated through prostaglandin-related thrombogenesis rather than the development of coronary atheroma. We now need to collect evidence to determine whether the addition of certain EFAs to the diet reduces indices of hypercoagulability in man. If so, such specific dietary supplementation should be tested formally to see if it is preventive in communities with a very high vascular mortality.

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¹ Sinclair, H M, *Lancet*, 1956, **1**, 381.

² Bronte-Stewart, B, *British Medical Bulletin*, 1958, **14**, 243.

³ Logan, R L, *et al*, *Lancet*, 1978, **1**, 949.

⁴ Insull, W, *et al*, *Journal of Clinical Investigation*, 1969, **48**, 1313.

⁵ Moncada, S, and Vane, J R, *British Medical Bulletin*, 1978, **34**, 129.

⁶ Nordoy, A, *et al*, *European Journal of Clinical Investigation*, 1979, **9**, 5.

⁷ Crawford, T, and Crawford, M D, *Lancet*, 1967, **1**, 229.

⁸ Fulton, M, *et al*, *British Heart Journal*, 1978, **40**, 563.

⁹ Renaud, S, personal communication, 1979.

SIR,—Before we rest on our laurels, satisfied 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" (Dr J I Mann, 17 March, p 732), may I make a plea for a more careful temporal analysis of this association before we pronounce judgment upon it?

I would be just as pleased as anyone else if this association were in fact causal, and if we were at last making inroads into the prevalence of this disease. In order to confirm this, however, and to avoid the trap of seeing only what we want to believe, we must demonstrate not just a parallel change in diet but the fact that the dietary change preceded the change in mortality. As we are dealing with a long-term degenerative process, it is naive to expect an instantaneous response to the modification of one of the many known risk-factors. If the dietary changes are indeed responsible for the decline in mortality, we would surely expect them to antedate the decline.

There has been a considerably accelerated decline in the mortality rate from ischaemic heart disease in the USA since the late 1960s, but what is equally significant is that the rate reached a plateau during the 1950s. In other words, there was a definite deceleration at least a decade earlier. The alteration in causal factors contributing to this deceleration must therefore have occurred at least in the early 1950s, if not in the late 1940s. Does this coincide with the dietary changes? Perhaps the dietary influence is merely accelerating an

already declining rate. If so, what is the cause of the original decline?

What I would like to see is an age- and sex-specific analysis of ischaemic heart disease rates in the USA since the 1940s, with cohort as well as cross-sectional analyses. When it is established just exactly which sectors of the population are contributing to the decline, and when this is tied to evidence of dietary changes in those sectors, perhaps we may then be a little more certain about the role of nutritional factors in this very important natural experiment.

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SIR,—Dr J I Mann (17 March, p 732) obviously has a touching confidence in the two trials of polyunsaturated fat (PUF) diets which made the flimsy cornerstones of the Royal College of Physicians-British Cardiac Society report. Mitchell disposes of the Finnish mental hospitals trial in the EEC report. Mann objects to secondary prevention trials because "the subjects are no longer young" but puts his faith in the Los Angeles Veterans trial on subjects aged from 55 to 88. Were they free of arterial disease?

Secondary prevention trials cannot be brushed off and ignored. If a valid causal factor in a long-lasting progressive disease is identified its reduction should logically retard further progression in identified patients and delay or reduce mortality. All published efforts to help by drug or dietary reduction of blood cholesterol have uniformly and convincingly failed. In addition to those quoted, a trial for eight years in Bavaria¹ by Stocksmeier failed to improve prospects (and support from the food industry was reduced). A trial in patients under 60 in the University of New South Wales² showed that those on PUF diets did much worse than those left on a free diet.

Primary prevention trials of cholesterol reduction by clofibrate have failed to produce convincing evidence of benefit. An interim report on a primary multifactorial dietetic (plus smoking and blood pressure control) trial in North Karelia³ has shown in the first three years an increased incidence of acute myocardial infarction from 14.9 to 18.4 per 1000 men and no change in incidence in the 30 to 64 age group. Even Dr Ruth M Kay (3 March, p 6283) admits that proof of benefit may be impossible. If we accept this why proceed with such massive propaganda which can benefit only commercial interests?

Since efforts at blood cholesterol reduction have failed interest is now shifting to high-density lipoprotein (HDL), alleged to be protective, and low-density lipoprotein (LDL), alleged to be damaging. The evidence quoted by Dr Kay that a PUF diet will favourably affect this ratio is equivocal.

Drs K G Taylor and D J Betteridge (17 February, p 485) believe that cholesterol feeding produces "atheroma," but experts like Benditt and Armstrong believe that the fatty lesion thus produced is a very poor model of the human disease. Impurities in cholesterol have vitiated many animal experiments.⁴ Purified cholesterol produces only xanthomatous lesions that never thrombose.⁵ Even in the rare homozygous form of familial hypercholesterolaemia the disease is quite different from the common form of coronary artery disease. It affects only the mouths of the coronary arteries, leaving the main vessels and their subepicardial branches unaffected. Dr Mann might consider this important difference in the cause of angina.

The Belgian report proved nothing, as the mortality in 1947 in North Belgium was already lower than in South Belgium before preference for margarine occurred. Furthermore comparisons are hardly acceptable between two different ethnic groups.

The Blumgart report⁶ on induced myxoedema is

criticised by Mann and Dr P B S Fowler (10 March, p 681). The method of cholesterol estimation at that time gave much lower values. The upper limit was taken as 230 mg (today >300 mg). All patients showed a rise in cholesterol, and five out of eight reached concentrations of 338 to 524, which were very gross. After many years of this induced hypercholesterolaemia there were no coronary occlusions. I give priority to Blumgart for this most meaningful result of a planned therapeutic experiment which does not require statistics to prove its validity.

We must not confuse correlations or risk factors with causes. The Framingham study showed that 82% of those with all four risk factors (cholesterol, smoking, blood pressure, and sedentary lives) were still alive after 10 years, so the predictive value of any one risk factor is very limited. Cigarette smoking indeed has the strongest correlation and demonstrations of damage to the vascular endothelium by cigarette smoke encourages recognition of this hazard.

Norum's poll of 214 doctors (only a limited number of whom were critical scientists) was answered by 85%. As 92% of those who answered were already following the diet the result was only an indication of wishful thinking.

I agree that death rates from coronary heart disease in different countries are difficult to compare. That France has been under-recording its deaths from this cause by more than half⁷ owing to different diagnostic habits is a salutary reminder of the fallibility of these figures. But a rise from the baseline may be meaningful in an individual population (for example, Sweden). It is certain that the high PUF diet of Israelis does not prevent a high incidence of the disease.

Dr J R Vane's approach (17 February, p 484) gives a rational twist to the story. Rather than concentrate on cholesterol reduction there are hopes that certain fatty acids may be precursors of anti-thrombotic substances, and this is to be pursued. It might even be relevant to plaque formation if Duguid's work⁸ is right. The real cause of coronary obstruction is thrombosis.

We need a fresh approach to the problem at scientific level and should avoid further public speculation and confusion by repeated propaganda through the media until we have clarified our own professional minds and shaken off what most critical doctors are likely to regard as an untenable hypothesis of causation.

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¹ Stocksmeier, U, *Influences of Saturated and Polyunsaturated Fats on Blood Lipids and some Socio-medical Variables*. National Dairy Council, London, 1978.

² Woodhill, J M, *et al*, *Drugs, Lipid Metabolism and Atherosclerosis*. New York, Plenum, 1979.

³ Milmo, S, *Doctor*, 9 February, 1978.

⁴ Imai, H, *et al*, *Archives of Pathological Laboratory Medicine*, 1976, **100**, 565.

⁵ Duguid, J B, *The Dynamics of Atherosclerosis*. Aberdeen, Aberdeen University Press, 1976.

⁶ Blumgart, H L, Freedburg, A S, and Kurland, G S, *American Journal of Medicine*, 1953, **14**, 665.

⁷ Richard, J L, *et al*, *INSERM*, 1973, **21**, 11.

Return to work after coronary artery surgery for angina

SIR,—Drs P G F Nixon and N G Weinstock should not be astonished (27 January, p 265) at the successful results published by Mr J Wallwork and others (16 December, p 1680). Not only do these results accord remarkably with other transatlantic experience but also with results in England.

From the United States, Cannon¹ reports that 63% of 375 survivors of saphenous vein bypass grafting for angina were working post-operatively, while a further 16% were angina free but not working. Similarly, Rimm² reports that 83% of 893 men (mean age 52.3 years) returned to employment.

At the meeting of the Society of Thoracic

and Cardiovascular Surgeons in Leeds in September last year we reported our experience from St Thomas's Hospital of 123 male patients operated on for ischaemic heart disease by one surgeon (BTW): 72% were working one year after operation, with a further 6% classified as fit to work but unemployed; 8% had retired; and only 14% were still classified as having symptoms. These results have been briefly reported¹ and we will be publishing full details of this study shortly.

We accept the reservation that no comparison has been made with other forms of therapy.

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¹ *The Times*, 30 September 1978.

Driving after anaesthetics

SIR,—I was most interested in the correspondence between Dr J D J Harvard and Dr P J F Baskett and Professor M D A Vickers (10 March, p 686) on the subject of driving after general anaesthesia. Many anaesthetists instruct their patients not to drive for at least 24 hours following general anaesthesia. Dr Harvard's suggestion that the time limit should be 48 hours certainly appears a little too cautious, but none the less safe.

Day-case anaesthesia is practised at many British hospitals, and these days anaesthetists do have a variety of short-acting anaesthetic agents with rapid recovery at their disposal. In recent years Kortilla¹ has published frequently on the hazards of driving following various anaesthetic techniques. I should add that a great deal of his work has involved human volunteers. However, patients frequently ignore medical advice in the immediate postoperative period. An assessment of 100 postoperative outpatient cases in 1972 by Ogg² revealed that 73% of car owners undergoing day-case surgery drove within 24 hours; 30% drove within 12 hours and 9% actually drove themselves home unaccompanied. No major traffic accidents were reported after this study.

With these results in mind, I have been attempting in Cambridge to discover why patients should disregard medical advice. Is this a sign of the times? I have studied memory function after day-case anaesthesia, and my results so far show that many outpatients fail to retain new information or advice given to them in the postoperative period for up to three hours after simple general anaesthesia. However, memory function can be enhanced in the postoperative period if patients are given both written and verbal instructions before surgery. It would appear from my recent research that outpatients do not disregard medical advice through malice. They fail to comply with instructions because of a failure of memory retention for new information after simple anaesthesia.

In my own outpatient anaesthetic practice I confine the duration of anaesthesia to 30 minutes. All patients are accompanied home by a responsible adult, and they are given preoperative instructions not to drive, operate heavy machinery, or return to work for at least 24 hours.

The whole question of when it is safe to

drive after general anaesthesia could be answered. Outpatient or volunteer anaesthetists could submit themselves to general anaesthesia and take driving tests at 24 and 48 hours at a police driving school on some deserted aerodrome. I know of no published work on this aspect of anaesthetic practice.

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¹ Kortilla, K, *et al*, *Anaesthesiology*, 1975, **43**, 291.

² Ogg, T W, *British Medical Journal*, 1972, **1**, 573.

SIR,—Most anaesthetists with substantial involvement in anaesthesia for short-stay patients will be aware of the advice contained in *Medical Aspects of Fitness to Drive*.¹ My attention was forcibly focused on the problem by a patient who drove through a local general practitioner's front fence on his way home after a general anaesthetic for an outpatient vasectomy at a local teaching hospital.

Apart from advising patients verbally and issuing written instructions for day-stay patients, I have studied a series of patients undergoing termination of pregnancy under general anaesthesia in our day-stay unit. The unexpected result of this study is that 50% of these patients did not return to work the following day and that 20% complained of drowsiness on the day after their anaesthetic. I ascribed this to the use of 10 mg diazepam in the anaesthetic "cocktail," and I have now deleted this from the cocktail and I am studying a further group of patients. It can be argued that termination of pregnancy is psychologically, if not physically, traumatic, but in the public's mind termination of pregnancy is clearly regarded as a "lunch-time operation" par excellence. Other workers studying day-stay for dental anaesthesia have found broadly similar results.²

Clearly, there is a need for careful clinical evaluation of the sequelae of anaesthesia for short-stay surgery to ensure that only genuinely short-acting agents are used and patients are not subsequently put at risk through ignorance.

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¹ Harvard, J D J, in *Medical Aspects of Fitness to Drive*, ed A Raffle, 3rd edn, p 43. London, Medical Commission on Accident Prevention, 1976.

² Muir, V M J, Leonard, M, and Haddaway, E, *Anaesthesia*, 1976, **31**, 171.

Use of car headlamps

SIR,—Dr D F Martin (17 February, p 490), in advocating the use of headlamps, particularly in snow and rain, would be adding to the trouble of glare from reflection even though the headlamps were properly aligned and dipped. Glare is bad enough even when roads are dry. Lots of people with normal eyesight cannot stand the glare of oncoming headlamps and it is one of the causes of accidents. Some cars have as many headlamps as rally drivers and think they can blaze their way through without any consideration for others. It is not so much requiring to be seen as being able to see anything else. One may remain blinded for an appreciable time after the offending car has gone by. It is also very often impossible to see indicator lights adjacent to headlamps, and a driver with headlamps on may think he has made adequate indication of his intention to

turn right or overtake when, in fact, his indicator has been completely drowned by his own headlamp.

Quite apart from people with normal eyes, there are many with perfectly adequate sight for driving who have chronic irritation from one cause or another (such as seborrhoeic blepharitis, dry eyes and rosacea, early lens opacity, old corneal nebulae, or just some degree of pigment deficiency) and who get blinded all the more easily by dazzle. One should not penalise our blue-eyed boys or anyone else. This suggestion of excessive use of headlamps is something the BMA should condemn. It is no substitute for good street lighting; and if that is good, as it should be for urban areas, then routine use of headlamps does more harm than good. One can flash them more effectively at crossings or other danger spots if they are not on at all than if they are.

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Preventing motorcycle accidents

SIR,—We entirely agree with the comments made by Dr J C Avery (10 March p 686) concerning the compulsory wearing of crash helmets. In addition, we feel there is a need for much more stringent legislation governing the use of motorcycles. In a recent survey in Newcastle¹ we noted nearly a 50% rise in the number of injured motorcyclists during 1978 compared with 1977; since the police, on whose figures national numbers are based, investigated only half the accidents in our survey, the true magnitude of the national problem may be even larger. In many accidents in this survey the lack of experience of the motorcyclists was a major factor, 60% having held a provisional licence for under six months. With the rates for fatal or serious accidents to motorcycle drivers now a staggering 1.3% per 100 vehicles in use,² legislation must be enacted towards preventing accidents.

We would suggest that the age of eligibility for a provisional licence should be raised to 17 years, similar to that of a motorcar driver (at present 16-year-olds can legally ride only mopeds, although many disregard this). Formal training before being allowed on the roads should be compulsory, and an enforced, stringent, motorcycle driving test is also needed to replace the current inadequate test, and this should include a section on motorcycle safety. Improved investigations and detection of motorcycle offences by the police would be of value. Many drivers in our survey admitted to speeding and to having excess alcohol levels, and only through prosecution and heavy fines or suspended licences will the message of road safety be delivered. In Britain further encouragement for the wearing of bright protective clothing may also help reduce the number of serious injuries; this is now compulsory in many European nations. Finally, manufacturers should be compelled to incorporate improved safety designs similar to those enforced by Californian regulations,³ and the motorcycle "lighting-up law" (front and rear lights on at all times) should be enacted. Californian records indicate that although their motorcycle population increased by 33%, fatal and serious accidents increased by less than 6% as a result of these measures.