oestrogen has been available for many years.

Using data generated in the Royal College of General Practitioners' oral contraception study, I was unable to show in 1974[1] any correlation between reports of hypertension in oral contraceptive users and the oestrogen dose. At the same time, there was a suggestion that the progestogen component might be implicated. Convincing evidence of this association was published in 1977[2] and additional data were made available in 1980.[3] I accept that some workers regard conclusions drawn from reports of the occurrence of hypertension as less reliable than analyses of blood pressure measurements, but in retrospect the doubts may not have been entirely justified.

We must also take account of the strong evidence that the excess of arterial disease in oral contraceptive users is associated with the progestogen activity[3-6] and that this is closely correlated with undesirable changes in high-density lipoprotein cholesterol concentrations[6-9] and carbohydrate metabolism.[10] Clearly, Khaw and Peart's advice to use oral contraceptives with the lowest effective progestogen activity must be emphatically endorsed.

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Coronary disease

SIR,—I was very pleased to read Dr J Tudor Hart's recommendation of a prudent diet for the prevention of ischaemic heart disease in general practice (31 July, p 347). The near absence of ischaemic heart disease in east Africa (four cases recognised in Africans in 40 years (1928-68)) and its relationship to a low-fat, low-sugar, high-carbohydrate diet is well known. The good correlation of the changing diet among Japanese immigrants is less well recognised (table).[1] Morris *et al*[2] showed that an increase of cereal fibre was particularly important, with five times the number of cases of coronary thrombosis occurring among the one-third of a group of 337 southern Englishmen who ate the least fibre over 20 years when compared with the third who ate most cereal fibre.[2]

In the present uncertainty about the relative importance of the different factors one can only recommend a reduction in total dietary fat, particularly the saturated portion, a reduction of sugar, and an increase in unrefined carbohydrate, particularly that containing cereal fibre.

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SIR,—We were interested in the article on the prevention of coronary disease by Dr Julian Tudor Hart (31 July, p 345) and note with approval that the prevention lobby is at last gaining momentum in this country. The fact that coronary disease varies widely in incidence in different parts of the world suggests that environmental factors are of major importance. Smoking does not seem to induce the disease in Japan and therefore appears to act only in conjunction with certain dietary factors.

There is a general pessimism in the United Kingdom that individuals will not readily alter their dietary habits, as has been the case with such beneficial results on the incidence of coronary disease in the United States. This view is probably partly based on the difficulty in reducing alcohol and cigarette consumption. We suggest that this is a false analogy, because alcoholics in particular are a special group with particular addictive difficulties, and there is evidence from the growing number of health food shops and health magazines that a sizeable proportion of the population would indeed be willing to listen to dietary advice. We suggest that part of the problem is that the information is confusing to the lay public. Advice on the consumption of saturated fats, carbohydrates, and fibre mean little or nothing to the lay housewife without even a superficial knowledge of nutrition. There is quite clearly a major information and comprehension block, which must be overcome.

We therefore decided to study two specific foods, meat and eggs, which often form the main constituent of a meal. Assuming three major meals per day there is a total score of 21 meals per week. Subjects were asked how many times they ate meat. Sixty-five patients who had survived a myocardial infarct established by electrocardiographic and enzyme studies were compared with 177 age- and sex-matched controls. The mean meat score in the myocardial infarct patients was 10·938 compared with 8·102 in the controls ($p < 0·001$). Egg consumption per week was also assessed, and the mean for the infarct group was 4·194 compared with 2·869 in the controls ($p < 0·001$). A full report and mathematical analysis will be published elsewhere but the evidence suggests that a diet containing a maximum of four meat meals per week and three eggs per week would significantly reduce the risk of coronary disease. Such simple advice is easily understood and deserves wide dissemination.

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Changes in diet among Japanese immigrants to Hawaii and USA[1]

	Starch (%)	Sugar (%)	Fat (%)	Comment
Japanese in Japan	52	11	15	No fibre figures given
Hawaiian Japanese	33	16	33	Fourfold increase in saturated fats
Californian Japanese	30	17	38	Further rise of saturated fats

Should every survivor of a heart attack be given a beta-blocker?

SIR,—The series "Should every survivor of a heart attack be given a beta-blocker?" was an excellent review of the state of knowledge by Professor J R Hampton (3 July, p 33), Professor A Breckenridge (p 37), and Professor Geoffrey Rose (p 39). We would like to offer comments on three issues: cardioselective versus non-selective beta-blockers, the importance of replicating findings, and the interpretation of cumulative mortality curves.

Firstly, as pointed out by Professor Hampton the Multicentre International Trial of practolol reported selective benefit for patients with anterior infarction. Interestingly enough, the same findings were observed in the Goteborg metoprolol trial (A Hjalmarson, personal communication). It is intriguing that the two largest trials of cardioselective beta-blockers showed a benefit restricted to patients with anterior infarcts. None of the large trials of non-selective beta-blockers has reported such results. This could be a chance finding but may also be real. Only continuing or further trials can resolve this question.

Secondly, we agree with the view that the value of very early treatment has not been established. Of six randomised placebo-controlled trials where intravenous treatment was started on admission (followed by oral treatment), three[1-3] showed a higher and three[4-6] a lower mortality in the beta-blocker group. Only the single-center Goteborg metoprolol trial[5] reported a significant ($p < 0·03$) short-term reduction in mortality. McIlmoyle *et al*,[6] using the same dosage of metoprolol, could not replicate the Swedish results. This raises the question whether the findings from the Goteborg group, which has extensive experience in beta-blocker treatment, can be generalised to other settings. In multicentre trials patients from varied geographical, socioeconomic, and medical care facilities are enrolled. Thus, results of collaborative trials enable one to generalise the findings with more assurance.

Thirdly, there seems to be a tendency to overinterpret cumulative mortality curves. Time segments of the curves are defined post hoc and form the basis for strong conclusions. We would like to caution against this practice. Clinical trials are usually designed to have the statistical power to detect a treatment effect over the entire patient follow-up phase. Thus, comparisons based on time segments will suffer from insufficient power and therefore large variability. This problem increases as one moves to the right on the curve. For example, in the beta-blocker heart attack trial[7] and the Norwegian timolol trial[8] less than 10% of all deaths occurred after the average length of follow-up (25 and 17 months in the two trials, respectively). Moreover, only analyses beginning at time zero, when because of randomisation the study groups are comparable, are completely valid. Because of different mortality patterns in the beta-blocker and placebo groups