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 1974¹ 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 1977² 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 activity³⁻⁶ and that this is closely correlated with undesirable changes in high-density lipoprotein cholesterol concentrations⁶⁻⁹ and carbohydrate metabolism.¹⁰ Clearly, Khaw and Peart's advice to use oral contraceptives with the lowest effective progestogen activity must be emphatically endorsed.

CLIFFORD R KAY

Director, Oral Contraception Study

RCGP Manchester Research Unit,
Manchester M20 0TR

¹ Royal College of General Practitioners. *Oral contraceptives and health*. London: Pitman Medical, 1974.

² Royal College of General Practitioners' Oral Contraception Study. *Lancet* 1977;i:624.

³ Kay CR. *J R Coll Gen Pract* 1980;30:8-19.

⁴ Meade TW, Greenberg G, Thompson SG. *Br Med J* 1980;280:1157-61.

⁵ Adam SA, Thorogood M, Mann JI. *Br J Obstet Gynaecol* 1981;88:838.

⁶ Kay CR. *Am J Obstet Gynecol* 1982;142:762-5.

⁷ Briggs MH. *British Journal of Family Planning* 1979;5:25-8.

⁸ Baggett B, Nash HA. *Contraception* 1980;21:115-20.

⁹ Wynn V, Nithyananthan R. *Am J Obstet Gynecol* 1982;142:766-72.

¹⁰ Wynn V. *Am J Obstet Gynecol* 1982;142:739-46.

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.

JOHN TROWELL

Great Parndon,
Harlow, Essex

¹ Shun-Ichi Y. In: Trowell HC, Burkitt DP, eds. *Western diseases*. London: Arnold, 1981:337-42.

² Morris JN, Marr JW, Clayton DG. *Br Med J* 1977;iii:1307-14.

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.

RONALD FINN
M A SMITH
J R GREEN

Departments of Medicine and Computational and
Statistical Science,
University of Liverpool and the Royal Liverpool
Hospital,
Liverpool L7 8XP

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 Hjalmarsen, 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), three¹⁻³ showed a higher and three⁴⁻⁶ a lower mortality in the beta-blocker group. Only the single-center Goteborg metoprolol trial⁵ 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 trial⁷ and the Norwegian timolol trial⁸ 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

Changes in diet among Japanese immigrants to Hawaii and USA¹

	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

in the first 12 months,^{7,8} a comparison of the curves beginning after year 1 may be confounded by variables related to survival. A number of presumably high-risk patients in the beta-blocker group were saved in the first year, most probably resulting in a patient population on that treatment which was, on average, sicker than that on placebo after one year. Thus, parallel and even converging curves beyond one year could be consistent with continued benefit. In addition, we do not know what would happen if the treatment were stopped at one year. Rose⁹ has posed the question, "Do beta-blockers merely keep the wolf from the door, and when protection is withdrawn, does he return?" We tend to conclude from the absolute difference in the number of deaths between the beta-blocker and placebo groups at the end of a trial how many lives had been prolonged. The results from the Norwegian timolol trial could imply that 54 deaths (152 minus 98) were delayed by an average of at least 17 months. Another interpretation could be that more than 150 patients had their lives prolonged by an average of six months. We have no way of knowing with certainty over what period of time during the entire patient follow-up protection is provided—that is, when the door is closed on the wolf. For these reasons, it seems prudent to recommend beta-blocker treatment to survivors of a heart attack for at least three years, the duration of the trial⁷ which followed patients for the longest time.

CURT FURBERG
LAWRENCE FRIEDMAN
JEFFREY CUTLER

National Heart, Lung, and Blood Institute,
Bethesda, Maryland 20205,
USA

- ¹ Evemy KL, Pentecost BL. *Eur J Cardiol* 1978;7:391-8.
- ² Andersen MP, Bechsgaard P, Frederiksen J, et al. *Lancet* 1979;ii:865-8.
- ³ Johansson BW. *Acta Med Scand* 1980;207:47-53.
- ⁴ Yusuf S, Ramsdale D, Peto R, et al. *Lancet* 1980;ii:273-6.
- ⁵ Hjalmarson A, Elmfeldt D, Herlitz J, et al. *Lancet* 1981;ii:823-7.
- ⁶ McIlmoyle L, Evans A, McC Boyle D, et al. *Br Heart J* 1982;47:189.
- ⁷ Beta-blocker Heart Attack Trial Research Group. *JAMA* 1982;247:1707-14.
- ⁸ Norwegian Multicentre Study Group. *N Engl J Med* 1981;304:801-7.
- ⁹ Rose G. *Br J Clin Pharmacol* 1982;14, suppl 1:41-3.

Letting intrauterine devices lie

SIR,—I write to congratulate Dr Mary Pollock on her leading article (7 August, p 395) and to compare my experience with hers. My hospital clinic for inserting intrauterine devices started in 1963 and during the next decade a wide range of devices was explored. The principle of "fit and forget" was used, and no device was removed without good reason, the commonest indication being the onset of intermenstrual bleeding. Admittedly there was an occasional spontaneous fracture of a Lippes loop but removal was easy enough. There was no suggestion that pelvic infection increased with duration of use, and the only device with which late pregnancy (over four years of use) was recorded was the Birnberg bow. I would not advise changing a traditional intrauterine device. I differ from Dr Pollock in that I see no reason for an annual medical examination. I believe that there is no complication which might occur after the first 12 months of which the patient would not herself be aware. She should be instructed to report symptoms if and when they arise. I also believe that with an apparent menopause after the age of 48 the

device should be removed after six months. To leave it for two years increases the risks of endometritis and a difficult removal.

Dr Pollock does not discuss the vexed question of the copper intrauterine device. The argument for its routine removal ("changing") is based on its alleged decreasing effectiveness, either from reduction in the available copper or from deposition of protein on its surface. In my own experience the rate of pregnancy with copper intrauterine devices parallels that with the Lippes loop and I have seen no pregnancy in the fourth, fifth, or sixth years of use, although I admit that my numbers have not been sufficiently large for a statistical evaluation. I shall be interested in the experience of others.

W G MILLS

Birmingham Maternity Hospital,
Birmingham B15 2TG

SIR,—In her leading article (7 August, p 395), Dr Mary Pollock considers the symptom-free woman who has been using an inert intrauterine device for many years and concludes that the patient should continue as she is, "Provided that she agrees to return for routine medical examination, including a cervical smear, at yearly intervals." The suggestion of yearly attendance for cervical smears clearly differs from the recent recommendations of the Department of Health and Social Security committee on gynaecological cytology.¹ The committee was set up partly in order to prevent a large amount of unnecessary screening. The report certainly does not recommend yearly screening for cervical cancer in a woman such as this. The only explanation must be the detection of actinomycetes-like organisms, which, as Dr Pollock mentions, are rare, and the value of treating asymptomatic infections is unclear.

A J EYNON-LEWIS

Charing Cross Hospital
Medical School,
London W6 8RF

- ¹ Draper GJ. *Health Trends* 1982;14:37-40.

SIR,—Although, as acknowledged in your recent leading article (7 August, p 395), pelvic actinomycosis is a well recognised complication of the use of the intrauterine contraceptive device,¹ spread of this infection to the brain has not previously been reported. We present a case in which this has occurred.

A 41-year-old woman presented with a seven-day history of numbness in the left leg spreading to the left arm. Three days later she experienced jerking in these limbs lasting half an hour, not associated with loss of consciousness or incontinence. She also complained of a right-sided headache, worse in the mornings, but no nausea or neck stiffness. Her menses were normal, but for six months there had been a non-itchy green vaginal discharge. A Lippes intrauterine contraceptive device had been in situ for 10 years. General examination was normal, but there was pyramidal weakness of the left arm and leg with reduced pain and proprioception. There was a profuse green vaginal discharge, but the uterus and adnexa were normal.

The white blood cell count was $15.0 \times 10^9/l$ and the erythrocyte sedimentation rate 52 mm in the first hour. Haemoglobin, Vickers screen, urine analysis, and skull and chest radiographs were all normal. Computed tomography of the brain demonstrated a mass in the right parietal region, enhanced by Conray, which was thought likely to be neoplastic. The patient was therefore treated with dexamethasone 4 mg six hourly. After five days of this treatment, however, there was no neurological response, and histological examination of endo-

metrial curettings obtained at the time of removal of the intrauterine contraceptive device had revealed chronic endometritis with actinomycetes-like filaments. She was therefore given gentamicin 80 mg eight-hourly, metronidazole 400 mg 12 hourly, and benzylpenicillin 2 megaunits six hourly. Neurological improvement ensued within 48 hours, and craniotomy yielded 20 ml of malodorous pus; penicillin 20 000 units was instilled into the abscess cavity. Gentamicin and metronidazole were continued for 10 days and six weeks respectively; and penicillin was given for a total of eight weeks. By this time the power in the left arm and leg were normal, there were no sensory signs, and a further computed tomography scan was normal.

The following organisms were isolated and identified by standard methods.^{2,3} From the intrauterine contraceptive device: *Streptococcus milleri* (group F), *Bacteroides asaccharolyticus*, and *Actinomyces* sp; and from the brain abscess: *Streptococcus milleri* (group F), *Bacteroides asaccharolyticus*, *Actinomyces* sp, *Fusobacterium polymorphum*, Anaerobic streptococci, and *Eubacterium* sp. The similar range of organisms found in both sites in this patient indicates that her brain abscess was secondary to rather than coincidental with her intrauterine infection. It seems likely that the mode of spread of the infection from the primary site to the brain was by a small bacterial embolus which either produced a clinically undetectable lung abscess or passed through a small patent foramen ovale or the paravertebral veins.

Pelvic actinomycosis in association with intrauterine contraceptive devices is commoner when the device has been left in situ for longer than two years.⁴ The risk of disseminated infection is small but as this case report demonstrates important because of potentially life-threatening sequelae.

A E CAPEWELL
S A BOBER
V WADEHRA
C KUMAR
J E HICKMAN
H R INGHAM

Newcastle General Hospital,
Newcastle upon Tyne NE4 6BE

- ¹ Valentic JF, Pappas AA, Graber CD, Williamson HO, Willis NF. *JAMA* 1982;247:1149-52.
- ² Cowan ST, Steel KJ. In: Cowan ST, ed. *Manual for the identification of medical bacteria*. London: Cambridge: Cambridge University Press, 1974.
- ³ Duerden BI, Collee JG, Brown R, Deacon AG, Holbrook WP. *J Med Microbiol* 1980;13:231-45.
- ⁴ Duguid HLD, Parratt D, Traynor R. *Br Med J* 1980;281:534-7.

The health visitor and prevention

SIR,—Dr R P Snaith makes a rather arrogant assumption that health visitors are unconcerned with psychiatric disorders on the grounds that one failed to mention postnatal depression in an article written within a limited number of words, an anecdotal experience, and a statement by a group of research workers (14 August, p 512).

All professions, of course, have members with particular strengths and weaknesses and could relate anecdotes. It was a health visitor who diagnosed Esther Rantzen's postnatal depression, and I have painful memories of trying to support a young mother with severe puerperal psychosis without being able to obtain help from her GP or a psychiatrist. No reasonable person, however, condemns a whole profession because some of its members fail some of the people some of the time.

Dr Snaith clearly wanted to obtain publicity for the Marcé Society by his letter. It is unfortunate that he had to criticise another profession to do so. Despite this, however, I am sure that health visitors, who are always