and rate of rise of melanoma mortality are known. One group is then subjected to educational material to which the control group is not exposed and changing patterns of presentation and mortality compared in the two groups. Although this is what was originally planned in Scotland, it was quickly apparent that television was by far the most powerful educational medium, and television channels are relatively unrestricted in their distribution. Furthermore, a story regarded as newsworthy in one area is quickly publicised by television, radio, and newspapers in adjacent areas. In addition, organisers of campaigns such as this have no control over the spread of information in the secondary wave of publicity in women's magazines, etc. Thus, although not ideal, it was necessary to accept the compromise of changes in thickness and mortality in the intervention group before and after intervention as a reasonable measure of efficacy.

Conclusion

In conclusion, audit of the west of Scotland melanoma education campaign shows encouraging evidence that for women the appropriate population has been targeted and patients are now attending for treatment with thinner primary melanomas. The number of thick melanomas diagnosed in women and the melanoma related mortality in women both showed a downward trend. Alternative approaches seem to be needed to achieve a similar result in men.

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1 MacKie RM, Aitchison TC, Hunter JAA, Hole D, McLaren K, Rankine R, *et al. Malignant melanoma in Scotland 1979-89. Lancet* (in press).
2 Doherty VR, MacKie RM. Reasons for poor prognosis in British patients with cutaneous malignant melanoma. *BMJ* 1986;**292**:987-9.
3 Doherty VR, MacKie RM. Experience of a public education programme on early detection of cutaneous malignant melanoma. *BMJ* 1988;**297**:388-91.
4 Cox DR. *The analysis of binary data.* London: Methuen, 1970.
5 Smith AJ. The Queensland melanoma project. An exercise in health education. *BMJ* 1979;i:253-4.
6 McLeod GR. Control of melanoma in high-risk populations. In: Elwood JM, ed. *Melanoma and naevi.* Vol 9. *Pigment cell.* Basel: Karger, 1988:131-9.
7 Field SI. Melanoma/skin cancer screening in Michigan. *J Am Acad Dermatol* 1987;**16**:578-83.

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Randomised controlled trial of cardioprotective diet in patients with recent acute myocardial infarction: results of one year follow up

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Abstract

***Objective*—To test whether a fat reduced diet rich in soluble dietary fibre, antioxidant vitamins, and minerals reduces complications and mortality after acute myocardial infarction.**

***Design*—Randomised, single blind, controlled trial.**

***Setting*—Primary and secondary care research centre for patients with myocardial infarction.**

***Subjects*—505 patients with suspected acute myocardial infarction. Those with definite or possible acute myocardial infarction and unstable angina based on World Health Organisation criteria were assigned to diet A (n=204) or diet B (n=202) within 24-48 hours of infarction.**

***Interventions*—Both groups were advised to follow a fat reduced diet. Group A was also advised to eat more fruit, vegetables, nuts, and grain products.**

***Main outcome measures*—Mortality from cardiac disease and other causes. Serum lipid concentrations and compliance with diet.**

***Results*—Blood lipoprotein concentrations and body weight fell significantly in patients in group A compared with those in group B (cholesterol fell by 0·74 mmol/l in group A *v* 0·32 mmol/l in group B, 95% confidence interval of difference 0·14 to 0·70, and weight by 7·1 *v* 3·0 kg, 0·52 to 7·68). The incidence of cardiac events was significantly lower in group A than group B (50 *v* 82 patients, p<0·001). Group A also had lower total mortality (21 *v* 38 died, p<0·01) than group B.**

***Conclusions*—Comprehensive dietary changes in conjunction with weight loss immediately after acute myocardial infarction may modulate blood lipoproteins and significantly reduce complications and mortality after one year.**

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Introduction

Epidemiological studies show that diet has a definite correlation with coronary heart disease[1] and that the risks associated with a high fat diet remain even after the occurrence of disease and acute myocardial infarction.[2] There is evidence that hypercholesterolaemia can inhibit the secretion of endothelial dependent relaxant factor[3] and predisposes to thrombosis and atherosclerosis.[4] Reduction in blood lipid concentration has been shown to be associated with a significant decrease in the rate of non-fatal infarctions.[5-8] Recently, patients having an increased intake of fish after acute myocardial infarction were shown to have a 29% decrease in all cause mortality compared with the control group after two years' follow up.[9] There is consistent evidence that common Indian foods, such as onion; garlic; guava; star gooseberry; fenugreek seeds; mushrooms; black, red, and Bengal grams; trichosanthes; bitter gourd; soya beans; ground nut, soya bean, and sunflower oils; and almonds and walnuts can modulate blood concentrations of lipids and glucose and fibrinolytic activity leading to reductions in atherosclerosis.[10-18] The clinical effects of diet rich in these foods may be the same as that of taking 180 mg aspirin daily.[19] We have previously shown that a diet rich in fibre, antioxidant vitamins, and minerals given to patients with acute myocardial infarction significantly reduced cardiovascular events over six weeks.[20] We report the results of a one year follow up of patients in this experiment.

Patients and methods

Details of methods, baseline findings, and design of the study have been described.[20] In brief, all patients admitted to our centre with a clinical diagnosis of suspected myocardial infarction (n=505) within the past 24 hours were considered for the study. Diagnosis of acute myocardial infarction was based on World Health Organisation criteria.[21] Patients were included if they had acute myocardial infarction, possible acute myocardial infarction, or unstable angina pectoris. Exclusion criteria were disliking the intervention

diet (12 patients); presence of cancer, diarrhoea, or dysentery and blood urea concentration more than 400 mg/l (25); death before randomisation (20); and non-cardiac chest pain (42). Patients were kept under surveillance for 24 hours after the clinical diagnosis of acute myocardial infarction, during which time dietary intake, drug treatment, and complications were recorded. Clinical, electrocardiographic, radiological, and biochemical data were also obtained on the patients during the first 24 hours of surveillance. Details of blood pressure measurement and criteria for presence of risk factors have been described.[20] All participating patients gave written informed consent.

STUDY DESIGN

Patients with suspected clinical diagnosis of acute myocardial infarction were individually randomised by the dietitian and pharmacists and assigned a diet by blindly selecting a precoded sequence of cards designated diet A or diet B from a stack with an equal number in each. The doctor was blind to the assigned diet. All the patients remained in the hospital for 10-30 days. In both diets meat, eggs, hydrogenated oils, butter, and clarified butter were replaced with vegetarian meat substitutes and soya bean, sunflower, and ground nut oils so as to provide a prudent diet reflecting the recommendations of the American Heart Association. Group A patients were also advised to eat fruit, vegetables, pulses, nuts, and fish.[20] The goal was to provide at least 400 g/day of fruits and vegetables. These foods are known for their high contents of soluble dietary fibre; free radical inhibitors vitamins A, C, E, and carotene; and minerals selenium copper, etc.[11,22] In both groups, patients had a mainly vegetarian diet, eating eggs 4-5 times a week and meat 1-2 times a week. Other health related advice, such as stopping smoking, reducing alcohol intake, counselling to relieve mental stress and on physical activity, was given to both groups. However, though patients in group A had the advice regularly reinforced, those in group B were left to usual care after the initial advice. The dietitian giving the advice was not blinded. Dietary intake before admission to hospital was estimated in both groups by taking a detailed history of prestudy food intake from spouses.

All the patients completed records of symptoms of angina pectoris, including daily doses of trinitrate and other drugs, and also dietary diaries with the help of the dietitian on days 3, 6, and 10 after infarction and then every week for six weeks and finally every one to 12 weeks. Dietary compliance was checked and reinforced daily during admission to hospital (10-30 days) by a dietitian and later one to 12 weekly, depending on the seriousness of the infarction and frequency of clinic visits, by showing food models and food portions and asking probing questions on diet. Each patient was counselled regularly at separate counselling sessions and asked to complete a questionnaire about dietary intake, smoking, alcohol intake, and drug intake to enforce drug and dietary compliance and to stop or reduce smoking and alcohol intake in both groups. Drug compliance was checked by counting tablets. Nutrient intakes were calculated from Indian food composition tables[22] based on the results questionnaires (and weight of fruits and vegetable) completed at one year. Body weights after removing clothes and shoes were recorded by the pharmacist to the nearest 0·1 kg.

DATA COLLECTION

Clinical data, complications, drug treatment, morbidity, and mortality were regularly recorded for one year by the doctor blinded to dietary regimens. Blood pressure, heart rate, and a 12 lead electrocardiogram were recorded at frequent intervals and whenever indicated at a visit to clinic. Angina pectoris and arrhythmias were diagnosed based on criteria described earlier.[20] Heart failure, left ventricular strain, heart enlargement, and diagnosis of cardiovascular events was based on criteria described earlier.[20,21]

Information from the questionnaires was quantified by a formula. A total score of 1 indicated 100% adherence to the recommended changes in the diet, smoking, alcohol intake, and weight and O indicated no adherence. Patients who consumed more than our minimum recommendation of 400 g/day of fruit and vegetables scored greater than 1. The scores were multiplied by 100 to convert into percentages.

Biochemical data were obtained at entry to study, after six weeks, and then after one year in all the participants. A venous blood sample was drawn in a fasting state and analysed for blood counts and haemoglobin, urea, glucose, cardiac enzyme, total cholesterol,[23] high and low density lipoprotein cholesterol, triglyceride,[24] and electrolyte concentrations. High density lipoprotein cholesterol was measured after precipitation of very low density lipoprotein and low density lipoprotein by an enzymatic method.[23] In all patients with higher blood lipid concentrations measurements were repeated to confirm the results. Cardiac enzymes were assayed as reported earlier.[20]

Data were analysed on the basis of intention to treat, and in all outcome analyses during follow up last available clinical or laboratory data were incorporated for patients lost to follow up or who had died. Those patients admitted with chest pain who showed no signs of definite or possible acute myocardial infarction but who had unstable angina pectoris were also included in the analysis. Only p values $<0{\cdot}05$ by the two tailed test were considered significant. We used Student's t test and the two sample t test to compare changes in the two groups and the Z score test to compare proportions.

Results

Over three years 505 patients with suspected acute myocardial infarction were considered for entry to the study, and 406 of them were randomly assigned to diet A or diet B. Details of baseline findings have been described.[20] Of the 406 patients who entered the trial, 204 (mean age 50·5 (SD 9·3) years, body mass index 24·3 (1·1) kg/m^2) received diet A (cardiovasoprotective) and 202 (mean age 52·0 (8·3) years, body mass index 23·3 (0·8) kg/m^2) diet B. At entry to the study risk factors such as male sex (180 (88%) group A *v* 185 (92%) group B), mean blood pressures (132·5/85·3 *v* 134·2/88·4 mm Hg), and lipoprotein concentrations, were comparable in the two groups. There were slightly more smokers (74 (36%) *v* 70 (35%)) and a slightly lower alcohol intake (45 (22·0%) *v* 50 (24·7%) g/day) in group A than in group B. Sites of acute myocardial infarction as determined by electrocardiography, and initial cardiac enzymes concentrations were similar in both groups. Drug treatment, which included daily doses of propranolol 40-240 mg (95 (46%) *v* 88 (44%) patients), verapamil 20-60 mg (50 (25%) *v* 55 (27%)), nitrates 60-180 mg (200 (98%) *v* 196 (97%)), frusemide 20-80 mg (29 (14%) *v* 36 (18%)), and aspirin 175-355 mg (185 (91%) *v* 197 (98%)), was not significantly different in the two groups. All the patients entered the trial within 24 to 48 hours of an acute myocardial infarction. The numbers of patients with a diagnosis of possible acute myocardial infarction (28 (14%) *v* 36 (18%)) and unstable angina (10 (5%) *v* 9 (4%)) were also comparable between the two groups. Most patients had left hospital by the 11th day in both group A (165 (81%)) and group B (168 (83%)).

Although the nutrient intakes before entry were similar in the two groups, after one year patients in group A received a significantly higher precentage of energy from vegetable proteins; complex carbohy-

drates; polyunsaturated fatty acids; fibre in the form of fruits, vegetables, nuts; fish; and soya bean, sunflower, and ground nut oils. They also received less total energy during the first six weeks after acute myocardial infarction and decreased cholesterol and saturated fatty acids during one year follow up than did group B. Adherence to the diet and health recommendations was significantly higher in group A than group B (table I). In group B the nutrient intakes before entry to the study were similar to those at one year follow up. However, after eight weeks they received a higher polyunsaturated to saturated fat ratio and lower cholesterol intake than before entry (tables I, II).

Though both group A and group B received a fat modified diet, at eight weeks group A also consumed significantly more fruits and vegetables. After 24 and 36 weeks group B had increased their intake of cholesterol and saturated fat while group A maintained excellent compliance with the diet (tables I and II). Of 406 patients initially randomised, six in group A and five in group B were lost to follow up. Of the remaining patients, 52 in group A and 58 in group B missed their follow up visits after discharge from the hospital but were later recovered to the study during clinic visits. Patients in group A who had missed follow up appointments showed substantial decreases in blood lipid concentration and weight, suggesting that they had been following the dietary advice. Side effects of the cardiovasoprotective A diet were mild belching and fullness in a few patients.

Blood lipids and glucose concentrations—Although lipid concentrations before the trial were similar in both groups, at one year there was a significant decrease in blood lipids. In both groups most patients (80·2%) had serum cholesterol concentrations of 4·64-7·02 mmol/l (table III).

Body weight—Initial body weights were (66·6 (SD 10·5), range 52-84 kg) in group A and (65·3 (8·8), 52-83 kg) in group B at entry to the study. After one year weight had fallen by 6·3 kg in group A (to 59 (7·7), 51-73 kg) and by 2·4 kg in group B (to 64·2 (10·3), 52-76 kg). In group A 117 patients lost more than 0·5 kg compared with 32 patients in group B. Within group A a separate analysis of data on the 117 patients who lost more than 0·5 kg showed a more significant change in blood lipoprotein concentrations compared with changes in the other patients (table IV).

Smoking—Smoking was completely stopped in both the groups during admission to hospital. However, after discharge from the hospital 24 out of 72 smokers in group A and 18 out of 70 in group B started smoking again (4-10 cigarettes/day) during the one year follow up.

Complications and cardiac events—The rate of complications was significantly lower in group A than group B and was lowest among those in group A who had lost more than 0·5 kg after one year of follow up (table V). Cardiac events (non-fatal acute myocardial infarction, fatal acute myocardial infarction, and sudden cardiac deaths) occurred significantly less often

TABLE I—*Energy and nutrient intakes before entry and one year after entry in patients receiving cardiovasoprotective diet A and diet B. Values are means (SDs)*

Nutrient intakes	Before entry		At 1 year follow up		
	Group A (n=204)	Group B (n=202)	Group A (n=204)†	Group B (n=202)†	p Value*
Total energy (kJ/day)	8828 (732)	9010 (780)	7581 (520)	8116 (669)	<0·001
Fruit and vegetable intake (g/day)	172 (23·5)	165 (18·8)	575 (91·4)	185 (25·4)	<0·001
% Energy from:					
Carbohydrate	58·5 (0·91)	59·0 (0·88)	62·8 (0·92)	56·8 (0·86)	<0·001
Complex	42·0 (0·63)	40·8 (0·63)	54·0 (0·89)	39·4 (0·63)	<0·001
Refined	16·5 (0·38)	18·2 (0·38)	8·8 (0·28)	17·4 (0·38)	<0·001
Protein	15·5 (0·63)	16·9 (0·63)	13·4 (0·43)	15·2 (0·41)	<0·001
Vegetable or fish	6·0 (0·31)	6·5 (0·31)	9·6 (0·39)	6·2 (0·24)	<0·001
Animal or milk	9·5 (0·36)	10·4 (0·36)	3·8 (0·18)	9·0 (0·35)	<0·001
Fatty acids	26·0 (0·53)	24·1 (0·52)	23·8 (0·49)	28·0 (0·49)	<0·001
Saturated	10·0 (0·39)	10·0 (0·38)	7·2 (0·24)	10·8 (0·36)	<0·001
Monounsaturated	9·3 (0·38)	7·6 (0·36)	8·0 (0·30)	10·2 (0·38)	<0·001
Polyunsaturated	6·7 (0·30)	6·5 (0·30)	8·6 (0·30)	7·0 (0·26)	<0·001
Ratio of polyunsaturated: saturated fat	0·67 (0·04)	0·65 (0·04)	1·19 (0·08)	0·64 (0·03)	<0·001
Cholesterol (mg/day)	300·0 (48·6)	310·0 (54·2)	146·8 (34·0)	286·6 (39·8)	<0·001
Fibre (g/day)	27·0 (10·5)	25·1 (10·1)	52·0 (14·8)	24·6 (9·8)	<0·001
Cereal	16·5 (4·1)	14·8 (3·2)	10·2 (4·8)	12·2 (4·4)	<0·001
Vegetable or fruit	10·5 (2·5)	10·3 (2·6)	41·8 (14·2)	12·4 (3·6)	<0·001
Alcohol (g/week)	44·4 (16·4)	42·1 (13·8)	26·0 (6·0)	34·3 (10·0)	<0·001
Caffeine (mg/day)	145·4 (19·5)	148·2 (21·5)	38·8 (14·8)	118·8 (20·8)	<0·001
Salt (g/day)	7·6 (1·2)	8·2 (1·4)	5·8 (0·96)	5·8 (0·88)	
Vitamins and minerals:					
Ascorbic acid (mg/day)	122·4 (17·2)	125·2 (19·5)	440·6 (100·6)	160·8 (38·0)	<0·001
Vitamin E (mg/day)	6·7 (1·8)	6·8 (1·8)	10·8 (2·5)	7·5 (2·0)	<0·001
Magnesium (mg/day)	310·4 (88·4)	305·0 (82·5)	980·0 (196·0)	410·6 (102·0)	<0·001
Potassium (mg/day)	1105 (205)	1114 (212)	2860 (550)	1206·5 (224·8)	<0·001
Copper (mg/day)	2·0 (0·51)	2·1 (0·51)	6·4 (0·71)	2·3 (0·51)	<0·001
Selenium (μg/day)	76·2 (18·2)	71·2 (17·1)	108·5 (30·6)	78·8 (20·5)	<0·001
Chromium (μg/day)	81·1 (15·2)	78·5 (14·1)	122·6 (24·6)	86·6 (17·5)	<0·001
Carotene (μg/day)	1502 (252)	1498 (245)	3212·2 (312)	1672·5 (278)	<0·001
Vitamin A	465 (48·2)	472 (50·1)	751·0 (165·5)	485·4 (54·5)	<0·001
Total adherence score (%)	65·2 (17·2)	63·0 (14·8)	123·0 (30·0)	71·0 (20·0)	<0·001

*Statistical significance by Student's *t* test for comparison of group A and B after one year.
†At one year dietary intakes data were available for 177 patients in group A and 159 in group B.

TABLE II—*Effect of dietary advice on daily nutrient intakes at different periods of follow up. Values are means (SDs)*

Nutrient intakes	8 weeks		24 weeks		36 weeks	
	Group A	Group B	Group A	Group B	Group A	Group B
% Energy from fat	23·9 (0·59)	28·0 (0·59)	24·5 (0·61)	28·4 (0·62)	24·2 (0·60)	29·0 (0·62)
Polyunsaturated: saturated ratio	1·17 (0·03)	0·89 (0·03)	1·14 (0·03)	0·83 (0·03)	1·16 (0·03)**	0·71 (0·03)
Cholesterol (mg/day)	186·8 (48·6)	216·6 (59·6)	160·5 (36·2)**	242·3 (68·4)	166 (43·4)**	275·4 (55·4)
Total fibre (g/day)	50·6 (15·6)*	24·6 (9·8)	50·8 (16·2)*	26·2 (10·1)	48·2 (14·2)	23·2 (8·2)
Fruit and vegetable (g/day)	582 (98·8)*	180 (28·6)	590 (95·4)*	190 (30·1)	575 (88·5)*	186 (25·6)
Adherence score (%)	124 (30·6)	70 (20·7)	124 (30·5)	67 (20·5)	121 (30·5)	72 (20·7)

* p<0·001, ** p<0·01 for comparison between groups A and B by Student's *t* test.

TABLE III—*Changes in biochemical variables and risk factors from baseline in patients receiving cardiovasoprotective diet A and fat reducing diet B. Values are means (SDs)*

	Group A		Group B		Difference between groups (95% confidence interval)
	Baseline (n=204)	Changes at 1 year (n=204)†	Baseline (n=202)	Changes at 1 year (n=202)†	
Total cholesterol (mmol/l)	5·83 (1·19)	−0·74	5·91 (1·11)	−0·32	0·42 (0·14 to 0·70)*
Low density lipoprotein cholesterol (mmol/l)	4·39 (0·73)	−0·54	4·31 (0·64)	−0·24	0·30 (0·07 to 0·53)*
High density lipoprotein cholesterol (mmol/l)	1·15 (0·29)	+0·07	1·10 (0·25)	−0·04	0·11 (−0·03 to 2·40)**
Triglyceride (mmol/l)	1·94 (0·29)	−0·29	1·97 (0·26)	−0·12	0·17 (−0·02 to 0·35)*
Fasting blood glucose (mmol/l)	6·94 (0·57)	−1·36	6·80 (0·53)	−0·65	0·71 (0·20 to 1·22)*
Blood urea (mmol/l)	4·03 (0·96)	−0·30	4·33 (1·08)	−0·53	0·23 (−0·06 to 0·52)
Blood pressures (mm Hg):					
Systolic	132·5 (5·4)	−13·4	134·2 (7·5)	−5·2	8·2 (3·0 to 13·1)**
Diastolic	85·3 (2·6)	−9·3	88·4 (4·2)	−3·5	5·8 (2·46 to 9·14)**

* p<0·01, ** p<0·05 by two sample *t* test.
† At one year data were available for 177 patients in group A and 159 in group B.

TABLE IV—*Changes in risk factors compared with baseline in patients following cardiovasoprotective diet A according to weight loss at one year. Values are means (SDs)*

	Loss ≥0·5 kg		Loss <0·5 kg		Difference between groups (95% confidence interval)
	Baseline (n=117)	Change at 1 year (n=117)†	Baseline (n=87)	Change at 1 year (n=87)†	
Total cholesterol (mmol/l)	6·07 (0·87)	−1·05	5·60 (0·73)	−0·47	0·58 (0·06 to 10·90)*
Low density lipoprotein cholesterol (mmol/l)	4·56 (0·73)	−0·81	4·12 (0·57)	−0·39	0·42 (0·04 to 8·0)*
High density lipoprotein cholesterol (mmol/l)	1·14 (0·25)	0·09	1·17 (0·29)	−0·03	0·12 (−0·02 to 0·26)**
Triglyceride (mmol/l)	2·03 (0·24)	−0·35	1·85 (0·19)	−0·15	0·20 (0·04 to 0·36)**
Fasting blood glucose (mmol/l)	7·08 (0·68)	−1·69	6·84 (0·56)	−0·91	0·78 (0·37 to 1·19)**
Blood pressures (mm Hg):					
Systolic	136·4 (7·5)	−18·6	128·5 (9·6)	−8·5	10·1 (2·63 to 17·57)**
Diastolic	86·8 (3·1)	−12·4	84·2 (5·1)	−5·6	6·8 (2·13 to 11·27)**
Body weight (kg)	68·5 (5·4)	−7·1	64·2 (4·2)	−3·0	4·1 (0·52 to 7·68)*
Adherence score (%)	46·0 (11·2)	95·0	48·2 (11·8)	40·0	55·0 (14 to 96)

* p<0·01, ** p<0·05 by two sample *t* test.
† At one year laboratory data were available for 102 patients who lost ≥0·5 kg and 75 who lost <0·5 kg.

TABLE V—*Complications and cardiac events in patients following cardiovasoprotective diet A and fat reducing diet B one year after acute myocardial infarction. Values are numbers (percentages)*

	Group A			Group B	Relative risk (95% confidence interval)
	Loss ≥0·5 kg (n=117)	Loss <0·5 kg (n=87)	All patients (n=204)	(n=202)	
Complications:					
Angina pectoris	6 (5)**	8 (9)	14 (7)***	46 (23)	0·30 (0·15 to 0·45)
Positive result on exercise test	22 (19)***	42 (48)	64 (31)***	128 (63)	0·49 (0·39 to 0·59)
Left ventricular strain	7 (6)***	12 (14)	19 (9)**	34 (16·8)	0·55 (0·32 to 0·79)
Left ventricular hypertrophy	3 (3)*	8 (9)	11 (5)**	24 (17)	0·45 (0·19 to 0·70)
NYHA class III and IV	3 (3)*	5 (6)	8 (4)	14 (7)	0·56 (0·18 to 0·95)
Ventricular ectopics (8/min)	6 (5)***	8 (9)	14 (7)***	42 (21)	0·33 (0·16 to 0·49)
Ventricular ectopics (3 in row)	1 (0·9)	2 (2)	3 (1)***	18 (9)	0·45 (−0·02 to 0·35)
Cardiac events	20 (17)***	30 (34)	50 (25)***	82 (41)	0·60 (0·31 to 0·75)
Non-fatal myocardial infarction	12 (10)***	18 (21)	30 (15)**	48 (24)	0·62 (−0·42 to 0·83)
Fatal myocardial infarction	5 (4)*	8 (9)	13 (6)	19 (9)	0·68 (0·32 to 1·03)
Sudden cardiac death (within 1 hour)	3 (6)*	4 (5)	7 (3)	15 (7)	0·46 (0·12 to 0·29)
Suspected cardiac death				1 (0·5)	
Death due to cancer				1 (0·5)	
Death due to stroke		1 (1)	1 (0·5)	2 (1)	0·49 (0·47 to 1·46)
Total cardiac mortality	8 (7)*	12 (14)	20 (10)**	34 (17)	0·58 (0·34 to 0·83)
Total mortality	8 (7)***	13 (16)	21 (10)**	38 (19)	0·55 (0·34 to 0·75)
Cardiac events plus other deaths	20 (17)***	31 (36)	51 (25)***	86 (43)	0·59 (0·46 to 0·74)

* p<0·05, ** p<0·01, *** p<0·001, obtained by Z test.
NYHA = New York Heart Association.

in group A than group B (50 *v* 82, p<0·001), and total mortality was also lower in group A (21 *v* 38 deaths, p<0·01). Within intervention group A the proportion of patients with cardiovascular events was significantly less in the subset of 117 patients who lost ≥0·5 kg compared with those who lost <0·5 kg (20 *v* 30, p<0·05); total mortality in these two subsets was not significantly different.

Discussion

Since most deaths after acute myocardial infarction occur during admission to hospital it may be more beneficial to start the diet immediately after suspicion of an attack. Our study showed that a diet with a high fibre, mineral vitamin, and numerial content associated with weight reduction (tables I-III) initiated within 72 hours of an acute myocardial infarction significantly decreased total mortality from cardiac disease (p<0·01) and all causes (p<0·01) over one year. There was a significant decrease in total cardiac events, including non-fatal acute myocardial infarction (p<0·01) in patients taking a cardiovasoprotective diet compared with patients taking a fat reduced diet (50 *v* 82, p<0·001) (table V). Underlying these beneficial effects patients in group A had significant decreases in blood lipoprotein and fasting blood glucose concentrations in association with a higher total adherence score compared with group B (tables III and IV). Patients in group A who lost more than 0·5 kg and also had higher adherence scores showed greater changes in blood lipid concentration and had fewer cardiac events compared with patients who had lost less than 0·5 kg in group A (p<0·05) and patients in group B (p<0·001; table IV and V). The strong relation between adherence to the intervention programme, lipid changes, and cardiac events indicates that the relation is causal because those who made the biggest changes showed highest pro-

tection from coronary events.[7 8] Weight reduction seemed to have an independent beneficial effect.

Several randomised trials have been published in which a low fat diet or one with a high polyunsaturated to saturated fat ratio was given to subjects who had had an acute myocardial infarction.[6 8 25-27] All the published trials contained less than 500 subjects. Despite a beneficial influence on reinfarction, none showed any reduction in deaths. In contrast with our study in these studies intervention was not initiated until six to eight weeks after acute myocardial infarction, and the diets did not aim at increasing intake of water soluble dietary fibre, antioxidant vitamins, and minerals.[6 8 9] There is epidemiological[28 29] and experimental evidence that soluble dietary fibre, vitamins, and minerals may protect against coronary artery disease by decreasing blood lipids concentrations as well as by inhibiting formation of lipid peroxides, which damage myocardial arterial cells.[11 28] Both of these mechanisms are also known to decrease free radical generation, decrease cell acidosis, inhibit sodium and calcium ion influx into the cell, and prevent structural and functional disorders of cell.[4 11] Recently diets rich in fruit and vegetables have been advised by the WHO and United States Department of Health and Human Services for prevention of chronic diseases.[30 31]

Restriction of energy intake can reduce atherosclerosis and coronary deaths[32] and weight reduction may be associated with reduction in coronary artery disease and all of its risk factors.[2 33] Weight reduction may also reduce cardiac enlargement, left ventricular strain, postexercise electrocardiographic changes, and arrhythmias,[2] possibly by reducing myocardial oxygen requirement and having other beneficial effects on cardiac indices.[33] Thus the decrease in complications in the intervention group may be due to weight loss as well as to lipid changes. A decreased intake of clarified butter,[34] a decrease in hyperinsulinaemia due to dietary changes, and a reduction in obesity induced coronary disease (as observed in Indian migrants[35]) are other possible causes for the reduction in cardiac events.

In dietary trials the issue of compliance to advice is a problem because patients in the intervention group may not eat exactly what they are advised. The control group may also follow some intervention programme. Monitoring of dietary compliance through questionnaires is again open to bias because subjects may have simply learnt to answer what the dietitian wants to hear. However, the seriousness of illness in acute myocardial infarction tends to motivate the patients to eat and follow what they are advised and respond truthfully to questions.

In conclusion, we have shown that a diet in which fruits, vegetables, cereals, nuts, and oil substitute for clarified butter, eggs, and meat together with weight reduction can significantly reduce the number of cardiac events and all cause mortality. An additional finding is the significant decrease in non-fatal acute myocardial infarction in the intervention group compared with the control group. A longer follow up would be necessary to confirm these observations.

1 Shakelle RB, Stamler J. Dietary cholesterol and ischaemic heart disease. *Lancet* 1989;i:1177-9.

2 Wong ND, Cupples A, Ostfeld AM, Levy D, Kannel WB. Risk factors for longterm coronary prognosis after initial myocardial infarction: The Framingham Study. *Am J Epidemiol* 1989;**130**:469-80.

3 Andrews RE, Bruckotorfer KR, Dunn RC, Jacobs M. Low density lipoproteins inhibit endothelium dependent relaxation in a rabbit aorta. *Nature* 1987;**327**: 237-9.

4 Horrobin DF, Huang YS. The role of linoleic acid and its metabolite in the lowering of plasma cholesterol and the prevention of cardiovascular disease. *Int J Cardiol* 1987;**17**:241-55.

5 Lamm G. Risk factor intervention and prognosis after acute myocardial infarction. *Cardiovascular Drug Therapy* 1988;**2**:133-7.

6 Leren P. Prevention of coronary heart disease some results from the Oslo secondary and primary intervention studies. *J Am Coll Nutr* 1989;**8**:407-10.

7 Lipid Research Clinics Programme. The lipid research clinic primary prevention results: reduction in incidence of coronary heart disease. *JAMA* 1984;**251**:351-64.

8 Research Committee. Low fat diet in myocardial infarction: a controlled trial. *Lancet* 1965;ii:501-4.

9 Burr ML, Fehily AM, Gilbert JF, Rogers S, Holiday RM, Sweetnam PM, *et al*. Effects of changes in fat, fish, and fibre intakes on death and myocardial reinfarction: diet and reinfarction trial (DART). *Lancet* 1989;ii:757-61.

10 Arntzenius AC, Kromhout D, Bartn JE, Reiber JHC, Bruschke AVG, Buis Van Gent CM, *et al*. Diet. Lipoproteins and progression of coronary atherosclerosis. The Leiden intervention trial. *N Engl J Med* 1985;**312**: 805-8.

11 Singh RB, Mori H, Kummerow FA, Kokatnoor M. *Nutrition in coronary heart disease*, Moradabad, India: ICN monograph, 1991.

12 Sharma RD. Hypocholesterolemic agents. *Proceedings of the 21st annual meeting of Nutrition Society of India, Nov 24-26, 1988*. Hyderabad: Nutrition Society of India, 1989:52-6.

13 Kurup PA. Nutritional factors and atherosclerosis. *Proceedings of 21st annual meeting of Nutrition Society of India, Nov 24-26, 1988*, Hyderabad: Nutrition Society of India, 1989:27-37.

14 Singh RB, Verma R, Mehta PJ, Laxmi B, Garg V. Nutritional intervention in acute myocardial infarction. *J Nutr Med* 1990;**1**:179-86.

15 Sainani GS, Desai DB, More KN. Onion, garlic, and atherosclerosis. *Lancet* 1976;ii:575-8.

16 Menon S, Kendal RV, Dewar HA, Newell DJ. Effect of onion on blood fibrinolytic activity. *BMJ* 1968;**3**:351-3.

17 Boulin DJ. Garlic as a platelet inhibitor. *Lancet* 1981;i:776.

18 Mathur KS, Khan MA, Sharma RD. Hypocholesterolaemic effect of Bengal gram in man. *BMJ* 1968;i:30-1.

19 Singh RB, Verma R, Mehta PJ, Rastogi SS. The effect of diet and aspirin on patient outcome after myocardial infarction. *Nutrition* 1991;**7**:119-23.

20 Singh RB, Rastogi SS, Verma R, Bolaki L, Singh R, Ghosh S. An Indian experiment with nutritional modulation in acute myocardial infarction. *Am J Cardiol* 1992;**69**:879-85.

21 World Health Organisation. *Myocardial infarction community register*. Copenhagen: WHO, 1976.

22 Narsinga Rao BS, Deostnale YG, Pant KC: *Nutrient composition of Indian foods*. Hyderabad: National Institute of Nutrition, 1989.

23 Wilson DE, Spiger MJ. A dual precipitation method for quantitative plasma lipoproteins measurement without ultracentrifugation. *J Lab Clin Med* 1973;**82**:413-82.

24 Van Handel E, Zilversmit BD. Micromethod in the direct estimation of serum triglycerides. *J Lab Clin Med* 1957;**60**:152-9.

25 Rose GA, Thompson WB, William RT. Corn oil in treatment of ischaemic heart disease. *BMJ* 1965;i:1531-3.

26 Medical Research Council Research Committee. Controlled trial of soya bean oil in myocardial infarction. *Lancet* 1968;ii:693-700.

27 Woodhill JM, Palmer AJ, Leelarthalpin B, McGilchrist C, Blacket RB. Low fat, low cholesterol diet in secondary prevention coronary heart disease. *Adv Exp Med Biol* 1978;**109**:317-30.

28 Riemersma RA. Risk of angina pectoris and plasma concentrations vitamin A, C, E and carotene. *Lancet* 1991;**357**:1-5.

29 Khaw KT, Barret-Conner E. Dietary fibre and reduced coronary heart disease mortality rates in men and women, a 12 year prospective study. *Am J Epidemiol* 1987;**126**:1093-102.

30 World Health Organization Expert Committee on Diet. *Nutrition and prevention of chronic diseases: report of a WHO study group*. Geneva: WHO (in press).

31 United States Departments of Agriculture and Health and Human Services. *Revised dietary guidelines for Americans*. 3rd ed. Washington, DC: US Department of Agriculture, 1991.

32 Schettler G. Cardiovascular diseases during and after World War II: a comparison of Federal Republic of Germany with other European countries. *Prev Med* 1979;**8**:581-90.

33 Bagatell CA, Heymafield SB. Effect of meal size on myocardial oxygen requirements: implications for postmyocardial infarction diets. *Am J Clin Nutr* 1984;**39**:421-6.

34 Jacobson MS. Cholesterol oxides in Indian ghee: possible cause of unexplained high risk of atherosclerosis in Indian immigrant populations. *Lancet* 1987;ii:656-8.

35 McKeigue PM, Shah B, Marmot MG. Relation of central obesity and insulin resistance with high diabetes prevalence and cardiovascular risk in South Asians. *Lancet* 1991;**337**:382-6.

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