LOW-FAT DIET IN MYOCARDIAL INFARCTION

A Controlled Trial

BY A RESEARCH COMMITTEE*

From the Central Middlesex, Edgware General, West Middlesex, and St. George's Hospitals, London

CORONARY-Artery disease is widely held to be connected with diet. Although diets low in saturated fats or high in polyunsaturated fats reduce the raised blood-cholesterol that is often present in this disease, it has not yet been established that such diets reduce the risk of recurrences.

To obtain evidence on this question, a controlled trial of a low-fat diet was carried out on a group of men with coronary heart-disease living in North-west London.

Clinical Material

264 men, under the age of sixty-five, who had recently recovered from a first myocardial infarction and who had been in the Central Middlesex, Edgware General, or West Middlesex hospitals took part in the trial. On leaving hospital they were allocated at random to one of two groups at each hospital. One group was placed on a low-fat diet, while the control group continued with their normal diet.

The trial, which ran from 1957 to 1963, was managed by four research medical registrars working at the three different hospitals. Three dietitians controlled the diets.

A diagnosis of myocardial infarction was accepted on the following evidence:

1. Cardiac pain with abnormal Q waves in the electrocardiogram (E.C.G.).
2. Cardiac pain for one hour or more with s-T changes on the E.C.G., and associated changes (raised serum-enzymes, raised white-cell-count, raised erythrocyte-sedimentation-rate, or pyrexia).

There were 32 patients who had chest pain at rest and s-T changes without laboratory evidence of infarction. These patients were described as having "acute ischaemic episodes", and are included in the 264, but have also been analysed separately.

Only patients who had had their attack within the previous three months were included; most were admitted to the trial after one month in hospital.

Patients on long-term anticoagulants were excluded. 29 patients received no anticoagulants, while the rest had anticoagulants for periods of up to fourteen weeks.

Patients with syphilis, diabetes, myxoedema, severe hypertension, notable cardiac enlargement or persisting heart-failure, and patients who could not cooperate were excluded from the trial. 12 patients admitted to the trial were later rejected because long-term anticoagulants were needed, because they relapsed before starting the diet, or because they did not cooperate. 252 patients remained for final analysis.

The mean follow-up time to first relapse or end of trial was 3-04 years for the low-fat group, and 3-05 years for the control group.

*Dietary Regimen

Patients in the diet group were allowed to take 40 g. fat daily. The daily allowance included 14 g. (½ oz.) butter, 84 g. (3 oz.) of meat, 1 egg, 56 g. (2 oz.) cottage cheese, and skimmed milk. The nature of the fat consumed was not altered, nor were any additional unsaturated fats given. The diet was often unpleasant, and where possible, it was modified to suit individual tastes. The main objections were to the skimmed milk, to the small butter ration which was especially hard on those who took sandwiches to work, and to the restriction on biscuits and cakes.

Those patients who were overweight were given reducing diets, irrespective of their group. In the control group this was done so far as possible by reducing carbohydrates rather than fats. 21% of the control patients and 15% of the diet group were advised to reduce, but apart from this advice control patients were told that no alteration in diet habits was needed. Nevertheless some did reduce dietary fat and a few used vegetable oil for frying.

The object of the trial was to see whether a low-fat diet in unselected patients under ordinary working conditions (as opposed to selected volunteers in a metabolic ward) could produce any clinical effect.

Method

To determine how far patients kept to their prescribed regimes, the following dietary controls were applied:

(1) Patients in both groups were asked to record the weight of all food consumed on a different day each week for the first seven weeks after admission to the trial, and thereafter on the first day of each month (these records being used as the basis of the dietary supervision and analysis).

(2) An independent dietitian interviewed a number of patients in each group.

The patient and his wife were interviewed by the doctor and dietitian at their first outpatient visit, about two weeks after leaving hospital. The patient then attended every two weeks for three months, every three months for two years, and every six months thereafter. At each visit a note was made about any further chest pain. The serum-cholesterol was estimated at each visit. A further E.C.G. was taken yearly. The diet records were checked by the dietitian at each visit and any problems about his diet were discussed.

Assessment of Relapse

Progress of the patients was assessed on two main criteria—reinfarction, and death from coronary heart-disease. Since, after reinfarction, a change in treatment was sometimes advised by the physician in charge, only the first relapse (including death) was counted in the assessment. The total mortality of the two groups during the period of observation has been analysed separately.

The definition of a relapse in an individual case is often very difficult, and for this reason it was made by one physician (K. P. B.) who did not know the dietary group of the patient concerned. Doubtful cases were discussed with a second physician (P. M. McAlen.). Difficulties often arose in patients having further attacks of chest pain without E.C.G. changes or laboratory evidence of infarction. By reviewing them "blind," the possibility of bias was removed.

The criteria used for relapse are listed in the appendix, and are being used in a current trial on the effect of a polyunsaturated fatty-acid diet on men with myocardial infarction.

*The members of the committee were: Dr. K. P. BALL (chairman), Central Middlesex Hospital, London, N.W.10; Dr. EDDA HANINGTON; Dr. P. M. MCALLEN; Dr. T. R. E. PILKINGTON; Dr. J. M. RICHARDS; Dr. D. E. SHARLAND (secretary); Dr. G. S. C. SOWRY; Dr. PATRICIA WILKINSON; and Mrs. J. A. C. CLARKE, Miss C. MURLAND, and Mrs. J. WOOD (dietitians).
Results
Evidence of Successful Randomisation

To assess whether the two diet groups were similar on admission to the trial they were analysed with respect to age, weight, social class, activity of work, duration of previous angina, severity of first attack, and the duration of anticoagulant treatment with method of its termination. Only two features require comment:

Weight—The mean weight of the controls was 166 lb., against 161 lb. for the low-fat group at the onset; but this difference does not quite reach the usual level of significance (0·1 > p > 0·05).

Stopping anticoagulants—This was more abrupt in the control group (83 were stopped within three days) than in the low-fat group (54 within this period). Among these 137 patients there were 17 relapses in the first six months compared with 16 relapses in 83 patients tailed off over four to forty-two days. Stopping anticoagulants abruptly did not predispose patients to relapse.

Changes in Serum-cholesterol

Samples were obtained every three months for two years and then every six months, except for one period

<p>| TABLE I—MEAN SERUM-CHOLESTEROL |
| Group | Serum-cholesterol (mg per 100 ml.) at: | |</p>
<table>
<thead>
<tr>
<th></th>
<th>Admission</th>
<th>6 mos.</th>
<th>1 yr.</th>
<th>2 yr.</th>
<th>3 yr.</th>
<th>4 yr.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low-fat diet</td>
<td>266 (90)</td>
<td>223 (71)</td>
<td>221 (63)</td>
<td>221 (61)</td>
<td>216 (54)</td>
<td>216 (22)</td>
</tr>
<tr>
<td>Control</td>
<td>266 (68)</td>
<td>251 (60)</td>
<td>242 (66)</td>
<td>242 (60)</td>
<td>233 (52)</td>
<td>241 (15)</td>
</tr>
</tbody>
</table>

*Figures in parentheses are the number of men whose serum-cholesterol was tested.

in 1958–59, when the results were found to be technically unreliable and had to be rejected (which accounts for the limited number of estimations). No significant difference was found with regard to age, severity of infarct, social class, or activity of work between those who had serum-cholesterol estimations and those who did not. Serum-cholesterol was estimated by the method of Abell et al. (1952).

The average levels for all serum-cholesterol estimations performed are shown in table I and fig. 1.

If only those patients whose cholesterol was estimated on every occasion are included, the picture is similar (table II).

The difference between the initial (baseline) cholesterol levels of the two groups is not significant. By six months, although there was a fall in both groups, there is a clear difference between them. The cholesterol levels in the low-fat group had fallen significantly more than in the control group (0·05 > p > 0·02). By one year, because of a continued fall in the control group without a corresponding fall in the low-fat group, the difference had diminished and was just below the conventional level of significance (0·10 > p > 0·05); the position at two years was the same. Thereafter, the numbers are smaller and trends not so clear, though those on the low-fat diet appear to maintain low levels for four years.

Weight Changes

The patients on the low-fat diet lost more weight than the controls which supports a real difference in their dietary behaviour (table III).

Dietary Analysis

Diets were analysed and the average daily intake of fat and calories were calculated using standard food tables (McCance and Widdowson 1946). Of the 252 men in the trial 25 (10%) died or relapsed before any diet sheets could be completed. Only 6 patients (2·4%) produced no suitable diet sheets. 69% of all the patients had between 80% and 100% of the possible sheets available for analysis.

The calculated total daily fat-intake shows a striking difference between the two groups (see fig. 2 and table IV) being approximately 45 g. per day in the low-fat group and 110–130 g. per day in the control group. The

<p>| TABLE II—SERUM-CHOLESTEROL FOR PATIENTS TESTED ON EVERY OCCASION |
| Group | Serum-cholesterol (mg per 100 ml.) at: |</p>
<table>
<thead>
<tr>
<th></th>
<th>No. tested</th>
<th>Admission</th>
<th>6 mos.</th>
<th>1 yr.</th>
<th>2 yr.</th>
<th>3 yr.</th>
<th>4 yr.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low-fat diet</td>
<td>31</td>
<td>263</td>
<td>224</td>
<td>229</td>
<td>223</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>41</td>
<td>266</td>
<td>253</td>
<td>243</td>
<td>239</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<p>| TABLE III—WEIGHT CHANGES DURING TRIAL |
| Group | Average weight (lb.) at: |</p>
<table>
<thead>
<tr>
<th></th>
<th>6 mos.</th>
<th>1 yr.</th>
<th>2 yr.</th>
<th>3 yr.</th>
<th>4 yr.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low-fat diet</td>
<td>161 (118)</td>
<td>148 (96)</td>
<td>148 (100)</td>
<td>149 (98)</td>
<td>151 (33)</td>
</tr>
<tr>
<td>Control</td>
<td>166 (132)</td>
<td>159 (101)</td>
<td>159 (100)</td>
<td>158 (97)</td>
<td>158 (31)</td>
</tr>
</tbody>
</table>

*Figures in parentheses are numbers of men whose weights were recorded.

The expected weight changes and serum-cholesterol changes during the trial are shown in fig. 3 and table V.

<p>| TABLE IV—AVERAGE DAILY INTAKE OF FAT AND CALORIES |</p>
<table>
<thead>
<tr>
<th>Time after entering trial</th>
<th>Low-fat diet group</th>
<th>Control group</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of patients</td>
<td>Average daily fat (g.)</td>
<td>Average daily calories</td>
</tr>
<tr>
<td>6 mos.</td>
<td>105</td>
<td>44</td>
</tr>
<tr>
<td>1 yr.</td>
<td>81</td>
<td>45</td>
</tr>
<tr>
<td>2 yr.</td>
<td>76</td>
<td>45</td>
</tr>
<tr>
<td>3 yr.</td>
<td>49</td>
<td>43</td>
</tr>
<tr>
<td>4 yr.</td>
<td>21</td>
<td>44</td>
</tr>
<tr>
<td>5 yr.</td>
<td>7</td>
<td>44</td>
</tr>
</tbody>
</table>

Fig. 2—Average daily fat-intake.
average daily caloric-intake also shows a difference (fig. 3 and table iv), being about 1900–2000 calories per day in the low-fat group and 2300–2600 calories per day in the control group. The difference in caloric intake between the two groups is less than would be accounted for by the difference in fat intake and was due to a higher carbohydrate intake in the low-fat group.

TABLE V—MEAN SUGAR AND TOTAL CARBOHYDRATE INTAKE FOR THE TWO GROUPS

<table>
<thead>
<tr>
<th>Patients</th>
<th>No.</th>
<th>Added sugar (g. per day)</th>
<th>Total carbohydrate (g. per day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control: No relapse</td>
<td>85</td>
<td>36</td>
<td>247</td>
</tr>
<tr>
<td>Relapse</td>
<td>36</td>
<td>26</td>
<td>225</td>
</tr>
<tr>
<td>Low-fat diet: No relapse</td>
<td>75</td>
<td>52</td>
<td>295</td>
</tr>
<tr>
<td>Relapse</td>
<td>31</td>
<td>61</td>
<td>300</td>
</tr>
<tr>
<td>Total</td>
<td>227</td>
<td>46</td>
<td>268</td>
</tr>
</tbody>
</table>

*Total available for analysis.

TABLE VI—RELAPSES IN RELATION TO ADDED SUGAR

<table>
<thead>
<tr>
<th>Added sugar (g. per day)</th>
<th>No. of patients</th>
<th>No. of relapses</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–5</td>
<td>58</td>
<td>20 (34%)</td>
</tr>
<tr>
<td>6–32</td>
<td>54</td>
<td>15 (29%)</td>
</tr>
<tr>
<td>33–67</td>
<td>57</td>
<td>14 (25%)</td>
</tr>
<tr>
<td>68–207</td>
<td>58</td>
<td>18 (31%)</td>
</tr>
<tr>
<td>Total</td>
<td>227*</td>
<td>67</td>
</tr>
</tbody>
</table>

*Total available for analysis.

An independent dietitian interviewed 30 unselected patients from the low-fat group and thought that 22 were keeping to the prescribed diet. Of 23 control patients seen, 18 admitted to some reduction of their fat intake because of weight restriction, or the influence of the Press, but the dietary weighings suggest that reduction of fat intake was not large.

From this evidence it is concluded that there was a real difference in the amount of fat consumed by the two groups.

TABLE VII—REINFARCTIONS AND DEATHS

Reinfarctions and deaths | Low-fat diet (123) | Control (129) | Total (252) |
--------------------------|--------------------|---------------|-------------|
Grade of infarction:     |                    |               |             |
Possible                  | 12                 | 10            | 22          |
Probable                  | 4                  | 7             | 11          |
Definite                  | 27                 | 27            | 54          |
Deaths without proven evidence of reinfarction, coronary thrombosis, or relapse | 3 | 4 | 7 |
All relapses               | 46                 | 48            | 94          |
Deaths at first incident   | 10                 | 12            | 22          |
All deaths (from all causes and occurring at a first or later relapse) | 20 | 24 | 44 |

Discussion

The absence of an improved prognosis of the group in this trial taking a low-fat diet contrasts with two American reports. Lyon et al. (1956) described a series of 280
patients who were put on a 50 g. low-fat and low-
cholesterol diet after a myocardial infarction. Although
the 125 patients who did not keep to the diet had a four-
fold recurrence rate compared with those who did, this
trial cannot be considered a fair comparison. Morrison
(1960) studied 100 cases, half of which were put on to a 25 g.
low-fat and low-cholesterol diet with added vegetable
oils and vitamins. The death-rates in 3, 8, and 12
years were 14%, 44%, and 62% in the diet group, and
30%, 76%, and 100% in the control group; but the
numbers were not considered large enough for statistical
evaluation. Our findings are similar to those of Rose et al.
(1965), who found no evidence of clinical benefit to two
groups of patients with ischamic heart-disease put on a
low-fat diet and who were given supplements of either
olive oil or corn oil, compared with a control group of
patients.

In two other long-term trials of patients with myocardial
infarction the serum-cholesterols were lowered with
estrogens. Oliver and Boyd (1961) showed no reduction
in reinfarction or mortality, whereas Stamler et al. (1963)
showed some benefit to the group of patients whom they
thought to have had the worst prognosis.

The reason for the continued fall of the serum-
cholesterol in the control patients of the present trial has
not been fully explained. Rather more of the control
patients were asked to reduce their weight (21%) than
were the diet patients (15%), and there was an average
weight loss of 7-8 lb. in the control group compared with
12-13 lb. in the diet group. No such trend in the control
cholesterol levels was found by Oliver and Boyd (1961) in
their oestrogen trial. Acheson and Hutchinson (1963),
however, noted a fall of cholesterol from 300 to 250 mg.
per 100 ml. in the placebo group of patients in a controlled
trial of 'Atromid'.

It may be considered that fat restriction was not
rigorous enough to produce an adequate fall of cholesterol
compared with the control group. The trial was, however,
carried out on working men, many of whom were in active
jobs. Much closer dietary supervision could not have
been carried out under ordinary clinical conditions.
Despite the difficulties of an unpleasant diet it is believed
that those in the diet group took less than half the amount
of fat compared with the control patients.

Analysis of the amount of added sugar taken showed no
evidence that this factor had any effect on the relapse rate
following infarction.

It is concluded that in men under the age of sixty-five
who have survived a first myocardial infarction, a low-fat
diet does not improve their prognosis.

**Summary**

A controlled trial of a 40 g. low-fat diet was carried out
on 264 men who had survived a first infarction. Despite
a lowering of the blood-cholesterol and a greater fall in
body-weight in the treated group, the relapse-rate was not
significantly different in the two groups.

A low-fat diet has no place in the treatment of myocardial
infarction.

We are very grateful to Prof. J. N. Morris, Dr. J. A. Heady, and
Miss Jean W. Marr, and to the computing staff of the social medicine
research unit of the Medical Research Council, for their continued
help in planning the trial and assessing the results. We are indebted
to the physicians at the Central and West Middlesex Hospitals, and
at the Edgware General Hospital for referring patients to us; to Dr.
Mary Fulton for help with the analysis of dietary sugar; to Miss H.
Kerselman for the serum-cholesterol estimations; and to Mrs. I.
Prentice for the diagrams. The trial was supported by the research
committee of the North West Regional Hospital Board and by the
Medical Research Council.

**References**

Chem. 185, 357.


86, 325.

of a trial of 'Atromid'.


Stamler, J., Pick, R., Katz, L. N., Pick, A., Kaplan, S. M., Berkson, D. M.,


**Appendix**

**Grades of relapse of infarction**

Definite

1. Development of new Q waves, Q waves which develop slowly as a result of the original infarct in patients on anti-
cogulants are not accepted as evidence of relapse.

2. Rest pain for more than one hour with raised serum-
glutamic-oxaloacetic transaminase or ischemic T-wave changes
indicating deterioration.

3. Death with necropsy evidence of recent infarction or
coronary thrombosis.

Probable

1. Rapidly increasing angina suggestive of acute coronary
insufficiency with ischemic t-wave changes indicating deterioration.

2. Rapid onset of heart failure with ischemic t-wave changes indicating deterioration.

3. An episode of rest pain with arrhythmia and persisting
electrocardiograph deterioration.

4. Sudden death with history of recent chest pain or
electrocardiographic changes (but without necropsy).

Possible

1. Rapidly increasing angina without ancillary evidence.

2. Rapid onset of heart failure though to be due to
infarction but without ancillary evidence.

3. History of rest pain for one hour or more without
ancillary evidence.

4. A history of spontaneous rest pain of less than one hour
with persisting electrocardiographic deterioration.

5. Sudden death without necropsy.

In every case surviving a relapse other causes, such as
anemia, must be excluded.

"... Professional standards mean much more than technical
competence. In medicine, as in all the ancient professions,
they mean that your attitude to the job must be the very opposite
of that which is expressed in the current phrases ' couldn't care
less ' and ' near enough is good enough '. Have nothing to do
with those watchwords of decadence. Professional men and
women engage to put their best endeavour into their work, and
recognise that it is their plain duty to do so. That duty springs
out of the personal relationship between doctor and patient.
They are not dealing with each other like merchants in the
market place, where a man must look after himself and caveat
emptor holds the ring. The patient comes to you with all the
knowledge on the one side, and all the need on the other. That
of itself, without more, creates between you a relation of trust
from which all the obligations on either side are naturally
derived. It is this fiduciary relation which is the essential mark
of a true profession, and which determines the attitude of the
professional man or woman both to their patients and to their
work. It rules out from the start anything like strike action or
the withdrawal of professional services as a means of protecting
or advancing your own interests. Conduct of that kind may or
may not be appropriate in industrial relations: in the case of a
professional it is, in plain terms, nothing less than a breach of
trust." — The late Sir Thomas Taylor, Speaking to Graduates: