Two-year results of a controlled study of residential rehabilitation for patients treated with percutaneous transluminal coronary angioplasty

A randomized study of a multifactorial programme

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Aims In a multifactorial lifestyle behaviour programme, of 2 years duration, to study the maintenance of achieved behaviour and risk factor-related changes.

Methods and Results Out of a consecutive population of 151 patients treated with percutaneous transluminal angioplasty under 65 years of age, 87 were randomly allocated to an intervention group (n=46) or to a control group (n=41). The programme started with a 4 week residential stay, which was focused on health education and the achievement of behaviour change. During the first year of follow-up, a maintenance programme included regular contacts with a nurse, while no further rehabilitative efforts were offered during the second year. One patient died (control). During the second year the proportion of hospitalized patients was lower in the intervention group (4% vs 20%; P<0.05). Patients in the intervention group improved several lifestyle dependent behaviours: diet (index at 0, 12 and 24 months): 10·5 ± 3·4, 12·9 ± 2·5 and 12·4 ± 2·6 in the intervention group (I) vs 10·1 ± 3·2, 10·7 ± 3·0 and 11·8 ± 3·2 in the control group (C); P<0.05, exercise sessions per week: 2·5 ± 2·3, 4·5 ± 1·9 and 4·4 ± 2·1 (I) vs 3·1 ± 2·2, 3·5 ± 2·3 and 3·7 ± 2·7 (C); P<0.05, and smoking: 18%, 6% and 9% (I) vs 12%, 21% and 18% (C); P<0.05. This corresponded to improvement in exercise capacity (0, 12 and 24 months): 156 ± 42, 174 ± 49 and 165 ± 47 W (I) vs 164 ± 40, 163 ± 49 and 156 ± 48 watts (C); P<0.05. There were no significant differences between the two groups with regard to serum cholesterol levels at 0 and 24 months: 5·4 ± 0·8 and 5·2 ± 0·9 mmol . l⁻¹ (I) vs 5·4 ± 1·0 and 4·9 ± 0·9 mmol . l⁻¹ (C); ns, low density lipoprotein cholesterol level: 3·6 ± 0·8 and 3·4 ± 0·8 mmol . l⁻¹ (I) vs 3·7 ± 0·9 and 3·3 ± 0·7 mmol . l⁻¹ (C); ns, triglyceride level: 2·2 ± 1·6 and 1·8 ± 1·3 mmol . l⁻¹ (I) vs 2·2 ± 1·4 and 1·6 ± 0·6 mmol . l⁻¹ (C); ns, body mass index (0, 12 and 24 months): 27·5 ± 4·5, 27·0 ± 4·3 and 27·4 ± 4·5 kg . m⁻² (I) vs 26·8 ± 2·8, 26·9 ± 2·7 and 26·9 ± 3·2 kg . m⁻² (C); ns, waist/hip ratio or blood pressure. The two groups did not differ in quality of life, or psychological factors. Return to work after 12 and 24 months was 74% and 78% (I) vs 68% and 61% (C); ns.

Conclusion This rehabilitation programme influenced important lifestyle behaviour and reduced some, but not all, important risk factors (Eur Heart J 1999; 20: 1465–1474)

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Key Words: Rehabilitation, risk factors, life style, behavioural modification.

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Introduction

In an extensive review of the impact of cardiac rehabilitation programmes, it was concluded that cardiac rehabili-

bilitation improved exercise tolerance, symptoms, blood lipids, proneness to quit smoking, psychological well-

being, feelings of stress and that it reduced mortality[11]. Comprehensive multifactorial programmes comprising prescribing exercise, changes in eating habits, smoking cessation, counselling and behaviour modification are the most effective. Some of these programmes have even been reported to affect the progression/regression of
coronary atheromatosis\[2–6\]. The escalating costs of cardiovascular disease require that investments in rehabilitative measures should be cost-effective in addition to enhancing quality of life. Nevertheless, transferral of potential candidates to such programmes remains low\[7\].

A reasonable prerequisite for a successful long-term outcome of a rehabilitation programme is to achieve sustained lifestyle changes. It has been recommended that all cardiac rehabilitation programmes should justify themselves by regular reports on the outcome of included components\[8\].

We developed a programme with the aim of achieving sustained and significant changes in risk factor-related lifestyle behaviour including exercise and dietary habits, stress management and smoking. In a previous report we described the 1 year outcome from 292 patients with various manifestations of coronary artery disease\[9\]. Significant effects were achieved in all included lifestyle areas. The lack of a proper control group, however, complicates the attribution of the changes to the intervention programme only. An alternative interpretation is that similar effects may be achieved by care provided within the traditional health care system, and that such care has increasingly come to include lifestyle change. The present study was planned to elucidate this issue by randomly assigning patients treated with percutaneous coronary angioplasty (PTCA) to a multifactorial lifestyle programme or to serve as a conventionally treated control group. An interim analysis after 1 year of follow-up demonstrated consistent effects on lifestyle behaviour\[10\]. This report presents data after 2 years of follow-up. The second year did not contain any active intervention. Thus, a main objective was to study maintenance of achieved behaviour and risk factor related changes.

Materials and Methods

The intervention programme

The rehabilitation programme comprised a 12-month period initiated by a 4-week residential stay at the intervention unit. This part of the programme consisted of intense health education and activities promoting behavioural changes. Although it included teaching sessions, the main emphasis was on training of practical skills and habit rehearsal. Lifestyle areas of particular emphasis were stress management, and diet, exercise and smoking habits.

Each participant was allocated to a group of five to eight persons. Education, discussions and skills training were mainly performed within these groups. Apart from seminars and lectures, the curriculum included physical exercise, food preparation, and training in ‘applied relaxation’\[11\]. Each participant was assigned a daily individual task including self-observation, Type A behavioural drills\[12\], relaxation training and exercise, to be conducted in between group sessions. The participants were served and trained to prepare a standard, low fat diet according to Swedish official guidelines (fat: \(<30\%\), saturated fat \(<10\%\), protein: 15% carbohydrates 50%)\[13\].

Each participant was assigned a specially trained nurse who conducted a series of individual structured interview sessions during the in-patient phase. The aim of these sessions was to achieve ‘individualization’ of all material covered elsewhere, thereby promoting motivation and preparedness for behavioural changes. A thorough analysis of the previous lifestyle was performed using the ‘Life-style Profile’ and ‘Stress Profile’ instruments\[14\], as well as robust planning of, and specific goal-setting for, future lifestyle modification. Discussions concerning relapse prevention were included\[15\].

An 11-month structured maintenance programme followed the in-patient phase. During this period, regular follow-up contacts were established between the patient and the nurse. These contacts were based on the agreed individual goal, and consisted of continued self-observation and recording of important aspects on everyday life in a diary, monitoring of behavioural changes, and, when needed, problem-solving and re-planning discussions. At discharge from the rehabilitation centre a referral note was sent to the family physician with information on achieved lifestyle changes. This maintenance programme was successively stepped down over the 11-month period, leaving the patients on their own during the second year of follow up.

Patients

Patients were recruited among those referred to the outpatient clinic of the Department of Cardiology, Karolinska Hospital for PTCA. Eligible subjects were those fulfilling the following criteria; (a) at least one significant coronary stenosis suitable for PTCA and at least one additional clinically insignificant coronary atherosclerotic lesion that could be evaluated by quantitative computerized angiography, (b) below the age of 65 years, (c) employed, (d) able to perform a bicycle ergometer test with a minimum capacity of 70 W following the PTCA, (e) absence of other diseases of importance for completion of the programme. Between February 1993 and December 1995, 151 consecutive patients fulfilled these criteria. Ninety-eight of these patients accepted participation following oral and written information. The PTCA was successful in 95 of these cases. During the post-PTCA bicycle ergometer test two patients were not able to reach the target of \(\geq 70\) W. The remaining 93 patients were randomly assigned to the intervention (n=48) or the control group (n=45) respectively. Two patients in the intervention and four in the control group were excluded soon after randomization at their own request leaving 87 subjects as the final patient population. Patients in the control group were subjected to ‘standard care’ which in our practice, after a PTCA procedure, is one outpatient visit at the clinic. Thereafter the patient is referred to his/her family physician for further secondary preventive efforts. Table 1 shows some characteristics of the study groups.
Assessment

Clinical end-points
The number of clinical events (acute myocardial infarction, coronary bypass surgery (CABG), or PTCA), number of hospital admissions and number of days in hospital attributable to cardiovascular causes were recorded during the 24-months of follow-up together with the proportionate return to work and time on sick leave. This information was collected from self-administered questionnaires which were to be confirmed by reviewing relevant hospital records and official registries. Quality of life was assessed through the ‘AP-QLQ’ questionnaire[16,17]. This self-rating scale was developed to assess quality of life related to angina pectoris. The analysis comprised a total quality-of-life score and four sub-factors: somatic symptoms, physical activity, emotional distress, and life satisfaction.

Lifestyle behaviour
Central aspects of the participants’ habitual lifestyles were assessed using a questionnaire developed for this intervention programme. This self-report scale was composed of items used in other Swedish assessment instruments, combined with specially developed questions. Variables analysed in the present study included frequency and intensity of stress reactions, relaxation frequency, knowledge of ‘heart-healthy’ diet, actual dietary behaviour, frequency of exercise, and smoking. An exercise session was defined as effort corresponding to a brisk walk for at least 20 min. Dietary behaviour was measured as a ‘diet index’. This was defined by averaging answers on several questions regarding the type of food generally eaten, emphasizing fat and fibre intake.

Psychological factors
Type A behaviour was assessed from the general ‘Bortner Type A’ index[18] and from a series of specific scales in the ‘HALTAM’ questionnaire[19], which cover different aspects of the Type A construct: ‘Hostility’, ‘Anger’, ‘Pressured Drive’, ‘Dominance’. An index of ‘Type A attitudes’ adapted from the ‘StressProfile’ instrument[14] was also included. Other psychological factors that were assessed include ‘cynical distrust’[20], anger[21,22], depression[23], anxiety[24], and health locus of control[25].

Medical risk indicators
Symptom limited exercise tests (W_{SL}) were performed in the sitting position on an electrically braked bicycle ergometer. The workload increased each minute by 10 W for women and 15 W for men. Perceived symptoms (anginal chest pain, dyspnoea) and exertion (leg fatigue) were recorded during the last 15 s of every minute and on the final workload, applying the Borg (0–10) scale[26].

Before the exercise test, weight and supine blood pressure were measured and a fasting blood sample for lipid analysis was obtained. Lipoproteins were determined by a combination of ultracentrifugation, precipitation of apo B-containing lipoproteins followed by lipid

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Table 1  Patient characteristics

<table>
<thead>
<tr>
<th></th>
<th>Intervention (n=46)</th>
<th>Control (n=41)</th>
<th>Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender (Male/female; n)</td>
<td>37/9</td>
<td>36/5</td>
<td>ns</td>
</tr>
<tr>
<td>Age (years ± SD)</td>
<td>53 ± 7</td>
<td>53 ± 7</td>
<td>ns</td>
</tr>
<tr>
<td>Tobacco use (Never/stopped/users; n)</td>
<td>13/25/8</td>
<td>9/24/8</td>
<td></td>
</tr>
<tr>
<td>Other diagnoses (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>48</td>
<td>63</td>
<td>ns</td>
</tr>
<tr>
<td>Previous myocardial infarction</td>
<td>43</td>
<td>34</td>
<td>ns</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>11</td>
<td>5</td>
<td>ns</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>9</td>
<td>20</td>
<td>ns</td>
</tr>
<tr>
<td>Medication (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acetylsalicylic acid</td>
<td>98</td>
<td>95</td>
<td>ns</td>
</tr>
<tr>
<td>Beta-blockers</td>
<td>70</td>
<td>90</td>
<td>p&lt;0.05</td>
</tr>
<tr>
<td>Nitrates</td>
<td>54</td>
<td>34</td>
<td>ns</td>
</tr>
<tr>
<td>Calcium antagonists</td>
<td>43</td>
<td>49</td>
<td>ns</td>
</tr>
<tr>
<td>ACE inhibitors</td>
<td>11</td>
<td>20</td>
<td>ns</td>
</tr>
<tr>
<td>Digoxin</td>
<td>0</td>
<td>2-5</td>
<td>ns</td>
</tr>
<tr>
<td>Diuretics</td>
<td>13</td>
<td>10</td>
<td>ns</td>
</tr>
<tr>
<td>Lipid lowering drugs</td>
<td>41</td>
<td>22</td>
<td>p=0.06</td>
</tr>
<tr>
<td>Antidiabetics</td>
<td>7</td>
<td>13</td>
<td>ns</td>
</tr>
<tr>
<td>Maximal exercise tolerance (W ± SD)</td>
<td>156 ± 42</td>
<td>160 ± 41</td>
<td>ns</td>
</tr>
<tr>
<td>Cholesterol (mmol . l^{-1})</td>
<td>5-4 ± 0-8</td>
<td>5-5 ± 1-0</td>
<td>ns</td>
</tr>
<tr>
<td>Triglycerides (mmol . l^{-1})</td>
<td>2-2 ± 1-5</td>
<td>2-2 ± 1-4</td>
<td>ns</td>
</tr>
</tbody>
</table>
analysis. Hourly ambulatory systolic, diastolic and mean blood pressures were recorded during 24 h by means of a digital monitor (SpaceLabs, Inc, Model No. 90207-30). Data were processed in a computer and presented as maximum, mean and minimum pressures with their standard deviations.

Statistics

Statistical tests were based on data collected at randomization and after 12 and 24 months of follow-up. Data are presented as mean ± standard deviation (SD). Unpaired two-tailed t-test and Fisher’s exact test (two-tailed) were used to describe and compare the groups at randomization. If measures were of a continuous character and judged normally distributed, a MANOVA with a repeated measures design was used to compare the groups over time. If not, a Wilcoxon rank sum test or Fisher’s exact test was used. A P value <0.05 was considered statistically significant.

The study protocol was approved by the ethical review board of the Karolinska Hospital, Stockholm.

Results

Clinical end-points

All patients, except one in the control group, survived the 2-year period. Seventeen (37%) of the patients in the intervention and 13 (32%) in the control group were admitted to hospital as a result of cardiovascular complaints at least once during the first 12 months of follow-up (ns). During the second year, two additional patients (4%) in the intervention group and eight (20%) in the control group were hospitalized (P<0.05). Only one control patient (2%) was admitted for acute myocardial infarction while 10 (22%) of the intervention patients and seven (17%) in the control group (ns) needed repeated PTCA during the 24 months to follow up. Five subjects (11%) in the intervention and six (15%) in the control group (ns) underwent coronary artery bypass surgery.

As reported in Table 2, measures of quality of life did not differ significantly between the two groups at baseline. Overall there was an improvement as regards total score, score for physical well-being, and score for somatic symptoms, but not for scores relating to psychological well-being and emotional satisfaction. Self-reported somatic symptoms were significantly less apparent in the intervention group, but there were no differences in angina pectoris. A high proportion returned to work after 1 year in both groups: 74% in the intervention group and 68% in the control group (ns). The proportion increased slightly in the intervention (78%) and decreased slightly in the control group (61%) during the second year of follow up (ns).

Table 2  Quality of life, symptoms and occupational status in the intervention and control groups

| Area                              | Variable                      | Intervention     | Control         | Difference*  
<table>
<thead>
<tr>
<th></th>
<th></th>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Month 0</td>
<td>Month 12</td>
<td>Month 24</td>
</tr>
<tr>
<td>Quality of life</td>
<td>QLQ (total)</td>
<td>4.1±1.0</td>
<td>4.7±0.8</td>
<td>4.7±0.8</td>
</tr>
<tr>
<td></td>
<td>Somatic symptoms</td>
<td>4.3±1.1</td>
<td>4.7±1.0</td>
<td>4.8±1.0</td>
</tr>
<tr>
<td></td>
<td>Physical activity</td>
<td>4.0±1.3</td>
<td>4.9±0.8</td>
<td>4.8±1.0</td>
</tr>
<tr>
<td></td>
<td>Emotional distress</td>
<td>4.4±0.9</td>
<td>4.7±0.8</td>
<td>4.8±0.8</td>
</tr>
<tr>
<td></td>
<td>Life satisfaction</td>
<td>3.8±1.1</td>
<td>4.2±1.0</td>
<td>4.2±1.0</td>
</tr>
<tr>
<td>Symptoms</td>
<td>Angina pectoris (index; 0–4)</td>
<td>1.2±1.0</td>
<td>0.7±0.7</td>
<td>0.7±0.8</td>
</tr>
<tr>
<td></td>
<td>Somatic symptoms (index; 0–4)</td>
<td>1.6±0.7</td>
<td>1.4±0.8</td>
<td>1.3±0.8</td>
</tr>
<tr>
<td>Return to work</td>
<td>% back to work</td>
<td>74</td>
<td>78</td>
<td>27</td>
</tr>
</tbody>
</table>

* Differences over time and between groups; MANOVA

Lifestyle behaviour

As shown in Fig. 1, exercise habits, knowledge of ‘heart-healthy’ diet, and self-reported diet habits improved significantly during the first year in the intervention group compared to the control group. These changes were maintained throughout the second year of follow-up. Frequency of stress reactions and relaxation sessions did not differ significantly between the two groups. One year prior to randomization the proportion of smokers was high in both groups and this was slowly declining towards the time of randomization. The proportion of smokers continued to decline during the first year in the intervention group but increased in the control group. During the second year there was a slight increase in the intervention group and a slight decrease in the control group. The difference was significant at 12 but not at 24 months.
Psychological factors

Type A attitudes measured with the ‘StressProfile’ instrument decreased significantly more in the intervention group compared with the control group (Table 3). Such a difference was not seen when Type-A behaviour was measured according to the Bortner scale, or HALTAM (total) scale, or when anger/hostility was measured as Anger Expression. Measurements of depression and anxiety revealed no significant differences between groups. Health-Locus-of-Control was significantly lower in the intervention group, indicating a higher degree of internal orientation (Table 3).

Medical risk indicators

As shown in Fig. 2, there were significant differences between the two groups during the first year as regards symptom-limited exercise capacity and dyspnoea as experienced at comparable workloads. These differences were maintained during the second year of follow-up.
Table 3 Changes in psychological factors

<table>
<thead>
<tr>
<th>Factor</th>
<th>Variable</th>
<th>Intervention</th>
<th>Control</th>
<th>Difference*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Month 0</td>
<td>Month 12</td>
<td>Month 24</td>
</tr>
<tr>
<td>Type-A behaviour</td>
<td>HALTAM (Total)</td>
<td>4.9 ± 0.7</td>
<td>4.7 ± 0.9</td>
<td>4.5 ± 0.8</td>
</tr>
<tr>
<td></td>
<td>Bortner scale</td>
<td>5.3 ± 0.9</td>
<td>4.9 ± 1.0</td>
<td>4.8 ± 0.9</td>
</tr>
<tr>
<td></td>
<td>Type-A attitudes</td>
<td>1.8 ± 0.8</td>
<td>1.2 ± 0.7</td>
<td>1.2 ± 0.7</td>
</tr>
<tr>
<td>Anger</td>
<td>Anger expression</td>
<td>24.3 ± 7.3</td>
<td>22.3 ± 7.4</td>
<td>22.2 ± 7.4</td>
</tr>
<tr>
<td></td>
<td>Cynical distrust</td>
<td>2.4 ± 0.7</td>
<td>2.4 ± 0.8</td>
<td>2.3 ± 0.7</td>
</tr>
<tr>
<td>Depression</td>
<td>Beck Depression Inventory</td>
<td>8.8 ± 7.5</td>
<td>8.7 ± 7.9</td>
<td>6.8 ± 6.8</td>
</tr>
<tr>
<td>Anxiety</td>
<td>Trait Anxiety</td>
<td>2.1 ± 0.6</td>
<td>2.0 ± 0.6</td>
<td>1.9 ± 0.5</td>
</tr>
<tr>
<td>Health-Locus-of-control</td>
<td>HLC**</td>
<td>3.3 ± 0.7</td>
<td>3.1 ± 0.7</td>
<td>3.0 ± 0.7</td>
</tr>
</tbody>
</table>

*Differences over time and between groups; MANOVA. **Lower values means more internal control
Scores for angina pectoris and leg fatigue remained low throughout the study period and were not significantly different between groups. Figure 3 shows changes in blood lipids (12-month data not available) and the impact of lipid lowering drugs on lipid levels. Overall

Figure 2 Results from exercise tests. Top: symptom limited exercise test, middle: dyspnoea at highest comparable workload and bottom: leg fatigue at highest comparable workload. ● = intervention group; ○ = control group. Error bars indicate standard deviations.

Figure 3 Blood lipids. Top: cholesterol levels; Total-C = total cholesterol, LDL-C = low density cholesterol and HDL-C = high density cholesterol. Middle: Tg = triglyceride levels. Bottom: percentual change in lipid levels in relation to lipid lowering medication in both groups. Added = patients who were not on lipid lowering drug at randomization but were prescribed a lipid lowering medication during the study period. Unchanged = patients who either had or were not on lipid lowering drugs through the entire study period. □ = intervention group; □ = control group. Error bars indicate standard deviations.
The present programme had a favourable, although modest influence on several self-reported lifestyle dependent behaviours. Achieved changes were maintained after 2 years of follow-up, and were accompanied by a significant improvement in exercise capacity. On the other hand, some important risk factors such as blood lipids, body mass or blood pressure did not differ significantly between patients subjected to the programme and those in ordinary care.

The overall morbidity and mortality was low and thereby hard to influence. The trend towards lower morbidity in the intervention group during the second year of follow-up is still of interest since it is possible that more distinct morbidity effects becomes apparent only after a considerable period of follow-up. The lack of effect of the intervention on perceived quality of life, which contrasted to the pervasive improvement seen in the total study cohort, is most reasonably explained by the successful PTCA procedure in a majority of patients in both groups. Thus, prevailing angina pectoris was rare. Somatic symptoms were significantly less apparent in the intervention group, which may explain the small quality of life differences in favour of subjects belonging to the intervention group.

Smoking is a major risk factor for coronary artery disease as illustrated by the high proportion of smokers one year prior to PTCA. The subsequent decline probably reflects that many of the patients quit in connection with their initial coronary event. The preservation of a non-smoking lifestyle was positively influenced by the programme.

The present improvement in exercise capacity compares well with previous reports on the long-term follow-up of multifactorial cardiac rehabilitation programmes. It may be argued that differences in symptom-limited exercise capacity may reflect a greater motivation to perform well in the actively treated group. This cannot be completely ruled out. It is, however, contradicted by the fact that patients in the intervention group experienced less dyspnoea at comparable workloads.

The most evident discrepancy with previous reports on comprehensive cardiac rehabilitation programmes is the lack of effect on serum lipids and weight. A possible explanation is a blunting effect of lipid lowering drugs. As shown in Table 4, additional prescribing of lipid lowering drugs was more prevalent in the control group and it was particularly patients with added lipid lowering drugs that experienced a reduction in total and LDL cholesterol. This is in line with reports of Hamalainen et al. and Watts et al. who both found a more efficient reduction in lipid levels when dietary recommendations were supplemented with lipid lowering drugs. Compared to our previous report on this programme, there was a substantial increase in the use of lipid lowering drugs in the PTCA sample we studied. This probably reflects an increased awareness among primary care physicians of the importance of cholesterol lowering therapy. Such relatively rapid changes in prescription patterns in routine care clearly underlines the importance of including control groups given standard care in studies of the present character. A second explanation for the lack of difference in serum lipids may be that the prescribed diet regimen was not as rigorous as the one used in other and more successful programmes for lifestyle modification. This may also contribute to the difficulty of maintaining weight reduction over a long period of time. Thus the achieved reduction of 1·6 kg after 1 year was totally lost after the second year of follow-up. A weakness with the presently used body mass index is that it does not take into account changes in body composition (fat/muscle). However, such changes are unlikely in the absence of changes in the hip/waist ratio. Other studies that address body mass index in a multifactorial cardiac rehabilitation setting reported results in favour of the intervention groups. In these studies there were concomitant and favourable effects on blood lipids.
Blood pressure did not differ between the two groups. Reviewing effects of cardiac rehabilitation, Wenger et al. concluded that such programmes had inconsistent effects on the lowering of blood pressure and that antihypertensive medication was a major confounding factor.

Psychological variables, except for self-reported ‘Type A attitudes’, did not differ significantly between the two groups, although there was an overall improvement in some Type-A related traits, and in anxiety. Previous studies reporting more impressive effects of behavioural modification in psychological parameters comprised post-myocardial infarction patients. It is reasonable to assume that a myocardial infarction, psychologically and physically, causes a more substantial trauma to the patient than angina and a subsequent PTCA. Accordingly, the post-infarction patient should be more open to behavioural change than the PTCA patient. Furthermore, the present methods of measuring behavioural change may be of limited sensitivity. They were typically developed to measure abnormal psychological responses and may be less appropriate to capture differences within a fairly ‘normal range’. The present programme had a short intervention period of 4 weeks. Other and more successful programmes have been based on regular group meetings over longer periods.

Study limitations that should be addressed are the format of the programme, patient selection and statistical power. The short, even though intense, initial phase of 4 weeks may not be sufficient to consolidate major lifestyle amendments such as dietary habits and type A behaviour. The maintenance part of the programme lacked, except for a referral note after the residential stay, formal cooperation with the patients’ regular care. Therefore the patients were not scheduled to attend for blood lipid measurements at appropriate points. The results from such tests would have provided important feedback on dietary habits and offered an opportunity to look more in detail at compliance with prescribed drugs. The reinforcement part of the programme only lasted 1 year. Only 87 of 151 patients finally participated. The options facing the subjects before randomization were either a 4-week residential stay at a place located about 600 km from home or to form part of a control group given standard treatment. Thus, it is likely that there was a selection bias towards patients motivated to lifestyle changes among the 65% of the total patient cohort that consented to participate in the study.

The achieved directional change in a majority of measured parameters favoured the intervention group, even though frequently failing to reach statistical significance. However, the present number of patients may have been too small statistically to confirm the observed differences.

In conclusion, this multifactorial behaviourally oriented intervention programme proved in a long-term follow-up to be modestly beneficial with regard to several lifestyle dependent factors. The lack of effects on other important risk factors (lipid levels, weight, blood pressure) necessitates its evaluation in terms of patient selection, and its comparison with a more domestically accessible and cheaper out-patient programme, before it can be generally recommended.

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References


