EFFECT OF AN EXERCISE CONSULTATION ON MAINTENANCE OF PHYSICAL ACTIVITY AFTER COMPLETION OF PHASE III EXERCISE-BASED CARDIAC REHABILITATION

by

Adrienne Rachel Hughes (BSc)

A Doctoral Thesis

Submitted in partial fulfilment of the requirements for the degree of

Doctor of Philosophy

University of Glasgow

Department of Medicine and Therapeutics

October 2003

ABSTRACT

There are many benefits associated with participation in cardiac rehabilitation exercise programmes following MI, CABG and PTCA. Sustaining these benefits requires maintenance of physical activity in the long-term. Evidence suggests that improvements in physical activity decline following completion of formal exercise programmes. The research is limited on interventions to improve maintenance of physical activity after completion of exercise-based cardiac rehabilitation. Exercise consultation is based on established behaviour change theories and uses strategies to promote and maintain physical activity. A pilot study found that exercise consultation improved short-term maintenance of physical activity following completion of a phase III exercise programme. However, the long-term effect of the exercise consultation has not been studied.

Aims: The primary aim of this randomised-controlled trial was to evaluate the effect of an exercise consultation (experimental condition) compared with standard exercise information (control condition) on maintenance of physical activity six and 12 months following completion of a phase III hospital-based exercise programme. Secondary aims included assessing the effect of an exercise consultation compared with exercise information on physiological and psychological variables at six and 12 months.

Methods: Seventy cardiac patients were recruited on completion of a phase III exercise programme. The experimental group received an exercise consultation and exercise information at baseline and six months, and a support phone call at three and nine months. The control group received exercise information at baseline and six months, and a support phone call at 3 and 9 months to maintain equal contact time between the groups. Measurements recorded at baseline, six and 12 months were; physical activity (Stanford Seven-Day Physical Activity Recall, CSA accelerometer and Stage of Exercise Behaviour Change); cardiorespiratory fitness, processes of exercise behaviour change, lipid profile, anxiety and depression (HADS), quality of life (SF-36), mortality and morbidity.

Results: At baseline, total activity measured by the 7-Day Recall was 300 minutes/week in the experimental group and 275 minutes/week in the control group, thus both groups were meeting guidelines for regular physical activity. Total activity assessed by the 7-Day Recall was maintained in the experimental group compared to the control group from baseline to 12 months (98%CI –295, -20). Total activity did not significantly change from baseline to six (98%CI -43, 191) and 12 months (98%CI -63,

154) in the experimental group. Similarly, total activity counts/week measured by the CSA accelerometer remained unchanged from baseline to six (98%CI -705643, 860599) and 12 months (98%CI -381927, 504719) in the experimental group. In the control group, total activity measured by the 7-Day Recall significantly decreased by 115 minutes/week from baseline to 12 months (98%CI -228, -28) and 63 minutes/week from six to 12 months (98%CI -126, -5). Additionally, CSA accelerometer counts/week decreased from baseline by 5.2% (98%CI -680366, 349212) at six months and 8% (98%CI -906564, 268168) at 12 months, although this decline was not significant. A comparable, significant decrease in peak oxygen uptake (VO_2) was recorded from baseline to 12 months by 6.7% (1.8 ml/kg/min; 98%CI -3.2, -0.3) in the experimental group and 9.4% (2.3 ml/kg/min; -3.8, -0.8) in the control group. Conversely, exercise duration (seconds) did not significantly change from baseline to six and 12 months in the either group. There was a significant improvement in the VO_2 at the lactate threshold (VO_2 LT) in the experimental group compared to the control group from baseline to 6 months (98%CI –185.4, -12.6) and 12 months (98%CI –216.7, -34.6). In the control group, there was a trend for a decrease in VO_2 LT by 52 ml/min (98%CI -116, 12) from baseline to six months. In the experimental group, there was a trend for an increase in VO_2 LT by 47ml/min from baseline to six months (98%CI -14, 109) and a significant increase by 81 ml/min from baseline to 12 months (98%CI 22, 140). The frequency of using nine out of 10 processes of exercise behaviour change did not significantly change from baseline to six and 12 months in either group. From baseline to 12 months, there was a significant decrease in the use of dramatic relief in the control group (98%CI -2.5, -0.5) and environmental re-evaluation in the experimental group (98%CI -3, -0.5). Weight and BMI did not significantly change over the study period in either group. Median lipid values at baseline were within the normal range in both groups. No significant changes in total cholesterol, triglycerides and low density lipoproteins (LDL) cholesterol were observed from baseline to follow-up in either group. There was a significant increase in high density lipoproteins (HDL) cholesterol by 0.11 mmol/l from baseline to 12 months in the experimental group (98%CI 0.04, 0.18), with no significant change in the control group. Median values for anxiety and depression were within the normal range at baseline and did not significantly change at six and 12 months in either group. Baseline scores for all quality of life subscales were high in both groups and did not significantly change from baseline to follow-up in either group. However, there was a trend for an improvement in social function, pain, role limitations due to physical problems, role limitations due to

emotional problems and energy/vitality from baseline to follow-up in the experimental group. In the control group, there was a trend for a decline in role limitations due to emotional problems, energy/vitality, and physical function from baseline to follow-up. Mortality was 5.7% (2/35) in the control group and 0% in the experimental group over 12 months. The revascularisation rate (i.e. CABG and PTCA) in the experimental and control group was 11.4% (4/35) and 0%, respectively.

Conclusions: This study demonstrated that the exercise consultation was more effective than exercise information in maintaining self-reported physical activity for 12 months after completion of a phase III exercise programme. However, the change in CSA accelerometer readings over the 12-month study period did not parallel the significant decrease in self-reported physical activity observed in the control group. The exercise consultation was not effective in maintaining exercise capacity for 12 months after completion of phase III. Significant decreases in peak VO_2 from baseline to 12 months were observed in both groups. In contrast, an improvement in the VO_2 at the lactate threshold, which is an index of submaximal endurance capacity, was recorded in the experimental group compared to the control group from baseline to follow-up. Finally, the exercise consultation had no significant effect on processes of exercise behaviour change, lipid profile and psychological function. These variables were normal at baseline and were maintained over the study period in both groups. The results of this study demonstrate that the exercise consultation may be an effective intervention for maintaining physical activity after completion of phase III hospital-based exercise programmes. The exercise consultation is a minimal intervention that could be delivered by physiotherapists to patients at the end of phase III or by British Association of Cardiac Rehabilitation (BACR) trained exercise instructors to patients in phase IV.

PUBLICATIONS

Papers

1. **Hughes AR,** Kirk AF, Mutrie N, MacIntyre P: Exercise consultation improves exercise adherence in phase IV cardiac Rehabilitation. *Journal of Cardiopulmonary Rehabilitation* 2002;22(6):421-425.

Abstracts

1. **AR Hughes**, N Mutrie, PD MacIntyre: Exercise consultation improves maintenance of physical activity during phase IV cardiac rehabilitation in Scotland. Medicine and Science in Sport and Exercise 2003;35(5):S175. Presented at the American College of Sports Medicine (ACSM) annual conference 2003
2. **AR Hughes**, N Mutrie, WS Hillis, PD MacIntyre: The effect of an exercise consulation on maintenance of physical activity following completion of phase III cardiac rehabilitation. Heart 2003;89(Supp 1):A23. Presented at the British Cardiac Society annual scientific conference 2003.
3. **AR Hughes,** AF Kirk, N Mutrie, PD MacIntyre: Exercise consultation improves exercise adherence in phase IV cardiac rehabilitation. Medicine and Science in Sport and Exercise 2002;35(5):S71. Presented at the American College of Sports Medicine (ACSM) annual conference 2002.
4. **AR Hughes**, N Mutrie, P MacIntyre, WS Hillis: The effect of exercise consultation of maintenance of physical activity and quality of life in phase IV cardiac rehabilitation. Presentation at the British Association of Cardiac Rehabilitation (BACR) annual conference 2002.
5. AF Kirk, **AR Hughes**, A Tait, T Thomson, PD MacIntyre: Physical assessment in clinical populations in Scotland. Medicine and Science in Sport and Exercise 2001;33(5):S56. Presented at the American College of Sports Medicine (ACSM) annual conference 2001.
6. **AR Hughes**, W Henry, AF Kirk, GP McCann, WS Hillis, PD MacIntyre: A questionnaire survey of uptake and adherence to phase IV community-based cardiac rehabilitation. Medicine and Science in Sport and Exercise 2000;32(5):S208. Presented at the American College of Sports Medicine (ACSM) annual conference 2001.
7. **AR Hughes**, F Gillies, GP McCann, PD MacIntyre, WS Hillis: Exercise

consultation: an intervention to improve adherence to phase IV exercise-based cardiac rehabilitation. Presented at the BACR annual conference 2000.

8. **AR Hughes**, FC Gillies, AF Kirk, N Mutrie, GP McCann, WS Hillis, PD MacIntyre: Exercise consultation: an intervention to improve adherence to phase IV exercise-based cardiac rehabilitation? British Journal of Sports Medicine 2000:34(2):148. Presented at the British Association of Sports Medicine 2000.
9. **AR Hughes**, W Henry, AF Kirk, N Mutrie, GP McCann, WS Hillis, PD MacIntyre: Uptake and adherence to phase IV community-based cardiac rehabilitation. British Journal of Sports Medicine 2000:34(2):148. Presented at the British Association of Sports Medicine 2000.

ACKNOWLEDGEMENTS

I would like to thank the following people who contributed to this thesis:

Dr Paul MacIntyre, my supervisor, for giving me the opportunity to study for this degree and his continued support and encouragement throughout the research. In addition, for funding me to attend the American College of Sports Medicine Annual Conference for the past four years, which enabled me to present my research findings.

Professor Nanette Mutrie, my supervisor, for her consistent support, encouragement and expertise, whilst conducting my research and writing my thesis. In addition, for giving me the opportunity to lecture on several cardiac rehabilitation courses, where I gained valuable teaching experience.

Dr Alison Kirk, my friend and fellow PhD student, who supported me throughout the entire PhD experience and accompanied me on frequent trips to the pub.

I am extremely grateful to the participants of the study, without whom, this research would not have been possible.

The cardiac rehabilitation team at the Royal Alexandra Hospital, who assisted in recruiting patients and gave me their expert advice on issues relating to coronary heart disease.

All the Chief Cardiac Scientific Officers (CCSO) at the Royal Alexandra Hospital who helped me conduct the exercise tests.

My family, who supported me financially and emotionally and must be relieved that I finally have a proper job.

Karen Goldie and Andrew McAuley who endured my complaining and kept me sane.

DECLARATION

I declare that this thesis was composed by myself and that all data were collected and analysed by myself. Neither the thesis nor the original work contained therein has been submitted to this or any other institution for a higher degree.

.....Signed ..... Date

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

ABBREVIATIONS

ACSM	American College of Sports Medicine
BMI	Body Mass Index
BACR	British Association of Cardiac Rehabilitation
CABG	Coronary Artery Bypass Graft
CDC	Centres for Disease Control
CHD	Coronary Heart Disease
CI	Confidence Interval
CSA	Computer Science and Applications
DLW	Doubly Labelled Water
HADS	Hospital Anxiety and Depression Scale
HBM	Health Belief Model
HDL	High Density Lipoprotein
HR	Heart Rate
LDL	Low Density Lipoprotein
METs	Metabolic Equivalents
MI	Myocardial Infarction
PA	Physical Activity
Peak VO_2	Peak Oxygen Uptake

PTCA	Percutaneous Transluminal Coronary Angioplasty
RPP	Rate Pressure Product
7DPAR	Stanford Seven Day Physical Activity Recall
SCT	Social Cognitive Theory
SF-36	Short Form 36
SEC	Socio-economic Classification
SIGN	Scottish Intercollegiate Guidelines Network
TPB	Theory of Planned Behaviour
TRA	Theory of Reasoned Action
TTM	Transtheoretical Model
VO_2	Oxygen Uptake
VCO_2	Carbon Dioxide Production
VO_2 max	Maximal Oxygen Uptake
VO_2 LT	Oxygen Uptake at the Lactate Threshold
VE/VO_2	Ventilatory Equivalent for Oxygen Uptake
VE/VCO_2	Ventilatory Equivalent for Carbon Dioxide
$PETO_2$	End Tidal Oxygen Partial Pressure
$PETCO_2$	End Tidal Carbon Dioxide Partial Pressure
UAP	Unstable Angina Pectoris

CHAPTER ONE

INTRODUCTION

This chapter reviews the literature that is relevant to this thesis. Section one describes the definitions and phases of cardiac rehabilitation, the benefits of exercise-based cardiac rehabilitation and participation in exercise-based cardiac rehabilitation. Section two discusses theoretical models that have been used to understand physical activity behaviour change. In addition, interventions based on theoretical models to improve maintenance of physical activity are discussed. Sections three and four critically review methods for measuring physical activity and psychological function, respectively. The literature review was conducted using 1) internet databases (i.e. Medline, Web of Science and Embase) using keywords and phrases appropriate to the topic being reviewed, 2) Evidence from systematic reviews and meta-analyses and 3) A search of the scientific literature using references cited in relevant literature reviews and published papers.

SECTION ONE

EXERCISE-BASED CARDIAC REHABILITATION

Definitions of Cardiac Rehabilitation

Cardiac rehabilitation has been defined as comprehensive, long-term programmes encompassing medical evaluation, cardiac risk factor modification, prescribed exercise, education and counselling. These programmes are designed to limit the physiologic and psychological effects of cardiac illness, reduce the risk for sudden death or reinfarction, control cardiac symptoms, stabilise or reverse the atherosclerotic process, and enhance the psychosocial and vocational status of selected patients.[1] Recent national guidelines on cardiac rehabilitation published by the Scottish Intercollegiate Guidelines Network (SIGN)[2] described cardiac rehabilitation as "the process by which patients with cardiac disease, in partnership with a multidisciplinary team of health professionals are encouraged and supported to achieve and maintain optimal physical and psychosocial health."

Four Phases of Cardiac Rehabilitation

In the UK, cardiac rehabilitation is divided into four phases. UK guidelines have provided recommendations on the structure and content of cardiac rehabilitation programmes.[3] However, few cardiac rehabilitation programmes actually adhere to these guidelines.[4] Thus, cardiac rehabilitation programmes vary considerably in content, client groups offered the service, length of intervention, the type of health professionals involved in the delivery and the setting for that delivery.[5]

Phase I (in-patient)

Phase I occurs during the inpatient stage or after a *step change* in the patient's cardiac condition. A *step change* is defined as myocardial infarction (MI), the onset of angina, any emergency hospital admission for coronary heart disease (CHD), cardiac surgery or angioplasty, or the first diagnosis of heart failure. The key elements of this phase are medical evaluation, reassurance, information/education, correction of cardiac misconceptions, risk factor assessment, mobilisation and discharge planning. Family and partners are usually involved from this stage.

Phase II (early out-patient)

Phase II is the immediate post discharge period and can vary in length from 4 to 6 weeks, depending on the cardiac condition (e.g. after CABG this phase can be approximately six weeks, whereas post MI this period may last four weeks). In this phase, patients and family receive support from hospital or community-based health professionals through home visits or telephone contact. Additional education and information is provided to reinforce risk factor modification and behavioural change. Further medical evaluation risk stratifies the individual for future management.

Phase III (intermediate out-patient)

Exercise training, education, psychological support and risk factor modification are the key components of phase III programmes. A recent survey of cardiac rehabilitation services in Scotland found that the majority of phase III programmes involved supervised exercise training once or twice per week in a hospital setting.[5] However, some programmes offered exercise training in community or home-based settings. The duration of phase III exercise programmes varied from five to 13 weeks, with a median duration of 11.5 weeks. Most of the programmes had an educational

component, which provided patients with information on the CHD process, risk factors, benefits of exercise, drug therapy, relaxation training and stress management. Some programmes also offered psychological and vocational counselling.

Phase IV (long-term maintenance)

Phase IV involves the long-term maintenance of physical activity and other lifestyle changes in the community. Cardiac support groups, which offer group exercise classes or phase IV maintenance exercise programmes delivered by exercise leaders trained by the British Association of Cardiac Rehabilitation (BACR) are available in some areas. Generally, patients do not receive follow-up from cardiac rehabilitation staff in phase IV. Instead, the majority of patients receive most or all of their care in primary care.[2] The patient's clinical status, medication and risk factors should be regularly reviewed by his or her general practitioner and other primary care health professionals during phase IV.

The structure of cardiac rehabilitation in North America is different from UK programmes. Phase I is the in-patient stage, which is similar to the UK. In America, patients in phase II attend supervised hospital-based exercise programmes lasting approximately 12 weeks. Phase III involves hospital or community-based supervised exercise programmes lasting three to six months. Phase IV is the long-term maintenance stage, similar to UK programmes.

Exercise Prescription for Health and Fitness

Exercise Prescription for Healthy Adults

In 1990, the American College of Sports Medicine (ACSM) published guidelines on the quantity and quality of exercise needed to develop and maintain cardiorespiratory fitness in the healthy adult.[6] These guidelines, summarised in Table 1, recommend 20 minutes or more of continuous, moderate to vigorous intensity aerobic exercise (60% to 90% of maximum heart rate or 50% to 85% of maximum aerobic capacity) on 3 days a week to improve cardiorespiratory fitness. To maintain the training effect, exercise must be continued on a regular basis, as a significant reduction in cardiorespiratory fitness occurs after 2 weeks of stopping training. Although the minimal level of exercise necessary to maintain fitness is not known, evidence

suggests that missing an exercise session periodically or reducing the frequency and duration of training will not adversely effect cardiorespiratory fitness as long as training intensity is maintained.[7]

More recent evidence demonstrates that lower levels of physical activity, than the amount required to improve fitness, can produce substantial health benefits. Therefore, the quantity and quality of activity needed to attain health-related benefits differ from what is recommended for fitness benefits.[6] In 1995, the American College of Sports Medicine (ACSM) and Centers for Disease Control and Prevention (CDC) published guidelines on the quality and quantity of physical activity required to improve health and prevent disease.[8] These guidelines, summarised in Table 1, recommend accumulating at least 30 minutes of moderate intensity physical activity on most days of the week. Thirty minutes of moderate activity each day can be achieved by adding some moderate physical activity into the daily routine. For example, taking the stairs instead of the lift, and brisk walking instead of driving short distances. Furthermore, many daily activities such as gardening, household chores and washing the car can contribute to the 30-minute per day total if performed at a moderate intensity (i.e. effort equal to a brisk walk).

The 1995 recommendation is intended to complement the 1990 exercise guideline. For example, sedentary individuals or those who are not regularly active will gain substantial health benefits by increasing their physical activity to achieve 30 minutes of moderate activity on most days. In addition, it is much easier for these individuals to incorporate small amounts of moderate activity into their daily routine rather than engaging in structured, planned programmes of exercise. However, individuals who are moderately active will gain additional health and fitness benefits by achieving the 1990 exercise recommendations.[6]

Table 1 Physical Activity Recommendations for Healthy Adults

1990 ACSM exercise recommendation to improve and maintain cardiorespiratory fitness: • A minimum of 3 x 20 minutes of continuous, moderate to vigorous intensity aerobic exercise per week. 1995 ACSM and CDC physical activity recommendation to promote health and prevent disease: • Accumulate at least 30 minutes of moderate intensity physical activity on five or more days per week.

The 1995 recommendation is distinct from the 1990 guideline as it emphasises moderate intensity activity, the 30 minute a day total can be achieved by accumulating short bouts of activity, and this amount of physical activity produces health benefits, but may not necessarily improve fitness.[8]

Exercise Prescription for Cardiac Rehabilitation Participants

The primary goals of exercise training in cardiac rehabilitation are to counteract the deleterious effects of bedrest, avoid the deconditioning effects of physical inactivity imposed by CHD, increase aerobic capacity and modify risk factors.[3] In 1995, the British Association of Cardiac Rehabilitation (BACR) produced guidelines on the amount of exercise required to achieve these benefits.[3] Patients in phase I (in-patient) are advised to engage in self-care activities, general range of motion exercise and walk short distances to counteract the deleterious effects of prolonged bed rest. In phase II, patients undertake daily living activities and gradually increase the duration, frequency and intensity of walking to increase functional and endurance capacity. In phase III, 20 to 30 minutes of moderate to vigorous intensity (70% to 85% of maximum heart rate) exercise on three days a week is recommended to improve aerobic capacity and modify risk factors. Results from a systematic review found that exercising at this level for three months increased exercise tolerance by 30% to 50% and peak VO_2 by 10 to 20%.[1] However, recent SIGN guidelines[2] state that participation in low to moderate intensity exercise (50% to 75% of maximal HR) is sufficient for most cardiac patients. Most phase III programmes offer supervised exercise once or twice a week, thus SIGN guidelines recommend that patients should be encouraged to

participate in moderate activity on days that formal exercise training is not taking place to achieve the exercise prescription.[2;3]

The exercise prescription for phase IV participants is not as clearly defined as the recommendations for patients in phases I to III. BACR guidelines recommended that patients in phase IV should engage in 20 minutes or more of continuous, moderate to vigorous intensity aerobic exercise on three days per week to improve and maintain fitness.[3;6] This exercise prescription is similar to the 1990 ACSM exercise recommendations to increase fitness in healthy adults.[3] Recent SIGN guidelines[2] state that phase IV participants should continue regular moderate intensity exercise in the long-term to sustain benefits, however the frequency and duration of this exercise was not described. American cardiac rehabilitation guidelines[9] have endorsed the 1995 ACSM/CDC physical activity recommendation of accumulating at least 30 minutes of moderate intensity physical activity on five days per week to promote health.[8] This level of physical activity may be easier for cardiac patients to incorporate into their daily routine and sustain in the long-term. Table 2 summarises the physical activity recommendations for cardiac patients in phases III and IV.

Table 2 Physical Activity Recommendations in Phase III and IV Cardiac Rehabilitation

Phase III Cardiac Rehabilitation

- 20 to 30 minutes (excluding warm-up and cool down) of low to moderate intensity aerobic exercise (e.g. walking, cycling, circuit training) on three days a week.

And

- Moderate intensity physical activity (e.g. walking) on days when not participating in formal exercise training.

Phase IV Cardiac Rehabilitation

- A minimum of 20 minutes (excluding warm-up and cool down) of moderate intensity exercise on three days per week.

And/Or

- Accumulate 30 minutes or more of moderate intensity physical activity on at least five days per week.

The Need for Cardiac Rehabilitation

Compared with the rest of the UK, Scotland has a disproportionately high incidence and prevalence of coronary heart disease (CHD).[5] Every year in Scotland, approximately 8,000 people survive a myocardial infarction, 6,000 people undergo coronary artery bypass surgery or angioplasty, and 13,000 people require emergency admission to hospital with chest pain (excluding MI). All of these patients would benefit from participation in cardiac rehabilitation services. In addition, cardiac rehabilitation would also be beneficial for an estimated 33,000 angina patients, 6,000 heart failure patients and 500 additional patients having other forms of cardiac surgery (e.g. transplantation). In total, approximately 66,500 patients annually may be eligible for cardiac rehabilitation in Scotland.[5]

Provision of Cardiac Rehabilitation

Despite the need for cardiac rehabilitation services, the provision of programmes in the UK is inadequate.[10-12] A recent survey estimated that only 17% to 25% of eligible patients attend cardiac rehabilitation annually in Scotland.[11] Another Scottish survey found that the proportion of patients discharged from hospital with a diagnosis of

CHD who attended cardiac rehabilitation varied between 2% to 46% in different health board areas.[10] A assessment of cardiac rehabilitation services in the UK, reported that approximately 14% to 23% of infarct patients, 33% to 56% of CABG patients and 6 to 10% of PTCA patients are enrolled in programmes.[12] Generally, cardiac rehabilitation is offered to patients following MI or CABG, whereas the provision is limited for women, elderly patients, and those with angina or heart failure.[10-14] Studies on the provision of cardiac rehabilitation in North America have found similar results.[15;16] Thus, it seems that many patients that would benefit from cardiac rehabilitation are not being offered these services.

Uptake to Cardiac Rehabilitation

Even when patients are offered cardiac rehabilitation, uptake rates to programmes are variable and can be low. The percentage of patients attending cardiac rehabilitation programmes, following invitation, ranges from 13% to 74%[14;17-20]. In 1996, Pell[18] found that 59% of MI patients, who were invited, participated in phase III rehabilitation programmes based in four Glasgow hospitals.

Factors associated with uptake to cardiac rehabilitation

Numerous studies have identified factors influencing uptake to supervised phase II and III cardiac rehabilitation exercise programmes, whereas the research is limited on variables associated with maintenance of physical activity in phase IV.

Research has found that participation in formal cardiac rehabilitation programmes following a MI was significantly higher for men (49%) compared to women (34%), and younger patients of both sexes were more likely to attend compared to older patients.[21] Other studies have shown comparable results for age[14;22;23] and gender,[17;23] whereas Pell[24] found that these factors did not influence participation in cardiac rehabilitation. The strength of the physician's recommendation to attend rehabilitation is an important determinant of participation,[23;25] and this recommendation was stronger for men compared to women.[23] Other reasons associated with non-participation in cardiac rehabilitation include; conflicting work and family commitments,[26] medical problems,[25] inconvenient timing of programmes,[25;27] transport problems,[10;25] social deprivation,[14;24] being under the care of a GP compared to a cardiologist,[18] inactive leisure habits,[17] and being moderately functionally impaired.[17] Utilisation of rehabilitation is lower for single or widowed

patients compared to those who are married and uptake rates are higher for employed patients or those who are better educated.[17] Patients with a strong belief that their cardiac disease is controllable and those who attributed their heart condition to their lifestyle are more likely to attend rehabilitation.[22] Finally, uptake is not predicted by a patient's intention to attend cardiac rehabilitation.[22]

Menu-Based Cardiac Rehabilitation

Generally, cardiac rehabilitation programmes provide a fixed package of rehabilitation due to resource limitations, which may not to meet the needs of many patients.[5] This may explain the low referral, uptake and adherence rates to cardiac rehabilitation programmes among certain patient groups. National guidelines recommend that rehabilitation should be menu-based, which means tailoring rehabilitation services to suit the patient's needs and their readiness to make lifestyle changes.[2] A flexible menu-based approach may improve participation in cardiac rehabilitation and prevent certain patient groups such as the elderly and heart failure patients from being excluded. However, few cardiac rehabilitation programmes in the UK are currently providing menu-based services.[2]

Benefits of Exercise-Based Cardiac Rehabilitation

This section reviews the benefits of exercise-based cardiac rehabilitation on mortality, morbidity, exercise capacity, physical activity, psychological functioning and coronary risk factors.

Terminology

In this literature review, cardiac rehabilitation programmes that include exercise training, education and risk factor modification are termed "comprehensive cardiac rehabilitation programmes." Early clinical trials of cardiac rehabilitation included exercise training as the sole intervention, thus these programmes are referred to as "exercise only rehabilitation." The term "exercise-based cardiac rehabilitation" includes both comprehensive and exercise only rehabilitation programmes. In addition, most the trials investigating the efficacy of exercise-based cardiac rehabilitation have included phase II and III cardiac rehabilitation programmes, whereas few studies have evaluated the effect of interventions in phase IV.

Therefore, evidence for the benefits of exercise-based cardiac rehabilitation are primarily based on phase II and III programmes.

Mortality and Morbidity

A number of randomised-controlled trials have reported a reduction in mortality with exercise-based cardiac rehabilitation compared to usual care following myocardial infarction, although only a few trials have reported a statistically significant reduction. In the majority of these studies, the sample size was too small to detect a significant difference between the intervention and control group.[28] Combining the data from these trials in a meta-analysis increases the power of the statistical analysis,[29] thereby providing a more accurate estimate in the size of the effect of exercise-based cardiac rehabilitation on mortality and morbidity.

Oldridge[30] conducted a meta-analysis investigating the effect of comprehensive cardiac rehabilitation following myocardial infarction on total and cardiovascular mortality and recurrence of non-fatal MI. Ten randomized-controlled trials of comprehensive rehabilitation, which included risk factor management and exercise training after myocardial infarction with at least a two year follow-up period were included in the analysis. In the majority of trials, the intervention was initiated 6 to 12 weeks after myocardial infarction. Results revealed a significant reduction in total mortality by 24% and cardiovascular mortality by 25% with cardiac rehabilitation compared to usual care. The duration of the rehabilitation programmes varied from 6 weeks to 48 months; subgroup analysis revealed that longer interventions had a greater benefit on total mortality than short-term programmes. In contrast, cardiac rehabilitation had no significant effect on the recurrence of non-fatal myocardial infarction. Similarly, another meta-analysis[31] of 22 randomised controlled trials of exercise-based cardiac rehabilitation after MI reported a significant decrease in total mortality by 20% and cardiovascular mortality by 22% after an average of three years follow-up. Furthermore, cardiac rehabilitation had a significant beneficial effect on the recurrence of fatal MI throughout the three-year follow-up period. In contrast, the rates of non-fatal MI were comparable between the two groups throughout the 3 years. The time from myocardial infarction to randomisation ranged from 1 to 3 months in the majority of studies. A recent meta-analysis conducted by Jolliffe[28] included more studies (41) and a greater number of patients (n = 7683) who had suffered an MI, angina, or undergone CABG or PTCA compared to the earlier meta-

analyses.[30;31] In addition, Jolliffe excluded a number of trials that were included in the previous reviews because they lacked a true control group or a clear method of randomisation. This study found that exercise only rehabilitation significantly reduced total mortality by 27%, cardiac mortality by 31% and fatal MI by 35% compared to usual care.[28] Comprehensive cardiac rehabilitation produced a non-significant reduction in total mortality by 13% and significantly lowered cardiac mortality by 26% and fatal MI by 28%. Conversely, both exercise only and comprehensive rehabilitation had no significant effect on rates of non-fatal MI, or revascularisation (i.e. CABG or PTCA) compared with usual care, although few trials reported CABG or PTCA as an outcome.

Although the three meta-analyses varied considerably in the trials they included and excluded, they consistently found that exercise-based cardiac rehabilitation improved survival.[28;30;31] In addition, all three meta-analyses determined the separate effect of exercise only and comprehensive cardiac rehabilitation on survival. Jolliffe[28] reported greater mortality benefits for exercise only rehabilitation compared to comprehensive rehabilitation. However, the difference in mortality between exercise only rehabilitation and comprehensive rehabilitation exercise were not significant. Oldridge[30] found that both types of intervention were comparable, whereas O'Conner[31] stated that comprehensive rehabilitation was more effective than exercise only rehabilitation. These inconsistent findings, the small number of exercise only trials and the possibility that advice on risk factors was included in exercise only interventions does not allow definite conclusions to be reached about the independent effect of the exercise component of cardiac rehabilitation on mortality. There are several mechanisms by which exercise-based cardiac rehabilitation may improve survival of cardiac patients. They include improved coronary blood flow, decreased ventricular arrhythmias, improved cardiovascular risk factor profile, reduced psychological distress[32] improved cardiovascular fitness, or increased patient surveillance.[30]

There are concerns regarding the results of these meta-analyses. Firstly, most studies involved predominantly middle-aged males following myocardial infarction, thereby limiting the generalisability of the results to women, elderly patients and other cardiac groups. Secondly, thrombolysis and medication was not reported in many trials, which would have affected clinical outcomes if more intervention

patients received these treatments compared to control patients. Finally, it is impossible to identify the optimal duration and content of cardiac rehabilitation programmes to improve survival due to the different interventions used in the studies. The exercise component of the programmes varied from supervised exercise training to home-based walking programmes, which differed in the frequency, duration and intensity of exercise. In addition, many programmes included stress management, diet, smoking cessation and psychological interventions. The duration of rehabilitation ranged from 6 weeks to greater than 1 year. The time from the cardiac event to initiation of the intervention, length of follow-up and type of participant were not consistent across trials, which creates problems in the interpretation of the results. A review of exercise-based cardiac rehabilitation stated that evaluating the cardiac rehabilitation literature is difficult due to the variability of the interventions and populations studied.[33]

Exercise Capacity

Exercise capacity may be reduced following a myocardial infarction due to infarct-related damage to the myocardium and the deconditioning effects of physical inactivity during hospitalisation.[34] Conversely, a spontaneous improvement in exercise capacity occurs in the first 3 months after myocardial infarction without exercise training.[35-41] In addition, increases in exercise capacity after treatment of CHD with CABG and PTCA have been observed in the absence of exercise training.[34] However, further improvements in exercise capacity with training following MI, CABG and PTCA have been documented, as discussed in the next section.

The effect of exercise-based cardiac rehabilitation on exercise capacity has been widely studied. A systematic review[1] published in 1995 found that the majority of randomised and non-randomised controlled trials reported statistically significant improvements in exercise capacity with exercise-only or comprehensive cardiac rehabilitation compared to usual care in patients with CHD. The review reported that exercise tolerance on the treadmill increased by 30 to 50% and peak oxygen uptake increased by 10 to 20% after three months of moderate to vigorous intensity exercise three times a week for 20 to 40 minutes. The current exercise prescription for phase III participants is based on these findings. However, most of the studies included in the systematic review involved predominantly middle-aged male patients of low to moderate risk following a MI. Thus, the benefit of exercise-based cardiac rehabilitation

to increase exercise capacity is well established in this patient population. Although, several trials have reported significant, comparable improvements in exercise capacity after exercise-based cardiac rehabilitation in men and women,[35;42-44] and in patients older and younger than 70 years of age.[45;46] This systematic review was published in 1995, thus more recent randomised-controlled trials evaluating the effect of cardiac rehabilitation exercise training on exercise capacity are described in Table 3. In addition, the review did not report the long-term effect of cardiac rehabilitation on exercise capacity, thus Table 3 also includes randomised-controlled trials reporting the long-term effect of cardiac rehabilitation on exercise capacity.

As illustrated in Table 3, eighteen of these controlled trials reported an improvement in exercise capacity with exercise-based cardiac rehabilitation compared to usual care.[35-37;47-50;54-63;67] In contrast, three randomised-controlled trials[51;52;79] reported no significant difference in exercise capacity between the intervention and control groups. Although the evidence suggests that participation in exercise-based cardiac rehabilitation improves exercise capacity, it is important to determine whether this increase in exercise capacity is maintained following programme completion.

Nine studies have investigated the effect of exercise-based cardiac rehabilitation on exercise capacity in the long-term after programme completion (see Table 3).[37;50;51;53;55;56;63;67;79] Liddell randomised 116 patients 5 weeks after MI to a phase I/II comprehensive rehabilitation programme or usual care.[56] In phase I (in-patient) patients received education and support, in phase II (out-patient) they participated in 5 months of supervised and home-based exercise training, group education on lifestyle modification, and received support phone calls. Patients in usual care received information on risk factors and leisure activities, and attended outpatient clinics one, three and 12 months after MI. Results revealed that exercise capacity was significantly higher in the intervention group compared to the control group at 6 months (end of programme) and at five years follow-up. However, the change in exercise capacity from baseline to follow-up was not documented in this study. Similarly, Lisspers and Hofman-Bang reported that the improvement in exercise capacity with 12 months of comprehensive cardiac rehabilitation compared to usual care was maintained for a further 12 months following programme completion.[63]

In contrast, seven studies have found that improvements in exercise capacity with cardiac rehabilitation exercise training are not sustained in the long-term after programme completion.[37;50;51;53;55;67;79] Carson[55] compared the effect of a 12 week exercise programme with usual care on exercise capacity in 303 male patients who had suffered a MI in the previous 6 weeks. The improvement in exercise capacity was significantly greater in the intervention group compared to the control group at 5 months (end of the programme), and at 1 and 2 years follow-up. However, exercise capacity was similar in the intervention and control groups at 3-years, due to a gradual decline in exercise capacity in the intervention group during the follow-up period. Similarly, Oldridge found that the increase in exercise capacity after 8 weeks of comprehensive rehabilitation had declined at 12 months, reaching a similar level to the control group.[37] Stahle compared three months of supervised exercise training with standard care on exercise performance and self-reported physical activity in 101 elderly patients (>65 years) discharged after a MI or unstable angina. Three months of exercise training significantly improved self-reported physical activity and exercise tolerance compared with standard care.[67] However, there were no differences in physical activity and exercise tolerance between the groups at 12 months.[67] The authors suggested that continued exercise training is required to prevent a decline in initial improvements in physical activity and exercise capacity.

Overall, the literature suggests that improvements in exercise capacity with exercise-based cardiac rehabilitation may not persist in the long-term, suggesting that many patients do not continue to exercise after they have completed a cardiac rehabilitation programme. However, only one of these trials assessed patients' physical activity levels during the follow-up period.[67] Cardiac rehabilitation guidelines recommend continuation of exercise following completion of formal exercise programmes to sustain the initial improvements in exercise capacity.[1]

Mechanisms for Increased Exercise Capacity after Exercise Training

The significance of an increase in exercise capacity is that CHD patients will be able to accomplish all submaximal activity at reduced physiological stress. Ades found that increases in self-reported physical functioning after 3 months of cardiac rehabilitation exercise training was significantly associated with improvements in exercise capacity.[69] The mechanism for the improvement in exercise capacity after training in individuals with CHD is primarily due to peripheral (skeletal muscle and

vascular) adaptations rather than central (cardiac) adaptations.[70-73]. Peripheral adaptations include increases in arteriovenous oxygen difference,[74] blood flow[74;75] and aerobic metabolism[76] in active skeletal muscle at maximal exercise. Furthermore, increases in fibre area, capillary density and oxidative enzyme activity have been observed in skeletal muscles after training.[74] These peripheral changes reflect an improvement in the supply of oxygen to active skeletal muscles, and enhanced oxygen extraction and utilisation by the exercising muscles. Some researchers have reported enhanced cardiac performance with exercise training, including increases in ejection fraction,[61] cardiac output,[77] stroke volume and oxygen pulse[61] at maximal exercise, which indicates an increased capacity of the cardiovascular system to transport oxygen to skeletal and cardiac muscle. Whereas, other studies found no changes in cardiac function with exercise training.[74;75] These inconsistent findings could reflect differences in the methods used to assess cardiac function and to variations in the duration and intensity of exercise training. It has been suggested that intense exercise training for one year or more may be required to increase cardiac function of individuals with CHD.[70;78]

Table 3: Effect of Exercise-Based Cardiac Rehabilitation on Exercise Capacity (EC) after MI, CABG and PTCA

Study	Subjects	Design	Results
Bethell[36] RCT	N=200, 6 wks post MI. 100% males, <65 years	Intervention: Community-based sup circuit training 3 times/week for 12 weeks. Usual care: Advice on unsupervised exercise. FU: 3 m from baseline (4-5 m post MI).	Sig greater improvement in estimated VO_2max in IG vs CG.
Oldridge[37] RCT	N=201, 6 wks post MI . 90% males, 52 years. Patients had moderate anxiety or depression.	Intervention: 8 wk hospital-based CP. Sup exercise (65% maxHR), 2 times/week for 50 mins. Weekly gp counselling. Usual care: not described. FU: End of programme & 12m.	Sig ↑ in EC in both groups from baseline to 8 wks & 12 m. Sig difference favouring IG at 8 wks but not 12 m. (EC ↓ in IG at 12 m).
DeBusk[35] RCT	N=70, 3 wks post MI 2 IGs & 1CG 100% males, 52yrs.	Intervention: 8 wks of Sup exercise 3 times/wk, 60 mins or home exercise 5 times/wk, 30 mins at 70-85% maxHR. Usual care: not described. FU: 11 weeks.	Sig ↑ in estimated METs in all three gps, but ↑ was sig greater in the 2 IGs vs CG. EC ↑ by 34% in CG, 41% in home gp & 53% in sup gp.
DeBusk[47] RCT	N=585, 3–6 wks post MI 78% males, 57 years (all <70 years).	Intervention: Home-based CP. Exercise, 60–85% maxHR, 5 days/wk, 30 mins. Behavioural intervention for smoking, exercise & lipids. Mail & phone contact for 1 yr. Usual care: Education on smoking, diet and exercise. FU: 6m.	EC sig ↑ by 2.1 METs in the IG group. At 6 m, EC was sig greater in IG vs CG (9.3 METs vs 8.4 METs). Baseline EC was not reported.

Dugmore[48] Non-randomised	N=124, 3 wks post MI 98% males. Subjects split into good & bad prognosis gps.	Intervention:12m hospital sup exercise 3 times/wk. Good prognosis:65 to 80% pkVO_2. Bad prognosis: 50 to 65% pkVO_2. Usual care: not reported FU: 4, 8 and 12 months.	Peak VO_2, RPP and exercise duration were sig higher in good and bad prognosis IG vs matched CG at 4, 8 and 12 m. Did not report change from baseline.
Dressend[49] RCT	N=50 4 wks post MI 3 IG & 1 CG 100% males, 54 years	Intervention: 5 wk phase II hospital-based sup exercise training (70% VO_2max), 35 mins/session either 1, 2 or 3 times/wk. Usual care: restricted to light activities (<50% VO_2 max) at home. FU: End of 5 wk programme.	Treadmill duration sig ↑ in IGs (20-25%) & CG (10%). Sig ↑ in VO_2 max in IGs by 16% (1/wk), 19% (2/wk), 20% (3/wk) & NS ↑ by 8% in VO_2 max in CG. ↑ in treadmill duration & VO_2 max was sig greater in the IGs training 2 & 3 times/wk vs CG.
Liddell[56] RCT	N=116, 5 wks post MI 87% males, 56 yrs (all <66 yrs)	Intervention: CP involving Phase 1 (inpatient) education & support. Phase II (outpatient) sup exercise for 1 hour/wk & education for 6m. Patients encouraged to exercise unsupervised. Frequent phone contact. Usual care: Information on risk factors, & attended outpatient clinics 1, 3 and 12m post MI & GP annually. FU: 6m (end of phase 2) & 5 yrs.	EC was sig higher in the IG vs CG at 6 m & 5 yrs. ↑ in EC from baseline to FU not reported.

Heldal[50] RCT	N=37, 4 wks post MI 100% males, 53 years (all <66 years).	Intervention: Hospital, outpatient intensive exercise programme for 4 wks. Sup exercise 5 days/wk, 2 hrs/session, 85% HR max. Usual Care: encouraged to walk at a moderate speed. FU: 4 wks (end of intervention) & 6m post MI.	From 0m to 4 wks, EC ↑ sig more in IG vs CG. From 0m to 6m, there was a NS ↑ in EC by 6% in CG & a sig ↑ by 14% in IG. Between gp diff for the change was NS.
Holmback[51] RCT	N=69, 6 wks post MI 97% males, 55 years	Intervention: 12-wk hospital prog involving sup exercise, 2 times/wk, 45 mins/session. Intensity 70-85% HRmax initially, then 100% max HR. Patients told to exercise after programme. Usual Care: regular medical care, no emphasis on exercise. FU: 1 yr post MI.	EC ↑ sig by 10% in IG & NS ↑ by 2% in CG. The between group difference for the change was not sig.
Kallio[52] & Hamalainen[53] RCT	N=375, 2 wks post MI 80% males, <65 years.	Intervention: CP including smoking & diet advice, psychological counselling & sup exercise. No detail on content & length of training. Programme was intense for 3m, but patients had contact with staff for 3 yrs. Frequent medical review. Usual care: follow-up by own GP. FU: 1, 2, 3, 6, 10 yrs post MI.	No sig difference in EC between IG vs CG throughout the 10-yr FU. But changes from baseline were not reported. Similar % of patients exercising regularly in IG & CG, but actual percentages not reported.
Roman[54] RCT	N=193 2m post MI 100% males, 54 years, (all <71 years).	Intervention: Hospital outpatient sup exercise, 50% VO_2 max, 3 times/wk for a mean of 42 m. Usual Care: not detailed. FU: 6 & 12m post MI.	Peak VO_2 sig ↑ IG after 6m of training and a further NS ↑ at 12m. EC did not sig change in CG. Sig diff between gps at 6 & 12m.

Carson[55] RCT	N=303, 6 wks post MI 100% males, 51 years (all <70 years).	Intervention: 12 wk outpatient circuit training twice/wk. Intensity not specified. Told to exercise after programme. Usual care: not detailed. FU: 5 months, 1, 2 and 3 years.	From baseline to 5m, exercise duration sig ↑ in both groups. EC was sig higher in IG vs CG at 5m, 1 & 2 yrs. No diff between gps at 3 yrs.
Froelicher[57] RCT	N=53, mean time from CABG to study entry was 2 yrs, range 6m - 9 yrs. 100% males, 54 years.	Intervention: 12m hospital sup exercise programme, 8 wks of exercise with ECG monitoring, 45 mins, 3 times/wk, 60% VO_2 max. After 8 wks, patients attended a walk/jog programme. Usual care: offered a walking programme <50% VO_2 max FU: 12m	Mean attendance at exercise sessions was 82% (range 65-97%). VO_2 max ↑ by 7.1% in IG & ↓ by 2.9% in CG. Between gp difference for the change in EC was sig.
Wosornu[58] RCT	N=81, CABG in past 12m (median 12 wks). 2 IGs & 1CG 100% males, 57 years.	Intervention: 6m hospital sup aerobic exer or strength training, 12 to 40 mins, 3 times/wk. Intensity based on ACSM 1990 guidelines. Usual care: Told to engage in usual activities. FU: 3 & 6m.	EC sig ↑ by 1.4 METs at 3m & 2 METs at 6m in the aerobic gp vs no change (0.2 METs) in CG. From baseline to 6m, METs ↑ by 29% in aerobic gp, 15% in strength gp & 2% in CG.
Oldenburg[59] RCT	N=86 4-8 wks post CABG 90% males, 59 years (all<70 years).	Intervention: Hospital CP, 6 weekly gp sessions for 3 hrs, booster sessions at 8 & 12m, sup exercise & education on risk factors. Behavioural skills included, goal-setting, skills training, self-monitoring & support. Exercise goals: 30-40 mins/day. Usual Care: not detailed. FU: 4 & 12m post CABG.	Baseline to 4m, peak VO_2 ↑ by 6.2 ml/kg/min (21%) in CG & 4.5 ml/kg/min (14%) in IG. Baseline to 12m, peak VO_2 ↑ by 6.7 (21%) ml/kg/min in IG & 5.5 ml/kg/min (19%) in CG. ↑ in EC was sig greater in IG vs CG at 12m but not 4m.

Willoughby[60] Non-randomised	N=92 6 wks post CABG 2 IGs & 1 CG 83% males, 69 years (all >63 years).	Intervention: 12 wk phase II hospital programme, sup exercise for 20-45 mins, 3 times/week, 65% maxHR (low intensity gp) or 85% maxHR (high intensity gp). Patients did not exercise outwith phase II. Usual care: no formal programme. FU: 12 wks (end of programme).	Exercise duration & VO_2 max sig ↑ in the 2 IGs vs CG. VO_2 max ↓ by 2% in CG & sig ↑ by 23% in low intensity gp & 38% in high intensity gp. The ↑ in EC was sig greater in high vs low intensity gp, thus found a dose response effect.
Higgins[79] RCT	N=99 post-PTCA Majority males, 48 yrs (all <64 yrs).	Intervention: 2m home CP, involved 3 home visits & monthly phone calls, unsupervised moderate walking & behavioural strategies to modify risk factors; goal-setting, self-monitoring, feedback, skills training & support. Usual care: in-patient education on CHD. FU: During admission, 10 wks & 1 yr post PTCA.	Similar sig ↑ in EC in IG & CG at 10 wks & 12m, no diff between groups.
Lisspers[62] & Hofman-Bang[63] RCT	N=87 2 wks post PTCA 85% males, 53 years (<65 years).	Intervention: 12-month CP. Phase I (4 wks): lectures, discussions, skills training, goal setting & relapse prevention to ↑ motivation for changing stress, exercise, diet & smoking. 11m home programme (phase II), regular contact, self-monitoring, feedback of changes, problem solving & replanning discussions. Usual Care: followed-up by own physician. FU: 12 & 24m.	Baseline to 12m, EC sig ↑ by 11% in IG & no change in CG, sig between group difference. This difference was maintained at 24m.

Belardinelli[61] RCT	N=118 25 days post PTCA 80% males, 57 years.	Intervention: 6m hospital sup exercise 3 times/week, 30 mins, 60% peakVO_2. Usual care: given a list of suitable "mild" activities & activity diary FU: 6m from study entry.	Peak VO_2, lactate threshold, & oxygen pulse at a given workload sig ↑ in IG vs CG. Peak VO_2 ↑ by 26% in IG. 83% of IG & 7% of CG ↑ their EC by ≥ 1 MET. 76% of CG had a lower peak VO_2 at 6m vs baseline.

Note. MI = myocardial infarction, CABG = Coronary artery bypass graft, PTCA = Percutaneous transluminal coronary angioplasty, FU = Follow-up, Gp = Group, IG = Intervention group, CG = Control Group, EC = Exercise capacity, RCT = Randomised controlled trial, m = month, wks = weeks, yrs = years, CP = comprehensive programme, diff = difference, prog = programme, NS = non-significant, Sup = Supervised, ↑ = increase, ↓ = decrease, Sig = Significant, RPP = rate pressure product.

Submaximal Endurance Capacity

In addition to increased exercise capacity, exercise training also improves submaximal endurance capacity in patients with CHD, as demonstrated by a significant reduction in patients' rate of perceived exertion (RPE) at submaximal levels of exercise[49;68;80] and a delayed onset of the lactate threshold.[61;75;81;82] These effects are beneficial to the daily activities of cardiac patients. The lactate threshold is the exercise VO_2 at which the concentration of lactate in arterial blood increases above the resting value due to the supplementation of aerobic energy production with anaerobic metabolism during heavy exercise.[83] Below the lactate threshold, exercise at a given work rate can be sustained for a prolonged period of time without fatigue and with relatively little stress to the subject. In contrast, work rates above the lactate threshold lead to metabolic acidosis, an increased ventilatory response and fatigue, thus exercise above this threshold cannot be sustained as long. The delayed onset of the lactate threshold after exercise training enhances an individual's ability to perform exercise at a given work rate. Proposed mechanisms for the higher lactate threshold after training include improved oxygen supply to active skeletal muscles, increased oxidative capacity of mitochondria and delayed activation of type II muscles fibres that function anaerobically.[83] Hambrecht reported a 7% increase in VO_2 at the lactate threshold after 12 months of unsupervised and supervised exercise training compared with an 8% decrease in the control group in patients with CHD.[84] Although, this study found that patients had to expend 1400 calories per week, which equated to 3.3 hours of vigorous intensity exercise, to delay the onset of the lactate threshold. In contrast, other studies have found significant improvements in the lactate threshold after 3 to 6 months of training at a lower intensity and frequency of exercise.[61;75;81]

Angina and Ischaemia

A major benefit of exercise training for patients with CHD is the delayed onset of angina and ischaemia during exercise. Angina and ischaemia occurs at a fixed rate pressure product (RPP), which is an index of myocardial oxygen demand and is the product of heart rate and systolic blood pressure (HR x SBP).[34] Several studies have reported a lower myocardial oxygen demand with exercise-based cardiac rehabilitation, demonstrated by a significant decrease in RPP during submaximal exercise.[49;57;58;80;82;85] A decline in RPP is a result of a decrease in heart rate and systolic blood pressure at a given submaximal workload. In addition, a lower

submaximal heart rate increases the period of diastole, thus coronary blood flow will be enhanced as 80% of blood flow through the coronary arteries takes place during diastole. The overall effect is that at any given submaximal level of exercise, myocardial oxygen demand is reduced and coronary blood flow is enhanced, thus patients can perform a greater amount of work before the onset of angina or ischaemia.[70] Debusk reported a significant reduction in angina and ECG determined ischaemia during exercise testing after 8 weeks of supervised exercise training compared to controls in a group of post MI patients.[35] Other studies found improved myocardial perfusion during exercise testing, indicating a reduction in myocardial ischaemia with exercise training in CHD patients.[61;86]

Physical Activity Levels

This section reviews the evidence on the effect of exercise-based cardiac rehabilitation on physical activity. The literature was searched from 1979 to 2003 using Embase and Medline databases. The following key words were used: cardiac rehabilitation, myocardial infarction, coronary artery bypass surgery (CABG), coronary heart disease (CHD), percutaneous transluminal coronary angioplasty (PTCA), coronary artery disease, exercise, physical activity, compliance and adherence. In addition, relevant studies were identified from references cited in published papers and reviews of cardiac rehabilitation. Thirteen randomised controlled trials,[39;56;59;62;63;67;79;84;88;90-94] 1 non-randomised controlled trial,[98] 1 case-control study[97] and 5 observational studies[20;87;89;95;96] investigating the effect of exercise-based cardiac rehabilitation on physical activity are described in Table 4.

Short-term Effect

Ten randomised-controlled trials[39;56;62;63;67;79;84;88;91;98] and two observational studies[89;96] reported increases in physical activity after participation in exercise-based cardiac rehabilitation (see Table 4). In contrast, two randomised controlled trials reported comparable significant increases in physical activity in both rehabilitation and control groups at follow-up. The major limitation of these trials involves the method used to measure physical activity. These studies relied on self-report measures of physical activity, and except for one study[39] objective measures of physical activity were not employed. In addition, only four studies[84;88;89;96] used a valid and reliable questionnaire to measure physical activity.

Physical activity guidelines for cardiac patients and healthy adults recommend accumulating 30 minutes of moderate activity on at least five days per week, which is equivalent to expending a minimum of 1000 calories per week during physical activity to achieve health and fitness benefits.[8;9;34] Thus, results from two of the observational studies discussed in Table 4 demonstrate that exercise-based cardiac rehabilitation improves patients' physical activity habits to levels recommended by current guidelines.[89;96] Although, these studies did not assess whether patients continued to achieve these guidelines in the long-term. However, greater amounts of physical activity than those recommended by current guidelines may be required to slow the progression of coronary atherosclerosis in CHD patients. Hambrecht found that expending a minimum of 1600 calories per week during leisure physical activity was required to slow the progression of CHD, whereas regression of CHD was noted in highly motivated patients who expended an average of 2200 calories per week, amounting to between 5 and 6 hours of vigorous physical exercise.[84] Although, it is unlikely that the majority of patients with CHD would engage in this amount of physical activity.

Long-Term Effect

Twelve studies investigated the long-term effect of exercise-based cardiac rehabilitation on physical activity (see Table 4).[20;56;59;63;67;79;84;87;91;94;96;98] Two randomised-controlled trials[59;63] found that improvements in physical activity with cardiac rehabilitation were maintained following programme completion. In addition, Hambrecht[84] found that physical activity related energy expenditure measured by questionnaire was significantly higher in the intervention group compared to controls at 12 months follow-up. In contrast, six randomised controlled trials[56;67;79;91;94;98] and three observational studies[20;87;96] found a decline in patients' physical activity levels following completion of cardiac rehabilitation exercise programmes. Bock evaluated the proportion of patients meeting current physical activity guidelines (i.e. accumulating 30 minutes or more of moderate activity on most days of the week) and measured physical activity by self-report in 62 cardiac patients who attended a 12-week supervised cardiac rehabilitation exercise programme.[96] At the beginning of rehabilitation, 43% of participants were meeting the physical activity guidelines. On programme completion, 96% of participants were meeting the guidelines and the amount of time spent in moderate and hard intensity physical activity had increased significantly. In contrast, three months after rehabilitation, the proportion of patients

achieving the recommendations had decreased to 80% and nearly 50% of participants had reduced their physical activity levels compared to the end of the programme. Similarly, Stahle reported a significant improvement in exercise capacity and physical activity with three months of exercise training compared to usual care.[67] However, physical activity levels and exercise capacity were similar between the groups at 12 months, as physical activity and fitness had declined in the intervention group.

Bethell reported a significant increase in exercise capacity and physical activity after three months of supervised exercise training compared to usual care.[91] In a follow-up study, the number of patients who continued to exercise regularly after completing the rehabilitation programme was assessed annually for 11 years using a postal questionnaire. The response rate was greater than 70% throughout the 11 year follow-up. In the intervention group, the proportion of patients exercising regularly rapidly fell from 70% to 40% during year one and then gradually declined over subsequent years, until only 25% of rehabilitated patients were exercising regularly from year seven onwards. In addition, 25% of patients in the control group reported exercising regularly throughout the 11-year follow-up period. These results suggest that a short-term exercise programme was more effective than usual care in improving physical activity habits for up to six years. However, these findings also show that the majority of patients were not exercising regularly in the long-term following cessation of supervised exercise training. Similarly, other studies (described in Table 4) have reported that only 12% to 50% of patients continue to exercise regularly 12 months to 9 years after completion of exercise-based cardiac rehabilitation.[20;56;87;94;95] The major limitation of these studies is definition of regular exercise, which varied greatly and was not based on current physical activity recommendations. For example, in studies by Bethell[91] and Prosser,[94] patients were categorised as regular exercisers if they engaged in vigorous physical activity (e.g. swimming, jogging etc), whereas moderate intensity activities were excluded. Current cardiac rehabilitation guidelines[2] recommend moderate intensity exercise for most cardiac patients, thus these studies may have underestimated the proportion of patients who maintained a sufficient level of physical activity to benefit their health. In other studies, patients were considered to be regular exercisers if they exercised twice a week[87] or "had started to exercise post MI."[56] Thus, the number of patients who remain regularly active after exercise-based cardiac rehabilitation is variable

and depends on the length of follow-up and the definition of regular physical activity. Only one study used the current physical activity guidelines to determine the proportion of patients who remained regularly active after exercise rehabilitation.[96] Nine randomised-controlled trials and three observational studies examined the long-term impact of cardiac rehabilitation on physical activity. Six of the nine randomised-controlled trials found that improvements in physical activity with cardiac rehabilitation declined in the long-term. In addition, all three observational studies reported that physical activity was not maintained in the long-term. However, the results of the observational studies must be interpretetd with caution as these studies are more prone to potential bias and confounding factors than controlled trials.

In summary, the evidence suggests that participation in exercise-based cardiac rehabilitation can improve physical activity levels. However, it seems that the impact of cardiac rehabilitation exercise programmes on long-term maintenance of physical activity is limited. Several studies reported that increased levels of physical activity are not maintained in the long-term following completion of formal programmes.

Table 4: Effect of Exercise-Based Cardiac Rehabilitation on Physical Activity

Study	Patients	Design	Results
Heath[88] RCT	N=65 10d post CABG 2IGs & 1CG	Intervention: 12-wk sup or home exercise programme, 30-40 mins 5 times/wk, 70-90% peakHR. Exercise counselling & prescription after programme. Usual Care: Information on risk factors & activity. FU: 6 – 8m post CABG. Outcomes: Leisure PA in kcals/wk assessed by questionnaire.	PA was sig lower in CG (1089 kcals/wk) vs home IG (2058 kcals/week) & sup IG (2,549 kcals/week) at FU.
Bertie[39] RCT	N=81 3 wks post MI	Intervention: 4 wk hospital CP involving risk factor advice, sup exercise twice/wk & encouraged to do an exercise video daily. Usual care: Information on risk factors. FU: 4m post hospital discharge. Outcomes: Mean km walked/day using a pedometer	At 0m, distance walked per day was 4.5 km in CG & 5.1 km in IG. At 4m, distance walked/day was sig higher in IG (8.2 km) vs CG (6.6 km).
Engblom[90] RCT	N=228, CABG. 87% males, 54 years (all < 65 years).	Intervention: Hospital CP. Phase I: 3d pre-CABG, information on surgery & recovery. Phase II: 6 wks post-CABG, risk factor education, & 21 hrs sup exercise for 3 wks. Phase III: 8m post CABG, 2d refresher of phase II. Usual care: Instructions on gradually ↑ activity. FU: 6 & 12m. Outcomes: Self-report PA. No definition of regular exercise.	From pre CABG to 6 & 12m FU, % of patients exercising regularly sig ↑ in IG (22% to 42%) & CG (↑ 10% to 38%), no diff between gps.

Bethell[91] RCT	N=200 6 wks post MI	Intervention: 12 wk community sup exercise 3 times/wk. Usual care: advice on unsupervised exercise. FU: annually for 11 yrs. Outcomes: Self-reported PA. Regular exercise defined as participating in vigorous exercise. Duration or frequency not assessed.	Questionnaire response rate was approx 70%. % of IG exercising regularly ↓ from 70% to 40% in yr 1, then gradually ↓ to 25% at yr 7, remained at that level. 25% CG exercised regularly over 11 yrs.
Stahle[67] RCT	N=101 6 wks post MI or unstable angina	Intervention: Hospital sup exercise 3 times/wk for 3m & once/wk for further 3m. After programme, patients encouraged to attend community classes. Usual Care: Advised to walk daily & attended monthly meetings. FU: 3 & 12m. Outcomes: PA on a 6-point scale: 1=sedentary & 6= strenuous exercise (e.g. jogging, swimming) ≥ 3hrs/wk.	PA sig↑ in IG (mean 4.5) vs CG (mean 3.9) at 3m. at 12m, PA was 3.8 in IG & 3.6 in CG, no diff between gps. Changes in PA at 3m were not maintained at 12m in IG.
Higgins[79] RCT	N=99 post PTCA	Intervention: 2m home CP, home visits & phone calls, unsupervised moderate PA & behavioural strategies to modify risk factors; goal-setting, self-monitoring, feedback, skills training & support. Usual care: in-patient education on CHD. FU: pre-PTCA, 10 wks & 1 yr post PTCA. Outcomes: % patients exercising = 1-3 times/wk for 20 mins/session.	% of IG exercising sig ↑ from 35% at 0m to 88% at 10 wks & 72% at 12m. % of CG exercising did not sig change from 53% at 0m, to 59% at 10 wks, to 61% at 12m. Sig diff between gps at 10 wks, not 12 m.

Oldenburg[59] RCT	N=86 4-8 wks post CABG 90% males, 59 years (all<70 years).	Intervention: 6wk hospital CP, 1 gp session/wk for 3 hrs & booster sessions at 8 & 12m. Sup exercise & education on risk factors. Behavioural skills: goals, skills training, self-monitoring, support. Exercise goals: 30mins/d walk Usual Care: not detailed. FU: 4 & 12m post CABG. Outcomes: Regular exercisers: ≥ 3/wk, moderate exercisers: 2/wk & non-exercisers: ≤ 1/wk.	PA sig ↑ in both gps from 0m to 4m & this level was maintained at 12m. No sig diff between gps. Did not report levels of PA participation.
Lisspers[62] & Hofman-Bang[63] RCT	N=87 2 wks post PTCA 85% males, 53 years (<65 years).	Intervention: 12-month CP. Phase I: 4 wks, lectures, discussions, skills training, goal setting & relapse prevention to change stress, exercise, diet & smoking. 11m home programme (phase II), regular contact, self-monitoring, feedback, problem solving & replanning discussions. Usual Care: followed-up by own physician. FU: 12 & 24m. Outcomes: Exercise session = effort equal to brisk walk for 20 mins.	From 0m to 12m, the frequency of exercise sessions ↑ sig in IG (2.5 to 5/wk) vs CG (2.5 to 3/wk), which was maintained at 24m. At 12m, 56% of IG & 41% of the CG exercised at least twice/wk.
Liddell[56] RCT	N=116, 5 wks post MI 87% males, 56 yrs (all <66 yrs)	Intervention: CP. Phase 1 (inpatient) education & support. Phase II (outpatient) sup exercise 1 hr/wk & education for 6m. Patients were encouraged to exercise unsupervised. Frequent phone contact. Usual care: Risk factor information & attended clinics 1, 3 & 12m post MI. FU: 1 & 5 yrs. Outcomes: PA assessed by statements "started to exercise post MI"	% patients exercising was sig greater in IG (67%) vs CG (28%) at 1 yr. No sig diff between IG (41%) & CG (28%) at 5 yrs.

Carlsson[92] RCT	N=168 4 wks post MI	Intervention: 3m CP, counselling on diet, smoking & PA. 40mins of sup exercise ≥2/wk. End of programme, received exercise plan & education. Usual care: medical evaluation, risk factor education & exercise programme. FU: 12m. Outcomes: PA: 1=sedentary, 2=walking/cycling daily for ≥30 mins, 3=sport activities ≥1/week, 4=sport activities ≥2/wk, 5=vigorous exercise.	At hospital admission, 69% IG & 78% of CG were physically active (i.e. categories 2 – 5). At 12 m, 77% of IG & 70% of CG were physically active. 78% IG & 67% CG who were sedentary at baseline were active at 12m, no sig diff between gps.
Hambrecht[84] RCT	N=62 with CHD.	Intervention: 3 wk inpatient programme; low fat diet & sup exercise 6 times/d for 10 mins at 75% maxVO_2. Out-patient programme; unsupervised exercise, 30 mins/d & 2 sup exercise sessions/wk 60 mins. Usual care: information on exercise & diet. FU: 12 months Outcomes: EE (kcals/wk) by Minnesota leisure time PA questionnaire.	At 12m, EE was 1,876 kcals/wk IG & 1,187 kcals/wk CG ($p<0.05$). In total gp, EE was sig lower in patients with progression of CHD (1022 kcals/wk), vs no change in CHD (1533 kcals/wk) or regression of CHD (2204 kcals/wk).
Haskell[93] RCT	N=300 with CHD years, 80% males.	Intervention: Home CP, individualised goals to modify risk factors. PA programme involved daily PA e.g. walking, climbing stairs, & aerobic exercise for ≥30mins on 5d at 70-85%maxHR. Phone & mail contact. Usual Care: medical review by GP. FU: annually for 4 yrs. Outcomes: PA measured by 7 day PA recall.	At baseline, 79% of IG & 72% of CG exercised regularly ($p>0.05$). At FU, % of IG exercising regularly sig ↑ to 90% vs CG (70%).

Prosser[94] RCT	N=291 6 wks post MI	Intervention: 3m sup exercise for 45 mins/session, twice/wk. Usual care: not mentioned. FU: 6-9 years post MI. Outcomes: Questionnaire on % patients exercising vigorously (> 6 METs).	> 70% response rate. 19% IG were exercising at FU vs 2% of CG.
Ben-Ari[98] Non-randomised controlled trial	N=175 1 wk post PTCA.	Intervention: 12 wk CP involving education on risk factors & sup exercise 45 mins, 2-3d/wk at 70-85% maxHR. Usual Care: information on risk factors & PA. FU: 3 & 18m. Outcomes: self-report PA.	% of patients exercising ≥ 2 times/wk 89% in IG and 72% in CG at 3m ($p<0.05$). No sig diff at 18m (75% IG vs 70% CG).
Morrin[89] Cohort	126 > 6 wks post cardiac event.	Intervention: 6m hospital CP, education, sup exercise twice/wk, 50-75% HRR & unsupervised activity for 200-400 mins/wk (1,200-2,000 kcals/wk). Individualised goals for risk factors, feedback on risk factors at 0, 3 & 6m. FU: 3 & 6m. Outcomes: EE in kcal/wk assessed by 7 Day PA Recall.	81% adherence rate to the programme. Sig ↑ in PA from 0m (830kcals/wk) to 3m (1345 kcals/wk) & 6m (1325 kcal/wk).
Perk[97] Case-control	N=157 6 wks post CABG	Intervention: 3m hospital CP. Education, sup exercise 2/wk, 100% maxHR & individualised home exercise programme. After 3m, patients attended a community programme. Usual care: medical review at 1 year. FU: Mean 38 months after CABG. Outcomes: PA assessed by a mailed questionnaire (not validated).	66% of IG were regular exercisers vs 46% of CG ($p=0.05$). IG, mean duration of training/wk was 6.5 hrs vs 5.2 hrs/wk CG ($p=0.07$).

Kinsey[87] Cohort	N=48 post-CABG	Intervention: 12 wk phase II home exercise programme. Patients were advised to walk or cycle 3- 4 times/wk after programme. FU: 4 yrs. Outcomes: leisure PA, including frequency, intensity &duration.	80% compliance to programme. 41% were exercising at least twice/wk at 4 yrs.
Fontana[20] Cohort	N=50 post MI or CABG.	Intervention: Hospital sup exercise for 35 mins/session, 3 times/wk, 85% maxHR for 12 wks. FU: 12 weeks, 6 and 12 months. Outcomes: % patients attending programme, % completing programme, & % continuing to exercise after programme.	52% attended programme. 39% completed programme. % continuing to exercise after prog was 60% at 12 wks, 30% at 6m & 37% at 12m.
Vidmar[95] Cohort	N=138 patients	Intervention: 12wk phase II hospital CP, exercise training & education on risk factors. FU: patients who had completed phase II in the past 8 yrs Outcomes: PA questionnaire (not validated).	39% (138/352) response rate. 87% of patients were exercising, either unsupervised or at a phase III maintenance programme. 44% engaged in moderate PA for 40 mins≥ 3/wk.
Bock[96] Cohort	N=62 after MI, CABG or PTCA.	Intervention: 12wk phase II CP, sup exercise 1 hr, 3 times/wk, 60-80% HRR, risk factor education & guidelines for home activity. FU: 12 wks (programme completion) & 6m. Outcomes: 7-Day PA Recall (mins/wk) & Stage of Exercise Behaviour Change. Regular PA = accumulate ≥ 30mins moderate PA on 5d /wk.	26% dropped out programme. At 0m, 47% patients were regularly PA. At 12 wks, 96% were regularly PA & mins/wk of PA sig ↑. 6 m, 80% were regularly PA. 12 wks to 6m, 42% participants regressed (i.e. mins/wk of PA ↓).

Note. MI = myocardial infarction, CABG = Coronary artery bypass graft, PTCA = Percutaneous transluminal coronary angioplasty, CHD = coronary heart disease, FU = Follow-up, Gp = Group, IG = Intervention group, CG = Control Group, EC = Exercise capacity, PA = Physical activity, EE = Energy expenditure, RCT = Randomised controlled trial, m = month, wks = weeks, Sup = Supervised, ↑ = increase, ↓ = decrease, Sig = Significant, d = days, diff = difference, CP = comprehensive programme, HRR = heart rate reserve.

Psychological Functioning

Psychological distress following myocardial infarction is common and can lead to poor outcomes.[2] The prevalence of depression in patients following MI ranges from 15% to 45%.[2] Patients diagnosed with major depression according to DSM-III-R (Diagnostic and Statistical Manual of Mental Disorders revised third edition) criteria (16%) seven days after a MI were at a significantly greater risk of dying over the subsequent 6 months than non-depressed patients.[100] Furthermore, the increased risk remained after controlling for previous MI, ejection fraction and other clinical indicators of disease severity. Another study reported that moderate symptoms of depression measured using the Beck Depression Inventory (score $\geq$ 10) immediately after MI predicted mortality over the following 18 months.[101] Again this effect was independent of other post MI risk factors. Mayou observed that patients suffering from psychological distress (15%) measured using the Hospital Anxiety and Depression Scale three days after MI reported poorer quality of life, more cardiac symptoms, exercising less, continuing to smoke, and greater use of health services over the subsequent 12 months compared to nondistressed patients.[102] Evidence also suggests that depressed patients are less likely to adhere to medical treatment or lifestyle changes including exercise programmes.[103-105] In addition, psychological distress after an acute cardiac event does not improve in many patients without treatment.[102;106] Therefore, as distressed subjects are at an increased risk of adverse outcomes following a MI, an aim of cardiac rehabilitation is to "limit the psychological effects of cardiac illness and to enhance psychosocial status".[1]

Numerous studies have examined the effect of exercise-based cardiac rehabilitation on psychological functioning, as described in Table 5.[32;37;38;51;59;61;63;66;67;79;89;107-109] Most of these studies found that exercise-based cardiac rehabilitation produced psychological benefits with exercise-based cardiac rehabilitation.[32;37;38;51;59;61;63;66;67;79;89;107-109] Similarly, systematic reviews found that the majority of controlled trials reported statistically significant improvements in psychological functioning with comprehensive rehabilitation compared to usual care.[1;246] However, it is impossible to determine which components of comprehensive cardiac rehabilitation programmes (e.g. exercise training, education, individual and group counselling etc) were responsible for the psychological benefits. The reviews also found that exercise training as the sole component of cardiac rehabilitation improved psychological wellbeing and quality of life.[1;246] However, the

direct effect of exercise training on psychological outcomes is confounded by the consequence of group interaction, the formation of social support networks during supervised exercise programmes, and support and guidance from cardiac rehabilitation staff, which are likely to affect anxiety, depression and quality of life. It is thought that improved psychological function with exercise training was a result of increased fitness. However, Newton reported that improvements in psychological functioning after a 10-week rehabilitation exercise programme compared with routine care were independent of changes in fitness.[38] Overall, evidence suggests that exercise-based cardiac rehabilitation enhances psychological wellbeing. In addition, Denollet found that reducing psychological distress with comprehensive rehabilitation improved long-term mortality.[32]

Long-term Effects

The long-term effects of exercise-based cardiac rehabilitation on quality of life and psychological functioning have been examined.[37;51;59;61;63;66;67;79;107;109;110] A randomised controlled trial of 118 patients following PTCA reported significant improvements in quality of life assessed using the Short-Form 36 after 6 months of supervised exercise training.[61] Furthermore, the positive change in quality of life was maintained six months after programme completion. In contrast, quality of life did not significantly improve in the control group over the 12-month study period. Similarly, other controlled trials have found that 6 to 12 weeks of comprehensive rehabilitation enhanced psychosocial functioning compared to usual care for 6 to 12 months after MI and CABG.[110;111]

Conversely, several randomised controlled trials found no significant difference in psychological functioning between the intervention and control groups in the long-term.[37;51;59;63;66;67;79] Stahle randomised 101 elderly patients 6 weeks following MI to a 3 month supervised exercise programme or usual care.[67] Significantly greater improvements in many quality of life dimensions, assessed by a disease-specific questionnaire, were observed after 3 months of exercise training compared to usual care. However, quality of life gradually improved in the control group and declined in the rehabilitated group, thus similar scores were observed between the groups at 12 months. Similarly, Oldridge[37] found that 8 weeks of supervised exercise training and psychological group counselling significantly improved anxiety, emotions and total quality of life measured using the Quality of Life after MI (QLMI) compared to

standard care. However, these differences were no longer significant at 12 months because anxiety levels and quality of life had significantly improved over time in the control group.

There are several explanations for the lack of long-term effects of exercise rehabilitation on psychological outcomes. Firstly, improvements in psychological functioning and quality of life following MI,[37;66;67] CABG[59] and PTCA[63;79] have been observed in both rehabilitated patients and control patients in the long-term. It is expected that many patients will experience an enhanced quality of life after CABG and PTCA due to relief from angina and increased functional capacity without rehabilitation. Similarly, improvements in quality of life after MI may be due to the natural course of recovery after an infarct. Therefore, it is possible that a short-term comprehensive rehabilitation programme accelerates the improvements in quality of life, whereas without rehabilitation the improvements occur at a slower rate. Secondly, in several studies usual care patients received more contact with health care professionals than normal,[59;67;79] which may have contributed to the gradual improvement in quality of life observed in control patients. Finally, compliance to lifestyle changes may decline after completion of exercise-based cardiac rehabilitation leading to the deterioration in quality of life in the long-term viewed by some researchers.[66;67]

In summary, studies determining the long-term effects of cardiac rehabilitation on psychological outcomes produced inconsistent results. Several authors reported no significant differences between intervention and control groups in psychological functioning and quality of life in the long-term, due to gradual improvements in these outcomes in control patients over time and possibly as a result of poor long-term compliance with lifestyle changes in rehabilitated patients. Furthermore, the instruments used to measure psychological outcomes differ widely (see Table 5), and these variations may account for the inconsistent results observed.

Psychologically Distressed Patients

The greatest psychological benefits of exercise-based cardiac rehabilitation may occur in patients with poor quality of life and high levels of psychological distress at study entry.[108;110;112;113] Lewin[110] randomised 176 patients 3 days after MI to a 6 week home-based comprehensive rehabilitation programme or usual care and

measured psychological distress using the Hospital Anxiety and Depression Scale (HADS) at 6 weeks, 6 months and 1 year. Fifty two percent of the study group were psychologically distressed at baseline, determined by a HADS score ≥ 8 on anxiety or depression subscales. In the distressed group, mean anxiety and depression scores significantly decreased in the rehabilitation group and remained unchanged in the control group at 6 weeks, 6 months and 1 year. In the non-distressed group, mean anxiety and depression scores were low at baseline and did not significantly change in either group during the 12-month study period. Thus, the psychological benefits of exercise-based cardiac rehabilitation were confined to patients who were psychologically distressed immediately after MI.

A large observational study found that depressed patients had a reduced exercise capacity, higher levels of anxiety and hostility (measured using the Symptom Questionnaire) and poorer quality of life (assessed with the Short-Form 36) compared to nondepressed patients who had recently experienced a MI, CABG or PTCA.[108] Following 12 weeks of comprehensive rehabilitation, improvements in anxiety, depression and most subscales of the Short-Form 36 were significantly greater in the depressed group compared to the nondepressed group. Overall, it seems that patients with poor quality of life or suffering from psychological distress following an acute cardiac event may be particularly responsive to the psychological effects of cardiac rehabilitation.

Table 5: Effect of Exercise-Based Cardiac Rehabilitation on Psychological Function

Study	Subjects	Design	Outcomes	Results
Oldridge[37] RCT	N=201 6 wks post MI 90% males, 52 years. All patients were moderately anxious or depressed.	Intervention: 8 wk hospital-based CP. Sup exercise (65% maxHR), twice/week, 50 mins. Weekly gp counselling. Usual care: not described. FU: 8 wks, 4, 8, & 12m.	QoL: Time Trade-off, Quality of life after MI (QLMI) & Quality of Wellbeing (QWB).	Similar, ig, ↑ in all QoL measures in IG & CG at 12m. Sig ↑ in QLMI & anxiety in IG vs CG at 8 wks. QLMI ↑ in 67% of IG & 52% CG at 8 wks ($p < 0.05$). At 12m, 70% of IG & CG had ↑ QLMI.
Stern[66] RCT	N=91, 6wks-12m MI 86% males, 54 yrs. Subjects were high risk.	Intervention: 12 wk out-patient sup exercise training 3 times/wk at 85% maxHR. 12 wks of gp counselling on managing stress & risk factors, relaxation & education. Usual care: told not to engage in sup exercise or counselling. FU: 3, 6, & 12m.	Scaled Interview to Assess Maladjustment (SSIAM), National Institute of Mental Health (NiMH) Self Report Mood Scale.	At 3m, IG had sig lower scores for depression & fatigue (NiMH), less dependent on spouses & more sociable (SSIAM) vs CG. Other QoL changes were similar in IG & CG from 0m to FU. QoL ↑ over time in CG & improvements in IG ↓ from 6 to 12m.
Newton[38] RCT	N=22 6 wks post MI	Intervention:10 wk hospital CP, 3 exercise sessions/wk (2 sup, 1 home), 60-80% maxHR & education on risk factors. Usual Care: Education on risk factors. FU: 10 wks (end of programme).	Beck Depression Inventory (BDI) and Profile of Mood States (POMS).	Anxiety, & depression (POMS) & depression (BDI) sig ↓ in IG vs CG. Sig improvement in anger & total mood disturbance (POMS) in IG vs CG. Sig ↑ in fatigue (POMS) in IG & CG.

Denollet[32] Non-randomised	N=150 3-6 wks post MI or CABG 100% males, 58 years.	Intervention: 3m hospital CP, sup exercise 3 times/wk at 50-75% pkVO_2, psychosocial gp sessions & individualised psychological therapy if needed. Usual Care: not documented. FU: 3m, mortality assessed at 9yrs.	Mortality & emotional distress (positive & negative affect) using Global Mood Scale (GMS).	Sig improvement in negative affect in IG v CG. Greater % of IG improved & smaller % deteriorated in negative affect vs CG. 17% CG & 4% IG died at 9yrs ($p<0.05$). Mortality was associated with deterioration in negative affect.
Engblom[107] RCT	N=228, CABG. 87% males, 54 years (all < 65 years). Patients were randomised pre-CABG.	Intervention: Hospital CP. Phase I: pre-CABG, information on surgery. Phase II: education, 21 hrs of sup exercise for 3 wks & psychology counselling. Phase III: 8m, 2-d refresher of phase II. Phase IV: 30m, 1-d refresher. Usual care: instructions on ↑ activity. FU: 2, 8, 30 & 60 months post CABG.	Beck Depression Inventory (BDI), Nottingham Health Profile (NHP).	Depression was sig lower in IG vs CG at 60m. 30% of IG & 34% CG were depressed (BDI ≥13) pre-CABG. At 60m, 22% IG & 29% CG were depressed ($p>0.05$). Physical mobility (NHP) was sig better in IG vs CG at 60m.
Milani[108] Observation	N=338 4-6 wks post MI, CABG or PTCA. 78% males, 61 years.	Intervention: 12 wk phase II hospital CP. Education on risk factors & psychological adaptation to CHD, sup exercise 3 times/wk, 30-40 mins, 70-85% maxHR & encouraged to exercise unsupervised 1-3 times/wk. FU: 12 wks.	Depression, anxiety & somatisation measured using Symptom Questionnaire. Depression: cutoff > 6. QoL assessed using Short-Form 36 (SF-36).	At 0m, depressed gp had poorer EC, SF-36 & more anxious vs non-depressed gp ($p<0.05$). FU, sig improvement in all scores in depressed gp. Sig greater improvement in scores in depressed vs non-depressed group. 67% of depressed gp were not depressed at FU.

Stahle[67] RCT	N=101 6 wks post MI or unstable angina 75% males, 71 years (>64 years).	Intervention: Hospital sup exercise 3 times/week for 3m & once/wk for a further 3m. After programme, patients were encouraged to attend community classes. Usual Care: Advised to walk daily & attended monthly information meetings. FU: 3 & 12m.	QoL: Disease-specific Karolinska Questionnaire Wellbeing: visual analogue scale.	Several QoL scales ↑ sig in IG and CG at FU. Sig diff between gps at 3m, for symptoms, PA & fitness, no sig diff at 12m 3m changes in IG were not maintained.
Morrin[89] Observation	N=126 > 6 weeks post cardiac event. 74% males, 61 years.	Intervention: 6m hospital CP, education, sup exercise twice/wk, 50-75% HRR & unsupervised activity for 200-400 mins/wk. Set individual goals for risk factors & given psychological counseling if required. Follow-up: 3 & 6m.	QoL assessed by Short-Form 36.	Sig ↑ in SF-36 scales at 3 & 6m, except mental health which sig improved by 6 months, but not 3 m. At baseline, QoL was lower vs USA norms. At 6m, most scales were similar in patients vs USA norms.
Holmback[51] RCT	N=69, 6 wks post MI 97% males, 55 years	Intervention: 12 wk hospital programme, sup exercise, 2 times/wk, 45 mins/session. 70 to 85% HRmax initially, then 100% max HR. Patients told to exercise after intervention. Usual Care: regular medical care. FU: 1 yr post MI.	Questionnaire (not validated) measured QoL (anxiety, depression, self-confidence) & perceived physical performance.	No diff between IG & CG for changes in QoL measures. But the change in QoL from 0m to 1 yr was not reported.

Oldenburg[59] RCT	N=86 4-8 wks post CABG 90% males, 59 years (all<70 years).	Intervention: Hospital CP, 6 weekly gp sessions for 3 hrs & booster sessions at 8 & 12m. Sup exercise & education on smoking, diet & exercise. Behavioural skills included goal setting, skills training, self-monitoring & support. Usual Care: not detailed. FU: 4 & 12m post CABG.	QoL assessed by STAI, depression, anxiety & somatisation scales of SCL-90 & General Health Questionnaire (GHQ).	QoL, subscales of the SCL-90 & % of patients with a high score on GHQ sig improved in IG & CG over time, with no diff between gps.
Higgins[79] RCT	N=99 post-PTCA Majority males, 48 yrs (all <64 yrs).	Intervention: 2m home CP, involved home visits & phone calls, unsupervised walking & behavioural strategies to modify risk factors; goal-setting, self-monitoring, skills training & support. Usual care: education on CHD. FU: During admission, 10 wks & 1 yr.	EC assessed with Seattle Angina Questionnaire (SAQ). Psychological Adjustment to Illness Scale (PAIS-SR).	PAIS-SR & EC sig ↑ in IG & CG over time, with no diff between gps.

Hofman-Bang[63] RCT	N=87 2 wks post PTCA 85% males, 53 years (<65 years).	Intervention: 12-month CP. Phase I: lectures, discussions, skills training, goal setting & relapse prevention for changing risk factors. 11m home programme, regular contact, self-monitoring, problem solving. Usual Care: FU by own physician. FU: 12 & 24m.	Anxiety using STAI & depression using BDI. Angina Pectoris QoL: activity, symptoms, emotional distress & life satisfaction.	No sig diff between gps for anxiety, depression or QoL at 12 or 24m. Many of these outcomes improved in both gps.
Belardinelli[61] RCT	N=118 25 days post PTCA 80% males, 57 years.	Intervention: 6m hospital sup exercise 3 times/week, 30 mins, 60% peakVO_2. Usual care: given a list of suitable "mild" activities & activity diary. FU: 6 & 12m.	QoL measured using Short-Form 36 & Duke Activity Status Index (DASI).	DASI and all SF-36 scales, except social functioning sig ↑ in IG, with no change in CG. Sig between gp diff for the change in SF-36 scores at 6 & 12m.
Sandler[109] RCT	N=215 6 wks post MI 90% males, 55 years (<72 years).	Intervention: 6 wk hospital CP, 2 1-hr sup exercise classes/wk, 1 education & relaxation session/wk, 3 1-hr gp sessions on psychological issues. Usual Care: not detailed. FU: 6, 12, 24 wks	Hospital Anxiety and Depression Scale (HADS), 10 was cut off score for clinical anxiety or depression.	At 0m, 24% IG & 18% CG were clinically anxious. % of anxious patients ↓ to 9% in IG & ↑ to 21% in CG at FU. Anxiety sig ↓ in IG, no change in CG. No change in depression in IG or CG, 3% of patients were depressed at 0m.

Note. QoL = Quality of Life, MI = myocardial infarction, CABG = Coronary artery bypass graft, PTCA = Percutaneous transluminal coronary angioplasty, FU = Follow-up, Gp = Group, IG = Intervention group, CG = Control Group, EC = Exercise capacity, RCT = Randomised controlled trial, m = month, wks = weeks, Sup = Supervised, ↑ = increase, ↓ = decrease, Sig = Significant, Diff = difference, CP = comprehensive programme, QoL = quality of life.

Lipids

Elevated levels of total cholesterol and LDL cholesterol, and low levels of HDL cholesterol are risk factors for recurrent coronary events after a myocardial infarction.[114;115] Randomised controlled trials have demonstrated that a reduction in total cholesterol and LDL cholesterol with lipid lowering therapy significantly decreased mortality, the incidence of non fatal MI and the need for coronary revascularisation in patients with CHD.[116-119] Thus, national guidelines recommend that individuals with CHD should have a total cholesterol and LDL cholesterol level less than 5 mmol/l and 3 mmol/l respectively to reduce the risk of recurrent cardiac events.[2;115] In addition, American guidelines state that triglyceride concentrations should be less than 5.2 mmol/l and HDL cholesterol should be greater than 0.91 mmol/l.[120] To achieve and maintain these lipid targets, interventions to modify diet, increase physical activity and reduce weight are recommended,[120] and lipid lowering medication should be prescribed to individuals with a total cholesterol $\geq$ 5mmol/l or LDL cholesterol $\geq$ 3 mmol/l.[2;115] Although, recent results from the Heart Protection Study indicate that CHD patients should be prescribed lipid lowering medication irrespective of their initial cholesterol level.[119] This study found that a reduction in total cholesterol and LDL cholesterol with medication in individuals with baseline concentrations of total cholesterol and LDL cholesterol below the recommended targets significantly reduced cardiovascular mortality, the incidence of non fatal MI and the need for coronary revascularisation.[119]

Evidence suggests that exercise only rehabilitation has little impact on blood lipids in patients with CHD.[1;28] Recently, a meta-analysis[28] reported that exercise only rehabilitation did not improve total cholesterol, LDL cholesterol, HDL cholesterol and triglycerides compared with usual care, although the results were based on only three studies. In agreement with these findings, a systematic review reported that studies using exercise only rehabilitation documented inconsistent effects on lipid levels.[1] In contrast, a recent randomised controlled trial[61] reported a significant decrease in total cholesterol, LDL cholesterol, triglycerides and a significant increase in HDL cholesterol after 6 months of moderate intensity exercise training compared to non exercising controls in a group of post PTCA patients. An observational study reported that three months of exercise training significantly increased HDL cholesterol by 8%, with no effect on the other lipid levels.[121] Patients with abnormal lipid levels at baseline demonstrated significant improvements with exercise training.

Conversely, comprehensive rehabilitation programmes, which combine dietary modification and exercise training, are effective in managing lipids in patients with CHD.[1;28;47;79;84;89;93;108;122;123] Results of a meta-analysis[28] found that comprehensive rehabilitation substantially decreased total cholesterol and LDL cholesterol, produced a small significant decrease in triglyceride levels and had no significant effect on HDL cholesterol compared to usual care. Similarly, a systematic review[1] reported that the majority of randomised and non-randomised controlled trials of comprehensive rehabilitation, which included exercise training, and dietary intervention documented significant improvements in total cholesterol, LDL cholesterol and triglyceride levels. Whereas, few trials recorded significant increases in HDL cholesterol with comprehensive rehabilitation compared to usual care.

Improvements in lipid levels with comprehensive rehabilitation viewed in several studies were influenced by increased use of lipid lowering medication during the study period in the intervention group.[47;89;93;123] Debusk found that 12 months of home-based comprehensive rehabilitation was more effective than usual care in reducing total cholesterol and LDL cholesterol, and significantly more intervention patients had an LDL cholesterol value below the recommended level at 6 and 12 months.[47] In contrast, there was no significant difference between groups for triglyceride or HDL cholesterol levels. However, a substantial proportion of patients in the intervention group (83%) had been prescribed lipid-lowering medication during the study period compared to the patients receiving usual care (20%). The authors attributed most of the reduction in total cholesterol and LDL cholesterol to the medication effects. Other trials documenting improved lipid profiles with exercise-based cardiac rehabilitation have not reported whether changes in lipid lowering medication occurred over the study period, although this factor may have confounded the results.[55;79;108;122]

The evidence suggests that cardiac rehabilitation exercise training is not recommended as a sole intervention for lipid modification because of its inconsistent effects on lipid levels. Comprehensive cardiac rehabilitation, which includes dietary advice, lipid-lowering therapy and exercise training is the most effective programme to produce favourable lipid profiles and reach national lipid targets in patients with CHD.[9]

Adherence to Exercise-based Cardiac Rehabilitation

Adherence (or compliance) is the extent to which recommendations (e.g. exercise, diet or medication) to patients by health professionals are followed.[1]

Supervised Exercise Programmes

In the UK, the majority of phase III cardiac rehabilitation programmes involve group exercise training for 5 to 13 weeks.[5] It has been estimated that between 9% to 49% of cardiac patients drop out of supervised rehabilitation exercise programmes lasting 12 weeks or less.[18;43;44] In addition, approximately 40% to 50% of patients dropout of longer-term supervised exercise programmes lasting 6 months or longer.[1;124-127] These dropout rates are similar to attrition rates from exercise programmes in the general population.[128] Additionally, Dorn[127] reported that adherence to a 3 year cardiac rehabilitation exercise programme declined over time and only a small proportion of patients were attending the programme at 3 years. However, a dropout from a supervised exercise programme does not necessarily mean that the patient has stopped exercising. Oldridge[19] found that 50% of dropouts from a 6-month supervised exercise programme continued to exercise regularly. One third of the dropouts stated that they discontinued the supervised programme because they felt better and had gained what they had wanted from the exercise classes, thus 6 months of supervised exercise training was not required for all patients.

Factors Influencing Adherence to Supervised Exercise Programmes

A number of demographic, medical and psychosocial variables have been shown to influence adherence to supervised cardiac exercise programmes. Factors associated with dropout from supervised exercise programmes include having a blue-collar occupation,[19;129;130] angina,[130] a reduced ejection fraction,[131] inactive leisure habits,[19;129] greater socio-economic deprivation,[24] and currently smoking.[19;129;130] Dorn[127] observed lower attendance rates at supervised exercise for men with a less favourable CHD risk factor profile; such as a current smokers, lower exercise capacity, higher levels of cholesterol and triglycerides and a BMI greater than 25. Common reasons for programme dropout or poor attendance at group exercise cited by patients included transport problems,[18;43] medical problems,[43;130] work or domestic commitments,[18;26;43;44] lack of motivation or interest,[26;130] inconvenient timing of programmes[26] and beliefs

that rehabilitation would not be beneficial.[18] Presence of anxiety or depression have been shown to be important predictors of non-compliance to cardiac rehabilitation exercise programmes.[103;104;131] A large observational study found that depressed patients were twice as likely to dropout of cardiac rehabilitation compared to nondepressed patients.[104] Age and gender have not shown any consistent pattern in predicting who will adhere. Higher dropout rates and lower adherence rates to cardiac rehabilitation have been reported for women compared to men,[44;132;133] although these findings have been inconsistent.[23;43] Several studies found that age did not influence adherence to cardiac rehabilitation[103;104;125;131] whereas, Dorn[127] reported that older men were more compliant than younger men. Oldridge[130] also found that younger patients were more likely to dropout of supervised exercise.

Maintenance of Physical Activity after Programme Completion

Several trials have shown a decline in patients' physical activity levels, fitness and quality of life following completion of cardiac rehabilitation exercise programmes.[19;20;56;67;79;91;94-96;98] Furthermore, the majority of patients do not continue to exercise regularly after cessation of supervised exercise training.[20;56;87;94;95]

Factors Influencing Maintenance of Physical activity

As previously discussed, numerous studies have identified factors that predict dropout and adherence in supervised exercise programmes. In contrast, the research is limited on the factors that influence maintenance of exercise and physical activity following programme completion. Understanding these factors is an important step in the development of interventions to improve maintenance of physical activity and exercise in the long-term.

Patients are expected to remain regularly active in the long-term in phase IV after completion of phase III exercise programmes. However, the transition from phase III to phase IV is a challenging time for cardiac patients as they do not receive the support and follow-up from cardiac rehabilitation staff that they received during phase III.[2] Instead, they have to maintain physical activity and other health behaviours on their own. Membership of cardiac support groups, which offer group exercise or attending cardiac maintenance programmes delivered by BACR trained exercise leaders may help patients to remain active in this phase. However, these exercise opportunities are not available in all areas. Furthermore, many patients

would not be able to attend community programmes due to barriers associated with supervised exercise training including transportation problems, limited access, and work and domestic conflicts. In addition, the effectiveness of phase IV exercise programmes to maintain the benefits gained during phase III exercise training including exercise capacity and increased physical activity has not been assessed in the UK.[2]

Improving Maintenance of Physical Activity in Cardiac Rehabilitation

In the UK, the majority of phase III cardiac rehabilitation programmes involve supervised exercise training lasting 6 to 12 weeks. Although supervised exercise is important to teach patients to self-monitor their exercise intensity and increase patients' confidence for exercise, it is unlikely that participation in a supervised exercise programme will facilitate independent exercise after programme completion.[2] This is reflected by the low proportion of patients who continue to engage in regular physical activity in the long-term after completion of supervised exercise programmes. In addition, high dropout rates from supervised exercise programmes is associated with barriers such as; the programmes may be inconvenient or may interfere with work or domestic commitments, the location may be difficult to access (especially in rural areas), and even if accessed, parking or transport may cause problems. It is now recognised that this restricted approach to exercise prescription does not meet the varied needs and goals of the CHD population. Therefore alternative models to traditional exercise-based cardiac rehabilitation have been proposed.

Studies have found that home-based exercise programmes for low to moderate risk patients are as effective as supervised exercise training.[41;80;88;179] Other trials have reported significant improvements in exercise habits, functional capacity and blood lipids with home based comprehensive cardiac rehabilitation compared with usual care.[47;79;86;110] Therefore, home exercise programmes may overcome some of the barriers associated with supervised exercise and improve exercise adherence. In addition, SIGN guidelines on cardiac rehabilitation recommend that participants of phase III supervised exercise programmes should also incorporate moderate physical activity into their daily lifestyle to produce better adherence to exercise in the long-term.[2]

In a recent study, Carlson[99] compared the effect of a traditional phase III programme, involving three supervised exercise sessions a week for 6 months, with a modified phase III programme designed to enhance patients' self-efficacy for independent exercise. The study found that both interventions produced significant and comparable improvements in body weight, exercise capacity, and blood lipids at 6 months. However, significantly fewer participants dropped out of the modified programme (8%) compared to the traditional programme (24%). In addition, the total number of exercise sessions and the mean number of sessions per week were significantly higher for patients in the modified programme compared to the traditional programme. These results show that encouraging phase III participants to exercise independently and enhance their self-efficacy for independent exercise improves exercise adherence. The high dropout rates from facility-based exercise highlights the importance for developing independent exercise behaviour in cardiac patients so that physical activity can be maintained in the long-term. Thompson[180] recommended that low risk cardiac patients should move rapidly from hospital-based exercise training to unsupervised exercise (when they are able to self-monitor their exercise intensity) with regular support from rehabilitation staff to ensure that increased exercise levels are carried over from formal rehabilitation programmes.

The study by Brubaker is the only trial to investigate the efficacy of phase IV exercise programmes.[181] This study randomized 31 patients, who had completed a 12 week phase III supervised exercise programme to either a 9 month phase IV home-based maintenance programme or standard care. In addition, a comparison group (n = 17) who attended a phase IV supervised maintenance programme after completion of phase III were randomly selected. The phase IV home-based programme lasted 9 months and included an initial one to one discussion with the patient on monitoring of exercise intensity, self-monitoring of daily activity levels, problem solving exercise barriers, and regular telephone contact to discuss exercise compliance, feedback on exercise levels and to provide support and encouragement. Standard care involved advice on continuing with exercise and other lifestyle modifications. The comparison group attended a phase IV supervised maintenance programme for 9 months, involving supervised exercise training three times a week and regular counselling on exercise compliance and modification of other risk factors. Exercise capacity, blood lipids, and body composition were assessed before and after phase III and at 9 months follow-up. Results revealed

comparable, significant improvements in these outcomes in all 3 groups on completion phase III. In addition, these improvements in blood lipids, exercise capacity and body composition achieved in phase III were maintained in all groups at 9 months follow-up. Thus, although the standard care patients received no further intervention in phase IV, they also maintained these changes. However, a number of patients stated knowing that they were being reassessed at 12 months motivated them maintain lifestyle changes. These findings suggest that a home-based exercise programme is just as effective for patients who cannot attend a phase IV supervised maintenance exercise programme. The fact that the standard care patients did just as well suggests that follow-up testing alone may be sufficient to maintain lifestyle changes after phase III.

SUMMARY OF SECTION ONE

Overall, there are many physiological and psychological benefits to be gained by participating in exercise-based cardiac rehabilitation following MI, CABG and PTCA. Achieving these benefits depend on good adherence to cardiac rehabilitation programmes. In addition, sustaining these benefits and preventing further cardiac events requires maintenance of regular physical activity and other lifestyle changes in the long-term. However, the evidence suggests that improvements in exercise capacity, physical activity and quality of life decline over time following programme completion. Many studies have examined the factors affecting dropout and adherence to supervised exercise programmes. Whereas, the research is limited on the factors that contribute to maintenance of physical activity following programme completion. Understanding these factors is an important step in the development of interventions to improve maintenance of physical activity and exercise in the long-term.

SECTION TWO

PHYSICAL ACTIVITY BEHAVIOUR CHANGE

Theoretical Models of Behaviour Change

In a recent review, Biddle states that predicting, understanding and explaining behaviour should be carried out within a theoretical framework.[134] A number of theoretical models of behaviour change have been used to understand adherence to exercise and physical activity in the general population. These include; the Health Belief Model, Theory of Reasoned Action, Theory of Planned Behaviour, Social Cognitive Theory, Decisional Balance, Relapse Prevention Model, and the Transtheoretical Model. Several of these models have also been used to understand participation in physical activity in cardiac rehabilitation settings. In addition, these models have provided a theoretical framework for the development of effective interventions to enhance adherence to exercise and physical activity in the general population.[134] There is little research on effective strategies to improve exercise adherence in cardiac rehabilitation settings.

Health Belief Model

Oldridge used the Health Belief Model (HBM), which predicts the likelihood of an individual taking recommended health action to prevent or control a disease,[135] to predict compliance and dropout in a 6 month cardiac rehabilitation exercise programme.[129] Results revealed that the components of the HBM; barriers to exercise, benefits of exercise, and susceptibility of disease were not significantly different between compliers and dropouts. In contrast, cues to exercise were significantly associated with compliance to cardiac rehabilitation. The life threatening nature of coronary heart disease may serve as a cue to initiation of exercise behaviour change for many patients. Furthermore, the severity of disease threat was significantly associated with compliance to the exercise programme, but in the opposite direction to that proposed by the HBM. The threat of disease was greater for dropouts compared to compliers, thus individuals who perceived their condition as more severe were less likely to comply with the exercise programme. In general, the study found that the HBM was of limited use in predicting compliance to exercise-based cardiac rehabilitation. Similarly, Biddle and Mutrie stated that the HBM has been somewhat unsuccessful in

predicting adoption and maintenance of physical activity and exercise in a variety of populations.[136] This may be because many people adopt and adhere to exercise for motives other than to prevent or control a disease.

Theories of Reasoned Action and Planned Behaviour

The theory of reasoned action (TRA)[137] proposes that behaviour is directly determined by an individual's intention to perform a behaviour. Intention is determined by two variables: 1) a person's attitude, which is indicated by the person's beliefs about the behaviour, as well as the evaluation of the likely outcomes of the behaviour; 2) subjective (social) norm, which is the beliefs of significant others (e.g. family, friends or health professionals) and the extent to which the individual is motivated to comply with such beliefs or people. This theory has been used to predict attendance at exercise-based cardiac rehabilitation.[22] Cooper found that 72% of cardiac rehabilitation patients stated that they intended to participate in cardiac rehabilitation, however the attendance rate was only 40%.[22] This finding suggests that intention alone may not predict future health behaviour. This may be because even though many people have a strong intention to exercise, there are barriers that may interfere with their ability to do so.

The Theory of Planned Behaviour (TPB)[138] extends the TRA by adding the factor of perceived behavioural control. Perceived behavioural control is defined as the perceived ease or difficulty of performing the behaviour, and takes into account barriers that may prevent individuals from performing the behaviour. A recent meta-analysis has provided support for the utility of the TBP to predict exercise behaviour in the general population.[139] Results revealed that attitude towards exercise and perceived behavioural control are important determinants of both intention to exercise and actual exercise behaviour. In addition, intention to exercise had a substantial effect on exercise behaviour, whereas subjective norms had a moderate effect on intention, and no effect on behaviour.

Blanchard[140] found that the TPB was useful for understanding exercise adherence during an 8-week phase II cardiac rehabilitation exercise programme. This study reported that attitude towards exercise, subjective norms and perceived behavioural control were significant predictors of exercise intention (i.e. intention to attend scheduled exercise sessions). In addition, exercise intention was the sole significant

predictor of adherence to the phase II exercise programme. However, as exercise intention explained only 12% of the variance in exercise adherence in this study, it seems that other factors may explain additional variance in exercise adherence during phase II cardiac rehabilitation. This theoretical model has not been used to understand exercise adherence after completion of cardiac rehabilitation exercise programmes.

Social Cognitive Theory

The Social Cognitive Theory (SCT) proposes that personal (e.g. cognitions, body composition, age), environmental (e.g. social support, weather, access to facilities, stimulus control) and behavioural (e.g. intensity, and frequency of physical activity) factors influence behaviour.[136] An important component of the SCT, social support, has been found to influence participation in cardiac rehabilitation.[132;141] A significant relationship between exercise adherence and perceived benefits and barriers to exercise has been demonstrated in patients with CHD.[142-144] Tirrell reported that individuals who perceived a greater number of benefits and fewer barriers to exercise reported greater adherence to exercise between 6 and 18 months following CABG.[143] Self-efficacy is defined as an individual's confidence in his or her ability to perform a specific behaviour.[145] There is a significant positive relationship between self-efficacy and compliance to exercise in cardiac rehabilitation settings.[95;142] A cross-sectional study examined the relationship between exercise self-efficacy and exercise adherence in a group of cardiac patients who had completed a 12-week phase II supervised exercise programme.[95] Questionnaires were used to assess exercise behaviour, and two components of self-efficacy; the individual's perceived ability to perform daily living activities (e.g. walking, jogging, climbing stairs etc) and the individual's perceived ability to continue to exercise regularly over the next 3 months despite certain barriers (exercise barriers efficacy). The study found that both components of self-efficacy were significantly higher for individuals who continued to exercise regularly following programme completion. Results suggest that identifying barriers to exercise and influencing sources of self-efficacy to overcome exercise barriers could improve maintenance of exercise following completion of supervised rehabilitation programmes.

Overall, it seems that self-efficacy, social support and perceived barriers to exercise have important influences on adherence to exercise in cardiac rehabilitation. Similarly, these factors are significant determinants of physical activity behaviour in the general

population.[128]

Interventions Based on Social Cognitive Theory (SCT)

General Population

Interventions based on the SCT have improved adherence to exercise in healthy subjects. Strategies such as enhancing social support,[146;147] goal setting,[148] decision balance-sheet techniques[146;149] and relapse prevention training[147;150] improved attendance at exercise programmes and maintenance of exercise following programme completion. Decisional balance-based interventions, which involve comparing the perceived costs (cons) and benefits (pros) of engaging in a behaviour, have been shown to increase attendance at supervised exercise programmes[146] and was effective in maintaining attendance at a fitness centre.[149] Providing individuals with personalised feedback and praise during exercise and involving individuals in setting flexible exercise goals significantly increased attendance at a 10 week exercise programme and enhanced maintenance of exercise for three months after termination of the exercise programme.[148]

Cardiac Rehabilitation

Several researchers have incorporated strategies based on the SCT into their cardiac rehabilitation programmes.[47;59;62;79;93;99;151;152] However, some of these studies[47;59;62;79;93] have used SCT strategies to modify multiple health behaviours, thus it is not always clear whether these techniques were applied to exercise in cardiac rehabilitation. Carlson[99] compared the effect of a traditional phase III rehabilitation programme, involving three supervised exercise sessions per week for 6 months, with a modified phase III programme that was designed to enhance patients' self-efficacy for independent exercise. At the start of the modified programme, participants attended three supervised exercise sessions a week, however the frequency of supervised sessions was gradually reduced to one supervised session every two weeks at 6 months to facilitate independent exercise. Participants of both programmes were encouraged to exercise for 30 minutes five times per week (either supervised or independently). At 6 months, both interventions produced significant and comparable improvements in body weight, exercise capacity, and blood lipids. However, adherence to the exercise recommendations (i.e. 5 x 30-minute sessions/week) was significantly greater for modified programme participants compared to participants of the traditional programme. These results

suggest that enhancing a participant's self-efficacy for independent exercise improves exercise adherence in phase III. It would have been interesting to assess exercise behaviour during phase IV to determine whether the modified phase III programme was superior to traditional rehabilitation for improving maintenance of exercise after phase III.

Debusk[47] reported a significant improvement in exercise capacity after a six-month home-based rehabilitation programme, which used SCT techniques to modify CHD risk factors (e.g. smoking, diet and exercise). The strategies used were self-monitoring, feedback, goal setting, relapse prevention training, problem-solving barriers, behavioural contracting and social support. However, as physical activity and exercise capacity were not assessed in the long-term after programme completion, it is not possible to tell if this programme was effective in maintaining changes in activity and fitness. Lisspers[62] reported that a 12-month rehabilitation programme following PTCA, which used health education and behavioural strategies derived from SCT to modify CHD risk factors was more effective than usual care in improving exercise capacity and exercise habits at 12 months. Furthermore, these improvements in fitness and exercise participation were maintained 12 months after the end of the intervention period.[63] In contrast, other studies have shown that short-term comprehensive programmes based on the SCT had no lasting effect on physical activity habits at 12 months.[59;79]

Relapse Prevention Model

Relapse is a breakdown or set back in a person's attempt to change or modify a target behaviour. The relapse prevention model was developed to treat addictive behaviours such as alcoholism and smoking.[153] The model proposes that relapse may result from an individual's inability to cope with situations that pose a risk of return to the previous behaviour. For example, a former smoker finds himself or herself in a social situation with lots of smokers. Thus, helping the individual to acquire strategies to cope with high-risk situations will both reduce the risk of an initial lapse, and prevent any lapse from escalating into a total relapse. Simkin[154] assessed coping responses to high risk situations for exercise relapse (e.g. negative mood, boredom, lack of time) in 29 healthy women who had adopted exercise without formal intervention. The womens' activity levels were measured weekly for 14 weeks. The study found that 66% of participants experienced a lapse (defined as not exercising for 1 week) and 41%

experienced a relapse (defined a not exercising for 3 or more consecutive weeks) over the 14 monitored weeks. Participants who experienced a relapse reported significantly fewer behavioural and cognitive strategies to cope with high-risk situations compared to participants who did not relapse. These findings suggest that acquiring effective strategies to cope with exercise barriers may prevent relapse.

The relapse prevention model has been used to improve exercise adherence in the general population.[147;150;155] Belisle reported that relapse prevention training increased attendance at a ten week exercise programme and improved maintenance of exercise for 12 weeks following programme completion.[150] King[155] evaluated the effect of self-monitoring and relapse prevention techniques on maintenance of physical activity for 6 months in a group of healthy subjects who had completed a six month programme of moderate intensity home-based exercise. Fifty-one subjects were randomised to receive strategies for maintaining exercise adherence, including daily self-monitoring of activity and relapse prevention or a comparison group who underwent weekly self-monitoring of activity. The results revealed that the intervention group engaged in significantly more exercise sessions over the 6-month period compared to the comparison group. Therefore, daily self-monitoring of activity levels and relapse prevention training improved exercise adherence.

The research suggests that relapse prevention training is an effective strategy for improving exercise adherence in the general population. Despite the high dropout rates from cardiac rehabilitation exercise programmes and poor long-term maintenance of physical activity following programme completion, there is limited research on the effect of relapse prevention training on exercise adherence in cardiac rehabilitation settings. Relapse prevention training[154] involves teaching individuals that a lapse from exercising (e.g. missing an exercise session) need not lead to a relapse (e.g. missing a week without exercising) and a lapse can be prevented from escalating into a complete relapse (e.g. return to a sedentary lifestyle). The individual is encouraged to identify situations that are likely to lead to a lapse. Potential high-risk situations relevant to exercise include bad weather, an increase in work commitments, change in routine, injury or illness. Individuals are encouraged to develop a plan to cope with these high risk situations; for example the plan for coping with bad weather could be using exercise video tapes, increased work commitments could be overcome by rescheduling an activity session or engaging in a shorter bout of activity. Such

coping is believed to prevent escalation of a lapse into a relapse.

Transtheoretical Model (TTM)

The Transtheoretical Model (TTM) proposes that individuals attempting to change their physical activity behaviour progress through five stages of change.[156] The stages differ according to an individual's intention and behaviour, and have been labelled; Precontemplation (inactive and no intention to change), Contemplation (inactive, but intending to change in the next 6 months), Preparation (engaging in some activity, but not regularly), Action (regularly physically active, but only began in the past 6 months), Maintenance (regularly active for more than six months). Thus, according to this model, maintenance of physical activity is defined as being regularly active for at least 6 months. Movement through these stages often occurs in a cyclic pattern because many individuals relapse back to an earlier stage when attempting behaviour change. In fact, many individuals progress and regress through the stages of change several times before reaching maintenance. Action is an unstable stage during which individuals are at high risk of relapse.

Studies have reported an increase in self-reported physical activity and cardiorespiratory fitness with advancing stage of change in healthy adults and cardiac rehabilitation participants.[96;144;157-159] A recent meta-analysis found an increase in physical activity as individuals moved to a higher stage of change.[159] The largest increase in physical activity occurred as subjects moved from preparation to action, this is the point at which individuals engage in regular physical activity.

Three components of the TTM are hypothesised to mediate the behaviour change process. These are the processes of change, the decisional balance and self-efficacy.

The processes of change are strategies that people use when changing their exercise behaviour.[160] There are ten processes, which have been grouped into five experiential and five behavioural processes and a description of each is provided in Table 6. In 1992, Marcus developed a questionnaire to measure the use of each process of change for individuals who were in different stages of change for exercise behaviour.[160]

Table 6: Processes of Exercise Behaviour Change[160]

Process of Change	Definition
Experiential	
Consciousness raising	Efforts by the individual to seek information and gain understanding and feedback about physical activity (e.g. reading articles about exercise to learn more on it).
Dramatic relief	Emotional experiences related to physical inactivity and its consequences (e.g. warnings about the health hazards of inactivity emotionally affect the person).
Environmental reevaluation	Evaluation by the individual of how inactivity affects the physical and social environment (e.g. the person wonders how their inactivity affects others).
Self-reevaluation	Emotional and cognitive reevaluation by the individual of the values of physical activity (e.g. what are the benefits of physical activity to me?).
Social liberation	The individual's awareness, availability and acceptance of physical activity in society (e.g. awareness of changes in society that make it easier for the exerciser).
Behavioural	
Counterconditioning	Substituting situations of inactivity for physical activity (e.g. taking the stairs instead of the lift).
Helping relationships	Trusting and using the support of others during attempts to change physical activity behaviour (e.g. having a friend who encourages the person to exercise).
Reinforcement management	Changing the contingencies that control or maintain inactivity (e.g. the person rewards himself or herself when they exercise).
Self-liberation	The individual's commitment to physical activity (e.g. the individual makes commitments to exercise).
Stimulus control	Control of situations that trigger inactivity (e.g. individual removes things that contribute to inactivity).

A recent meta-analysis[159] found that use of the processes of change vary across the five stages of change, as depicted in Figure 1. The use of experiential and behavioural processes increases with advancing stage, with the largest increase occurring from precontemplation to contemplation and preparation to action. Furthermore, the use of behavioural processes is more important than experiential processes from the contemplation stage onwards. There is little change in process use from the action to maintenance stages, implying that maintenance of physical activity does not require further changes in experiential and behavioural strategies, or that individuals use additional strategies to those proposed by the processes of change. Similarly, an observational study of patients who had previously attended a cardiac rehabilitation programme found that the experiential and behavioural processes were used more frequently with advancing stage of exercise behaviour change.[144] In contrast, Jue reported no significant difference in process use among the five stages of exercise behaviour change in a group of elderly patients after CABG.[161] However, the process of change questionnaire was developed for a younger, working population, and therefore may not be valid for an elderly, retired population.

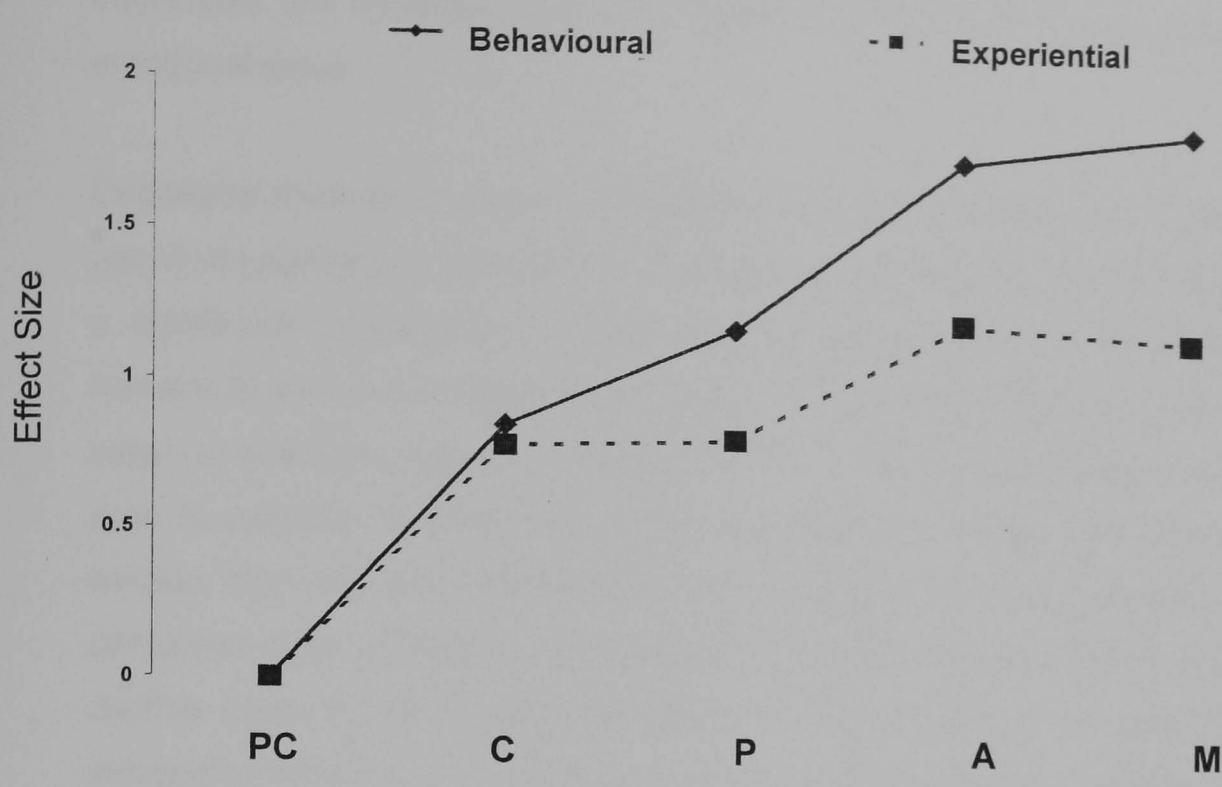


Figure 1 Relationship between the stages and processes of exercise behaviour change[159]

PC = Precontemplation, C = Contemplation, P = Preparation, A = Action, M = Maintenance.

A longitudinal study measured changes in the stages and processes of change for exercise behaviour from baseline to 6 months in a group of healthy subjects.[162] At 6 months, subjects were categorised into four groups; stable sedentary (remained in either precontemplation or contemplation at both assessments), stable active (remained in preparation, action or maintenance at both assessments), adopters (progression from precontemplation, or contemplation to preparation, action or maintenance), and relapsers (regression from preparation, action or maintenance to either contemplation or precontemplation). This study found that process use did not change for subjects in the stable active or stable sedentary categories. However process use was significantly greater for subjects who remained active compared to those who stayed inactive over the study period. Subjects in the adoption group reported a significant increase in the use of experiential and behavioural processes, whereas those in the relapse group reported a significant decline in the use of all behavioural processes and 1 experiential process (dramatic relief). These findings suggest that continued use of behavioural strategies may be important to prevent relapse. Furthermore, a significant decline in dramatic relief among relapsers suggests that belief in the health benefits of physical activity may decrease considerably when

individuals are no longer physically active or that inactivity is no longer seen as an emotional issue.

Decisional balance involves a comparison of the perceived costs (cons) and benefits (pros) of engaging in a behaviour. As previously described, studies have demonstrated a significant relationship between exercise adherence and perceived benefits and barriers to exercise in patients with CHD.[142-144] Decisional balance is also related to the stage of exercise behaviour change.[144;159;163;164] The meta-analysis[159] reported that the pros of exercise increase with advancing stage of change, with the largest increase evident from the precontemplation to the contemplation stage (see Figure 2). The perceived cons of change decreased across the stages, with the most pronounced decline occurring from precontemplation to contemplation. Therefore, it seems that increasing the pros of exercise and decreasing the cons to exercise are important to enhance behaviour change. Similarly, Hellman reported a decline in the perceived barriers to exercise and an increase in the perceived benefits of exercise with advancing stage of change in a group of patients who had previously attended in-patient cardiac rehabilitation.[144]

Self-efficacy was integrated into the TTM from Bandura's Self-Efficacy Theory,[145] and is defined as an individual's confidence in his or her ability to perform a specific behaviour. As previously discussed, self-efficacy is an important determinant of exercise compliance in cardiac rehabilitation.[95;142] Findings from a recent meta-analysis demonstrated a significant relationship between exercise self-efficacy and stage of change, as illustrated in Figure 3. The graph shows that exercise self-efficacy increases with each forward movement in stage of change. Individuals in the precontemplation stage demonstrate the lowest self-efficacy, whereas those in maintenance have the highest self-efficacy. Furthermore, the relationship between exercise self-efficacy and stage of change is nonlinear, and self-efficacy seems to be especially important when moving from action to maintenance. Similarly, Hellman[144] reported that exercise self-efficacy is significantly related to stage of exercise behaviour change in cardiac rehabilitation participants.

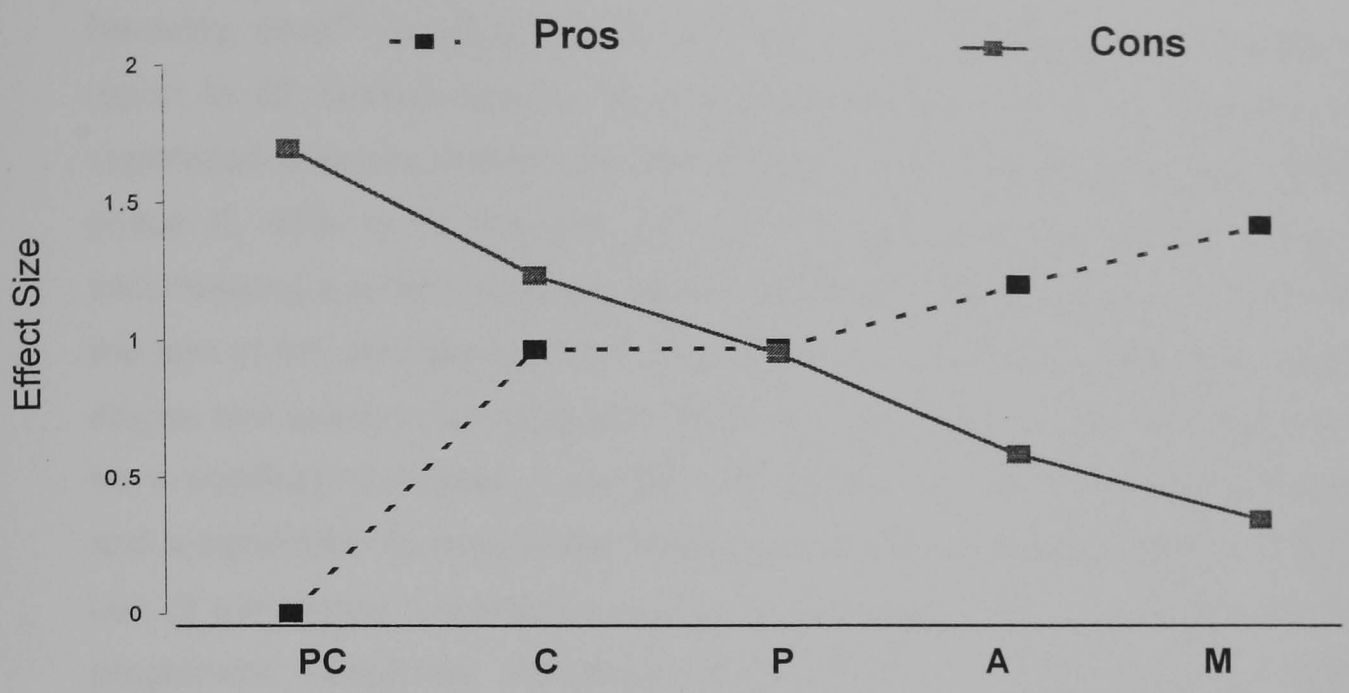


Figure 2 Relationship between decisional balance and the stages of exercise behaviour change.[159]

PC = Precontemplation, C = Contemplation, P = Preparation, A = Action, M = Maintenance.

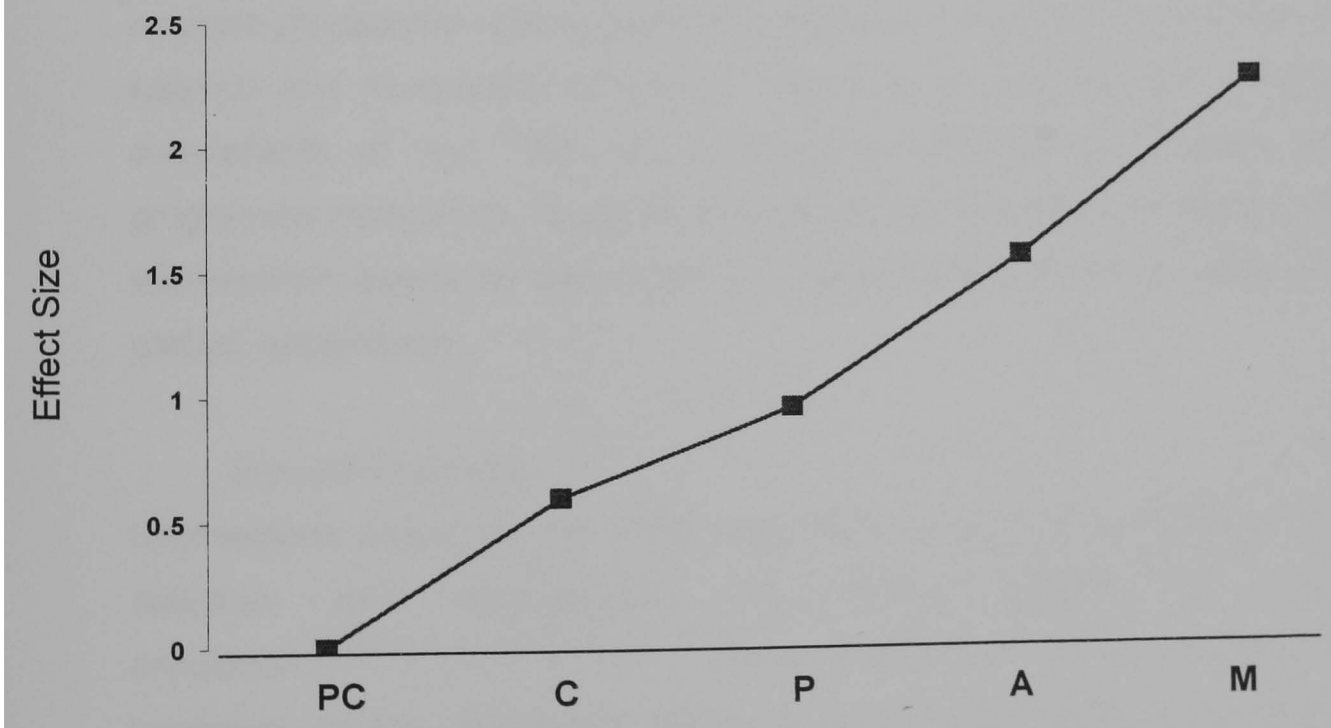


Figure 3 Relationship between self-efficacy and the stages of exercise behaviour change.[159]

Application of the Transtheoretical Model

Cardiac Rehabilitation

Recently, Bock[96] measured the components of the TTM and physical activity by self-report in 62 cardiac patients at the beginning and end of a 12 week phase II supervised exercise programme and at three months follow up. At the beginning of phase II, 43% of participants were in the action and maintenance stages (i.e. accumulating a weekly minimum of 30 minutes of moderate activity on most days). At the end of the programme, 96% of participants were in the action and maintenance stages and self-reported physical activity had significantly increased. Moreover, there were significant increases in exercise self-efficacy, the use of behavioural processes, and a significant reduction in the perceived barriers to exercise, with no change in the use of experiential processes or perceived benefits of exercise. Three months after programme completion, the proportion of patients in the action and maintenance stages had decreased to 80% and nearly 50% of participants had reduced their physical activity compared to the end of the programme. Individuals who had regressed at the 3 month follow-up had significantly lower scores for self-efficacy, use of behavioural processes, and more negative decisional balance scores at the end of the programme compared to participants who remained physically active at 3 months. Thus, maintenance of exercise behaviour after completion of a cardiac rehabilitation exercise programme appears to be associated with changes in self-efficacy, decisional balance and behavioural processes. The findings suggest that enhancing these components of the TTM may promote maintenance of physical activity after programme completion. However, there is limited research investigating the effect of interventions based on this model to improve maintenance of physical activity in cardiac rehabilitation.

General Population

Interventions based on the TTM have been shown to be effective in improving adoption and maintenance of physical activity in the general population.[60;74;77;165;166;166;167;167;168;168] These Interventions use the concept of matching treatment to the individual's stage of change for exercise behaviour. Marcus randomised 194 sedentary adults to receive either an individualised, stage-matched intervention or a standard intervention over a 6-month period.[167] The stage-matched intervention involved providing participants with individualised feedback about their physical activity behaviour and stage-matched self-help manuals, which were designed

to enhance the components of the TTM. The standard intervention involved providing participants with standard self-help booklets to promote physical activity, developed by the American Heart Association. A significantly greater proportion of participants in the stage-matched group were regularly active and had reached the action stage compared to individuals receiving standard treatment at 6 months. In addition, self-reported physical activity had increased significantly more in the stage-matched group compared to the standard group at 6 months. In a follow-up to this study, a significantly greater proportion of participants who had received the stage-matched intervention were regularly active and were in the action or maintenance stages 6 months after the intervention period compared to subjects who received the standard intervention.[168] These findings suggest that an intervention tailored to an individual's stage of exercise behaviour change was more effective than a standard intervention in improving adoption and maintenance of physical activity in a group of sedentary healthy adults.

In summary, the transtheoretical model proposes that by identifying an individual's stage of exercise behaviour change, key components such as the processes of change, exercise self-efficacy and decisional balance can be influenced to encourage stage progression and relapse prevention. For example, maintaining physical activity and preventing relapse may require continued use of behavioural processes and enhancing self-efficacy.

Overall, several theories have been used to understand and explain adherence to exercise in healthy subjects and in cardiac rehabilitation settings. These theories have identified a number of factors influencing participation in physical activity including: social support, exercise self-efficacy, perceived behavioural control, perceived benefits of exercise, perceived barriers to exercise, ability to cope with high risk situations, and use of cognitive and behavioural processes. In addition, the evidence suggests that interventions based on theoretical models of behaviour change such as social cognitive theory, the relapse prevention model and the transtheoretical model are effective in increasing and maintaining physical activity in the general population. Effective compliance enhancing strategies include; goal setting, decision balance sheets, enhancing exercise self-efficacy and social support, self-monitoring, written agreements, relapse prevention training, and enhancing the use of cognitive and behavioural processes. In contrast, there is little research on the effect of theory-based

interventions to encourage maintenance of physical activity after completion of formal cardiac rehabilitation.

Physical Activity Counselling

Recent cardiac rehabilitation guidelines[9] by the American Heart Association and the American Association of Cardiovascular and Pulmonary Rehabilitation recommend that physical activity counseling should be a core component of cardiac rehabilitation programmes to promote an active lifestyle for patients with CHD. Physical activity counselling should include an evaluation of the individual's current physical activity level, stage of change, self-efficacy, barriers to increasing physical activity and social support in making positive changes. Interventions should include providing support, advice and counselling about physical activity needs, and set goals to increase physical activity that include 30 minutes per day of moderate physical activity on ≥5 days a week. In addition, patients' daily schedules should be explored in order to suggest how to incorporate physical activity into their daily routine; e.g. parking farther away from entrances, walking up 2 or more flights of stairs and walking for 15 minutes during lunch breaks.

Physical activity counselling is based on the transtheoretical model of behaviour change and social cognitive theory. In addition, this approach is closely linked with the 1995 physical activity recommendations of accumulating 30 minutes of moderate intensity physical activity on most days of the week. Thus, these interventions use stage-matched strategies to encourage participants to integrate moderate intensity physical activity into their daily lives. Physical activity counselling has produced improvements in stage of exercise behaviour change and physical activity levels in initially sedentary healthy adults.[169-173]

Exercise Consultation

In 1995, Loughlan and Mutrie[174] developed a set of guidelines on exercise counselling of sedentary adults. This intervention, known as the exercise consultation, is a client-centred one to one counselling approach. It is based on the transtheoretical model and social cognitive theory and comprises strategies to increase and maintain physical activity. Several randomised-controlled trials have investigated the effect of the exercise consultation on physical activity adoption and adherence. Loughlan and Mutrie[175] showed that the exercise consultation was as

effective as standard fitness assessments in increasing physical activity at 3 months. Though these initial increases in activity were not maintained at 6 months in the fitness assessment group, the exercise consultation group were significantly more active at 6 months compared to baseline. In a similar study, Lowther and Mutrie reported that participants who had received an exercise consultation remained more active compared to participants receiving the fitness assessment, who had regressed to baseline values at 1-year follow-up.[176] Kirk et al found in a group of sedentary people with type 2 diabetes that an exercise consultation significantly increased physical activity assessed by self-report and accelerometry from baseline to six months compared to standard exercise information.[177] In addition, these improvements were maintained from six to 12 months in the exercise consultation group. Results from these trials suggest that the exercise consultation may be a successful intervention for long-term maintenance of physical activity.[175;176]

The exercise consultation intervention was initially aimed at sedentary individuals, however recently strategies to prevent relapse were incorporated into the consultation procedure to enhance maintenance of physical activity. A pilot study[178] demonstrated that the exercise consultation significantly improved short-term exercise adherence during phase IV cardiac rehabilitation in a group of patients who had completed a phase III supervised exercise programme.

SUMMARY OF SECTION TWO

In summary, theoretical models of behaviour change have identified a number of factors that influence participation in physical activity in the general population and cardiac rehabilitation settings. Furthermore, interventions based on theoretical models such as the TTM, SCT and relapse prevention are effective in increasing and maintaining physical activity. Current traditional phase III rehabilitation programmes do not facilitate independent exercise after programme completion. However, the research is limited on effective interventions to encourage long-term maintenance of physical activity after completion of cardiac rehabilitation exercise programmes. Physical activity counselling and exercise consultation, based on the TTM, SCT and relapse prevention training, use evidence-based strategies to increase and maintain physical activity. In addition, these intervention are closely linked to the 1995 physical activity guidelines, thus encourage individuals to integrate moderate intensity activity into their daily lives, which may overcome the many barriers associated with

supervised exercise programmes. A pilot study found that the exercise consultation improved short–term maintenance of physical activity following completion of a phase III exercise programme.[178] However, the long-term effect of the exercise consultation on maintenance of physical activity after programme completion has not been studied.

SECTION THREE

MEASUREMENT OF PHYSICAL ACTIVITY

The primary aim of this study is to evaluate the effect of an exercise consultation on maintenance of physical activity following completion of a phase III cardiac rehabilitation exercise programme. Therefore, to detect changes in this variable it is important to accurately measure physical activity. This section reviews the methods that have been commonly used to measure physical activity. This review was then used to select the most appropriate methods to measure these outcomes based on the requirements of the study.

Components of Physical Activity

Physical activity is a complex, multidimensional behaviour, which is difficult to measure. Physical activity has been defined as "any bodily movement produced skeletal muscles that results in energy expenditure."[182] Therefore, physical activity can be quantified by measuring energy expenditure, either directly (e.g. using doubly labelled water) or indirectly (e.g. measuring bodily movement using motion sensors). Physical activity has three dimensions; frequency, intensity (light, moderate or vigorous) and duration, which are associated with health and fitness.[7] Physical activity can be performed in a number of different settings including leisure-time, housework, and occupation.[182] Although exercise is often used interchangeably with physical activity, it is actually a subcategory of physical activity. Exercise is physical activity, which is planned, structured and undertaken to improve or maintain one or more components of physical fitness. Physical fitness has been defined as a set of attributes (e.g. cardiorespiratory fitness, muscle strength and flexibility) that people have or achieve that relates to the ability to perform physical activity. Although, more active people tend to be fitter, physical fitness and the ability to improve fitness with training have a sizeable hereditary component and are affected by gender, ageing and habits other than physical activity.[183] Therefore, measuring fitness to quantify an individual's physical activity has its limitations. Overall, physical activity involves various components, most or all of which may need to be recorded to accurately quantify an individual's physical activity behaviour. Accurate measurement of physical activity is required to determine the proportion of a study population meeting current physical

activity recommendations[6;8] and to examine the effectiveness of physical activity interventions.

Methods to Measure Physical Activity

There are numerous methods available to measure physical activity, making it difficult for researchers to select the most appropriate method(s). Physical activity measures can be divided into subjective (e.g. recall questionnaires, and activity diaries) and objective methods (e.g., doubly labelled water, indirect calorimetry, pedometers, accelerometers and heart rate monitoring). The method selected to measure physical activity in intervention studies needs to be valid, reliable, and responsive to change, without influencing an individual's behaviour.

Validity is the extent to which a method measures what it is designed to measure.[184] Testing the validity of a method involves comparing it with another physical activity measure, known as the criterion method. In addition, the method has to demonstrate good reliability.[184] A reliable method consistently provides the same results under the same conditions. Reliability of a method can be tested by repeatedly measuring a physical activity bout on the same subjects, this is known as test-retest reliability. High reliability is important in intervention studies to ensure that changes in physical activity are caused by the intervention, and not because of variation in activity scores due to low reliability.[185] Finally, to obtain a good estimate of an individual's physical activity habits, a minimum of 5 or 6 days of recordings are required.[186] This is because physical activity varies from day to day, and measuring physical activity over several days reduces this variability. Furthermore, if the study population includes subjects that are working, then both week and weekend days must be sampled.[186]

A number of methods can be used to measure physical activity, which are discussed below.

Indirect Calorimetry

Indirect calorimetry estimates energy expenditure by directly measuring oxygen uptake (VO_2) and carbon dioxide production (VCO_2).[183] This method can be conducted in two ways; using a whole room calorimeter or a metabolic cart. Whole room indirect calorimeter is an airtight room, which is usually equipped with a desk, chair, toilet, sink, TV, a bed and a stationary bike etc. Oxygen uptake and carbon dioxide production are

measured from expired gas and used to calculate energy expenditure. A whole room calorimetry accurately measures energy expenditure,[187] however these rooms are expensive to build and operate, they are confining, and some types of activity cannot be performed, such as gardening or sports. Thus, whole room calorimeters cannot be used to measure physical activity habits under free living conditions.

Energy expenditure can also be estimated by measuring oxygen uptake and carbon dioxide production in expired air using a metabolic cart.[183] With this procedure, the subject wears a face mask or nose clip and mouthpiece. Stationary metabolic carts are used in the laboratory to estimate energy expenditure at rest, during treadmill or cycle exercise, and other laboratory-based activities. Portable metabolic carts can be used to estimate the energy cost of field-based activities. However, portable systems cannot be used to measure habitual physical activity in free-living conditions due to the limitations of the equipment.

Doubly Labelled Water (DLW)

Doubly labelled water (DLW) can be used to measure total energy expenditure (TEE) for 1 to 3 weeks under free living conditions.[188] DLW consists of the stable isotopes of water $^{2}H_2O$ and $H_2{}^{18}O$, which is ingested by the subject as a liquid. Elimination of these isotopes in water (e.g. urine, sweat or saliva) and CO_2 is analysed using mass spectrometry over several days, and energy expenditure and oxygen uptake can be estimated from CO_2 production using established equations. Physical activity related energy expenditure is calculated by subtracting the resting metabolic rate and the thermic effect of food from total energy expenditure. The DLW method accurately measures energy expenditure, therefore it is considered to be the "gold standard" for measuring energy expenditure under free-living conditions.[188] However, this method has limited applicability for measurement of physical activity in intervention studies and epidemiology based trials of large populations because the stable isotope of water is expensive, and the analysis of samples using the mass spectrometer is costly and complex. Moreover, as DLW measures energy expenditure, it does not provide information on physical activity patterns (i.e. intensity, duration or frequency). However, DLW has been used to validate other instruments because it accurately measures energy expenditure.

Overall, indirect calorimetry and doubly labelled water are frequently used as criterion

methods to validate other physical activity measures, such as motion sensors, questionnaires and heart rate monitors.

Motion Sensors

Motion sensors, such as pedometers and accelerometers are devices that measure physical activity by detecting motion or acceleration of a limb or trunk, depending on the placement of the monitor on the body.[189]

Accelerometers

When a person moves, the limbs and body are accelerated, theoretically in proportion to the muscular forces responsible for the accelerations and thus to energy expenditure.[189] Portable accelerometers measure these accelerations caused by bodily movement in either one (uniaxial) or three (triaxial) directions. A number of accelerometers, varying in size, cost and weight are commercially available, such as the Caltrac, Computer Science and Applications and the Tritrac. Accelerometers record activity counts, which are the product of the frequency and intensity of movement, sampled at set intervals. They have a large memory capacity, thus are able to store minute by minute data for several weeks. In addition, the accelerometer recordings can be used to estimate oxygen uptake, energy expenditure, and the time spent engaging in light, moderate and vigorous physical activity using established regression equations.

Uniaxial accelerometers

Uniaxial accelerometers detect bodily accelerations in the vertical (z) direction, and have been widely used in research to assess physical activity.[190] The Computer Science and Applications (CSA) is a uniaxial accelerometer, which has been frequently used in physical activity studies due to its small size, lightweight, ease of use, durability, large memory capacity and can be worn on the ankle, wrist or waist. Studies have found that the CSA accelerometer is very reliable for measuring physical activity. Melanson assessed the test-retest reliability (r=0.93 to 0.99) of the CSA accelerometer worn on the waist, ankle and wrist by collecting CSA recordings during treadmill walking and jogging on two separate occasions in a group of healthy subjects. High correlation coefficients, ranging from 0.93 to 0.99 between CSA recordings collected during repeat testing were reported in this study. In addition, no significant differences were observed between CSA activity counts recorded during repeat testing of field-

based activities.[191] Fairweather tested the inter-instrument reliability of the CSA accelerometer by attaching CSAs to a mechanical accelerometry system, which simulated body movement. A minimal difference (3%) was observed between CSA accelerometers attached to this system.[192]

The validity of the CSA accelerometers to measure physical activity and estimate energy expenditure has been studied under laboratory conditions in adults and children.[193-195] Melanson[193] assessed the validity of the CSA accelerometer worn at all three attachment sites against direct measurement of VO_2 during treadmill walking and jogging in a group of young adults. The study found a significant increase in activity counts from the monitors attached to all sites with increasing treadmill speed. Furthermore, significant linear correlations ($r > 0.80$) were observed between activity counts and measured VO_2 and energy expenditure during walking and jogging. In contrast, there was no significant relationship between activity counts and measured VO_2 or energy expenditure for changes in the gradient of the treadmill at a constant speed. Therefore, the CSA accelerometer detected changes in treadmill speed but not gradient. Other studies have demonstrated that the CSA accelerometer is a valid instrument for estimating energy expenditure during treadmill walking and jogging under controlled laboratory conditions.[190;193-195] Freedson developed a regression equation to estimate energy expenditure from activity counts recorded during treadmill walking and jogging in a group of young adults.[195] The study found no significant difference between estimated and measured energy expenditure during treadmill exercise. Similarly, Trost found that the CSA accurately estimated energy expenditure during treadmill exercise in a group of children.[194]

The validity of the CSA to measure physical activity and estimate energy expenditure has also been examined in field settings.[191;196-199] Hendleman[191] reported a significant correlation ($r = 0.77$) between accelerometer recordings and oxygen uptake measured by indirect calorimetry during overground walking at a range of self-paced speeds. These accelerometer recordings measured during overground walking were used to develop individual regression equations to estimate oxygen uptake. These equations were used to estimate VO_2 from activity counts recorded during golf, washing windows, vacuuming, lawn mowing, dusting and planting shrubs. The VO_2 estimated from activity counts was significantly lower than measured VO_2 for all activities. The difference between measured and predicted VO_2 ranged from 30.5% for golf to 56.4%

for planting shrubs. Other studies have found that the CSA significantly overestimated the energy cost (METs) of walking[196] and significantly underestimated the metabolic cost of leisure, recreational and household activities.[191;196;197]

The CSA has been compared with DLW to measure total and physical activity related energy expenditure.[198;199] Leenders[198] found that total daily energy expenditure estimated from CSA recordings was significantly lower by 59%, compared to energy expenditure measured using the DLW method in a group of 13 healthy women. Furthermore, a modest correlation (r = 0.45) between measured energy expenditure and activity counts was reported. In contrast, Ekelund[199] documented that the CSA accurately estimated total and physical activity related energy expenditure in a group of children. However, a significant proportion (40% to 55%) of the variation in energy expenditure was not explained by activity counts.[199]

In summary, strong linear correlations have been observed between activity counts collected at the three attachment sites and measured VO_2 and energy expenditure during level walking and jogging on a treadmill and overground walking.[193-195] However, poor to modest correlations have been observed between activity counts and measured VO_2 and energy expenditure during uphill walking on a treadmill and field-based activities.[191;196] Furthermore, the CSA accelerometers substantially underestimated the energy cost of household, gardening and leisure activities and energy expenditure under free-living conditions.[191;196-198]

Triaxial accelerometers

Human movement is multi-directional, thus measuring accelerations in three planes may provide a more accurate assessment of physical activity and energy expenditure compared to measuring movement in one direction.[189] The Tritrac is the most commonly used triaxial accelerometer and measures accelerations in the anterior-posterior (x), medio-lateral (y), and vertical (z) direction. The test-retest reliability of the Tritrac has been demonstrated at rest,[200] during treadmill exercise,[200-203] stepping,[202] and stationary cycling.[202] However, a study assessing the inter-instrument reliability of the Tritrac, found a 22% difference in activity counts between two monitors during treadmill walking.[204]

Similar to the CSA, the Tritrac is able to detect changes in treadmill speed,[201;205] but

not gradient.[201;206] Studies assessing the validity of the Tritrac to estimate energy expenditure during laboratory-based activities have reported inconsistent findings.[201;202;205;206] Sherman[207] found that compared to measured energy expenditure, the Tritrac accurately estimated energy expenditure at rest and during treadmill exercise at 3 different speeds at 0% gradient in a group of young adults. In contrast, other researchers observed that the Tritrac significantly overestimated energy expenditure during treadmill walking at 0% gradient,[201;202] and significantly underestimated energy expenditure during treadmill walking at an incline, stepping, stationary cycling, and slideboarding.[201;202;206] These conflicting findings indicate that estimating energy expenditure with the Tritrac may depend upon the activity performed, or the placement of the Tritrac; on the hip[201;202] compared to the lower back.[205]

Chen[187] evaluated the validity of the Tritrac to estimate energy expenditure using a whole room calorimeter, thus simulating conditions that were close to free living. Compared to measured energy expenditure, the Tritrac significantly underestimated total and physical activity energy expenditure. The Tritrac manufacturer's prediction equation was used to estimate energy expenditure from accelerometer recordings. However, Chen discovered that the estimations of total and physical activity energy expenditure were significantly improved when their own prediction equation was used. The investigators concluded that the inaccurate estimates of energy expenditure from the Tritrac were due to the equation used to predict energy expenditure.

The validity of the Tritrac to measure physical activity and estimate energy expenditure has been tested in under free-living conditions.[191;198] Compared to DLW, the Tritrac significantly underestimate energy expenditure by 35%.[198] Furthermore, only a modest correlation was found between measured energy expenditure and Tritrac recordings ($r = 0.54$). Similarly, Hendelman[191] reported that the Tritrac significantly underestimated the energy cost of various household and leisure activities. The difference between measured and predicted METs ranged from 32.1% for golf to 53.1% for planting shrubs.

Limitations of accelerometers

There are limitations of accelerometers for measuring physical activity. Firstly, these devices are designed to measure acceleration during bodily movement, thus static

work such as lifting objects cannot be accurately quantified by accelerometers. In addition, activities that increase energy expenditure without a proportional increase in bodily acceleration such as walking uphill will not be detected by accelerometers.[193] These instruments cannot be worn in water, thus will not be able to record swimming or other water activities. Additionally, accelerometers may not record upper body movement, unless they are worn on the wrist. Overall, as accelerometers cannot detect all types of physical activity, they will be unable to accurately estimate energy expenditure in free-living conditions. Additionally, the inaccurate estimations of oxygen uptake and energy expenditure from accelerometers may be due to the equations used to predict these parameters from accelerometer recordings. The equations were developed from accelerometer readings and oxygen uptake recorded during walking and jogging under controlled laboratory conditions. However, the relationship between activity counts and oxygen uptake under laboratory conditions differs from the relationship in field settings. For example, a lower correlation between oxygen uptake and activity counts was observed for overground walking compared to treadmill walking.[191] Furthermore, the relationship between accelerometer counts and oxygen uptake or energy expenditure may be specific to a given type of activity. For example, a stronger relationship between activity counts and VO_2 was observed for walking[193-195] compared to other activities (e.g. gardening and housework).[191;196;198] In addition, Hendelman[191] found that the CSA and Tritrac did not accurately estimate the energy cost of various household, gardening and leisure activities using regression equations developed during overground walking. Thus, researchers have recommended using the raw output from accelerometers (i.e. activity counts) to quantify physical activity rather than converting the accelerometer readings to energy expenditure.

Comparison of the CSA and the Tritrac

It has been proposed that measurement of bodily movement in 3 directions is better than measuring acceleration in one direction. Therefore, studies have compared triaxial and uniaxial accelerometers to determine whether one instrument is superior for measuring physical activity.[191;198;203;208] A slight improvement in the estimation of VO_2 and energy expenditure was observed with the Tritrac compared to the CSA, although both monitors still significantly underestimated energy expenditure.[198;208] In contrast, Welk[203] found that the CSA accurately estimated METs during treadmill walking at 3 different speeds on the level, whereas the Tritrac did not. Finally, another study found that the Tritrac and the CSA underestimated the energy cost of various

leisure and household tasks by a comparable amount.[191] Although there is some evidence to suggest that the Triaxial accelerometer may provide a better estimate of energy expenditure, these findings are inconsistent and both types of accelerometer do not accurately estimate VO_2 and energy expenditure under free-living conditions.[191;198;208] Furthermore, the CSA accelerometer has some advantages compared to the Tritrac for measuring physical activity under free-living conditions. The CSA is smaller, lighter, less expensive and more compact than the Tritrac, and can be worn on the waist, ankle or wrist, whereas the Tritac can be positioned on the waist or lower back. One study found that 20% of elderly subjects did not comply with wearing the Tritrac for at least 3 days.[200] Therefore, the CSA may allow for less obtrusive data collection and better compliance.

Pedometers

Pedometers are small, inexpensive and are designed to count steps during locomotion.[189] They contain a horizontal, spring-suspended lever arm that moves up and down with vertical acceleration of the body during locomotion. With each step, the lever arm makes mechanical contact and one step is recorded. The output from the pedometer is in total steps. Although, some pedometers can estimate total distance walked, if stride length is provided, or they can estimate energy expenditure.[189]

Bassett[209] reported that the Yamax Digi-walker was very accurate for counting steps and estimating total distance during self-paced overground walking. Hendelman[191] also tested the utility of the Digi-walker to assess moderate intensity overground walking. Pedometer recordings were highly correlated with actual steps counted ($r=0.84$), walking speed ($r=0.86$) and VO_2 ($r = 0.75$). However, pedometers underestimate step frequency at slow walking speeds, as the vertical acceleration of the body during slow walking may be too low to be captured by the instrument.[191;209] In addition, pedometers underestimate the total distance walked at fast walking speeds, despite accurately counting step frequency.[209] This is because stride length tends to increase at fast walking speeds. Therefore, estimating total distance walked from pedometry recordings introduces measurement error. In addition, pedometers cannot differentiate between walking and running, therefore do not reflect the intensity of movement.[189] Furthermore, pedometers do not accurately assess energy expenditure under free-living conditions.[196-198] Leenders[198] found that the Digi-walker significantly underestimated physical activity energy expenditure by 59% when compared to energy

expenditure assessed by DLW. Additionally, other studies reported that pedometers significantly overestimated the energy cost of walking[196] and significantly underestimated the energy cost of various moderate intensity activities.[196;197]

Montoye[189] stated that "pedometers were designed to count steps in locomotion and should not be expected to measure other kinds of activities or estimate energy expenditure." In agreement, the literature suggests that pedometers may be useful for measuring walking behaviour by counting steps. However, they can be inaccurate at very slow walking speeds, may not accurately measure total distance walked at very fast walking speeds, and provide inaccurate estimates of energy expenditure under free-living conditions. These limitations of the pedometer make it less suitable for measuring habitual physical activity, unless walking is the predominant form of activity.

Heart Rate Monitoring

There is a linear relationship between heart rate and oxygen uptake during exercise under laboratory conditions.[210] Therefore, monitoring heart rate has been used to assess physical activity by estimating VO_2 and energy expenditure. Advantages of heart rate monitoring include their ability to measure the frequency, intensity and duration of physical activity. In addition, they are relatively inexpensive, simple to use, robust, and versatile in a wide variety of field settings, non-intrusive and well tolerated by subjects.[210] However, there are limitations of assessing physical activity by monitoring heart rate. Factors such as emotional stress, high ambient temperature and high humidity will raise the heart rate with little effect on oxygen uptake.[211] In addition, arm exercise elicits a higher heart rate than leg exercise even though the oxygen cost is the same, because of the smaller muscle mass involved with arm only activity.[197] Individual variations in age, medication (e.g. beta-blockers), fitness level and gender can also affect the relationship between heart rate and oxygen uptake (HR-VO_2).[211]

These individual differences in the HR-VO_2 relationship resulting from variations in age, medication, gender and fitness can be overcome by establishing a HR-VO_2 calibration curve for each individual.[211] This involves simultaneously recording an individual's heart rate and VO_2 responses to exercise at different intensities under laboratory conditions. A HR-VO_2 calibration curve is established for each individual, which is then used to estimate VO_2 and energy expenditure from heart rates recorded under free living conditions.[197] Using this method, Racette[212] found that heart rate

monitoring produced similar estimates for total and physical activity related energy expenditure compared to DLW. However, other investigators have reported that heart rate monitoring significantly overestimated the oxygen cost of various field-based activities.[197;213]

The relationship between heart rate and VO_2 is poor at low levels of exercise,[204] thus, the HR FLEX method was developed to overcome this limitation.[214;215] This technique involves monitoring each subject simultaneously for heart rate and VO_2 at rest, during sedentary activity and exercise, resting metabolic rate is also obtained. This information is then used to develop the individual's HR-VO_2 calibration curve. The FLEX HR is determined by taking the highest heart rate recorded from rest and sedentary activity and the lowest heart rate recorded during light exercise. Above the FLEX HR value, there is a strong relationship between HR and VO_2, below it they are poorly correlated. During heart rate monitoring of physical activity in the field, if a given heart rate is below the FLEX HR then energy expenditure is determined from resting metabolic rate. Whereas, if a given heart rate is above the FLEX HR then energy expenditure is estimated using the calibration curve.[216] Studies have reported no significant difference between measured and estimated energy expenditure using the FLEX HR method in a group of children[215] and adults.[214] However, there are some drawbacks of the FLEX HR method to estimate energy expenditure. Firstly, the calibration test and the data analysis is time consuming and costly. Secondly, the HR-VO_2 relationship derived from activities in a controlled laboratory setting may not accurately represent the relationship in free-living activity.[211] Finally, heart rate can be affected by factors other than physical activity, as previously discussed.

Simultaneous use of heart rate monitoring and accelerometers

Heart rate monitoring and accelerometers have limitations for measuring physical activity and estimating energy expenditure. However, Strath[197] found that the simultaneous use of heart rate and motion sensors improved the estimation of VO_2 of selected field-based activities, compared with heart rate monitoring and accelerometers independently. This study established an individual calibration curve between HR and VO_2 for both arm and leg exercise in the laboratory. In the field setting, the activity monitor was used to differentiate between arm and leg movement and heart rate was used to estimate VO_2 from the corresponding calibration curve. This differentiation allowed the investigator to predict VO_2 from an individualised arm

or leg HR-VO_2 calibration curve, as the relationship between HR and VO_2 differs for arm and leg exercise.[211] Another advantage of this technique is that the accelerometers can differentiate between an increase in heart rate caused by physical activity and that caused by other factors. The simultaneous use of heart rate and accelerometers to measure physical activity could be a promising method to measure physical activity, however this method is very time-consuming.

In summary, the objective methods that could be used to measure physical activity in this study were reviewed. Although motion sensors and heart rate monitors have limitations, an objective method was selected because they eliminate the subjectivity associated with self-report methods. An accelerometer seemed to be the most appropriate instrument to use in this study for the following reasons. Firstly, accelerometers have a large memory capacity, which is important because at least 5 days of physical activity should be recorded to obtain a good estimate of an individual's physical activity behaviour.[186] Heart rate monitoring was not selected, as it requires the development of individual calibration curves, which is very time-consuming, whereas analysing accelerometer recordings is relatively quick and simple. Additionally, accelerometers are reliable, which is important in an intervention study to ensure that changes in physical activity are a result of the intervention, and not because of variation in activity scores due to low reliability. As previously discussed, accelerometers cannot record all types of activity under free-living conditions, however there were limitations to all other objective methods that could have been used in this study. The CSA accelerometer was chosen instead of the Tritrac because it is smaller, lighter, and more compact than the Tritrac, which may allow for less obtrusive data collection and better compliance. One study reported that 20% of the study sample did not comply with wearing the Tritrac for at least 3 days.[39] In addition, the CSA is less expensive than the Tritrac. Finally, the CSA is similar to the Tritrac for measuring physical activity. Schutz recommended using multiple methods to measure physical activity as no single current technique is able to quantify all aspects of physical activity under free living conditions.[217] Thus, a self-report method was selected to gain additional information on patterns of physical activity.

Self Report

Numerous self-report instruments have been developed to assess physical activity. Self-reports include self-administered or interviewer-administered recall

questionnaires, activity logs or diaries. They are practical for use in field studies as they are relatively inexpensive and require less equipment compared to objective measures.[218] Furthermore, some self-reports measure the dimensions of physical activity (type, frequency, intensity, and duration), therefore an individual's pattern of physical activity can be examined. This information can also be used to determine if individuals are meeting current physical activity recommendations.[219] However, there are limitations to self-report methods. They are subjective, therefore can be influenced by the subject's perceptions and emotions. In addition, subjects may not recall their activities accurately, and social desirability bias can lead to over-estimation of the duration or intensity of activities. Recall questionnaires have an advantage over activity diaries and activity logs, as they are less time consuming and do not affect physical activity behaviour (unless subjects know in advance that they will be completing one).

Recall Questionnaires

Recently, Sallis reviewed the validity, reliability and content of numerous self-report questionnaires that have been frequently used over the past decade to measure physical activity in adults.[219] The components of physical activity measured by the questionnaires varied widely. The majority of self-reports measured the dimensions of physical activity (i.e. duration, frequency, and intensity); some assessed both moderate and vigorous intensity activities. Leisure physical activity was included in all self-reports, whereas, assessment of household and occupational activity was less common. A few questionnaires measured long-term habitual physical activity over the previous year, whereas others assessed recent or usual activity. All of the self-report questionnaires reviewed demonstrated modest to high levels of test-retest reliability.[219] However, the test-retest correlation coefficients tended to be higher for total and vigorous physical activity compared to moderate physical activity. The validity of the questionnaires for measuring physical activity was evaluated by comparing them with motion sensors and heart rate monitoring. In general, low to moderate correlation coefficients were observed between self-report measures and objective methods, ranging from 0.14 to 0.50. However, motion sensors and heart rate monitoring have limitations for measuring physical activity, therefore they are inadequate for use as a criterion method. Doubly labelled water accurately measures total and physical activity related energy expenditure, thus has been used to validate questionnaires. However, DLW does not measure the intensity, frequency and duration of physical activity, therefore this method cannot be used to validate the various components of physical

activity measured by self-report questionnaires. In general, determining the validity of self-report questionnaires is difficult due to the lack of an acceptable criterion method.

In summary, there are numerous questionnaires that can be used to measure physical activity, each demonstrating varying levels of reliability and validity. Recall questionnaires are useful in assessing habitual physical activity, as they are inexpensive, easy to use and do not alter physical activity behaviour. In addition, unlike accelerometers, they can provide information on mode, intensity, duration and frequency of physical activity and whether an individual is meeting current physical activity recommendations. For these reasons, it was appropriate to use a recall questionnaire to assess physical activity in this study. The seven-day physical activity recall[220] was selected, as it measures all dimensions of physical activity (i.e. type, frequency, duration and intensity), and measures physical activity during both occupation and leisure time. In addition, the questionnaire measures recent, instead of usual activity, which is important in an intervention study to assess changes in physical activity. Physical activity patterns vary from day to day and from weekday to weekend, therefore the questionnaire assesses physical activity behaviour for seven days. Finally, the instrument is administered in an interview format, which may generate more accurate and reliable information than self-administered questionnaires.[221] This is because some subjects find it difficult to recall physical activity or classify the intensity level of various activities. Furthermore, individuals may not always define what they are doing as physical activity, for example housework. Therefore, the interviewer can advise the subject and use prompts to facilitate recall.[220]

Stanford Seven-Day Physical Activity Recall

The Stanford Seven-Day Physical Activity Recall (7DPAR)[220] has been widely used in research to quantify physical activity. This instrument is an interview administered questionnaire that measures the time spent in sleep and light, moderate, hard and very hard intensity physical activity for seven days. An estimate of energy expenditure can be calculated from the time spent in various intensities of physical activity, using established MET values for each intensity category. The reliability and validity of the seven-day recall has been reviewed by various investigators.[218;219;222;223] The seven-day recall demonstrated high test-retest reliability (r=0.7 to 0.9) for estimating energy expenditure when the questionnaire was administered over a two-week period.[223-225] Other studies have observed modest test-retest correlation coefficients for the recall of

moderate, hard, very hard and total physical activity.[219;222;226-228] However, the time interval between questionnaire administrations ranged from 2 weeks to 1 month, therefore the reliability estimates reflect both the ability of the individual to recall their activity and actual changes in physical activity. Studies have reported poorer test-retest reliability for the recall of moderate intensity physical activity compared to recall of total, hard, very hard and occupational physical activity.[219;222;226;227] Vigorous activities tend to be planned or structured, thus individuals are more likely to accurately recall these types of activity. Whereas, it is more difficult to accurately recall and categorise the intensity of moderate activities that are part of normal daily life.[229]

The validity of Stanford Seven-Day Recall (7DPAR) to measure physical activity and estimate energy expenditure has been tested. Studies have found that energy expenditure estimated by the 7DPAR was similar to energy expenditure measured by DLW in a group of obese[212] and normal weight women.[198] In contrast, other studies have found that energy expenditure estimated by the 7DPAR was significantly overestimated by 11%[230] to 30%[231;232] compared to DLW. Leenders reported high correlation coefficients (r=0.82 - 0.94) between energy expenditure estimated from the 7DPAR and accelerometers recordings (CSA and Tritrac).[233] Furthermore, the questionnaire and accelerometers similarly estimated the time spent in light, moderate, hard, very hard and total physical activity. In contrast, Duncan found that sedentary, middle aged subjects significantly overestimated the time spent in moderate intensity activity measured by the questionnaire compared with actual moderate activity determined by heart rate monitoring.[223] Other studies have observed lower correlations for moderate intensity activity compared to hard, very hard intensity and total activity between the 7DPAR and accelerometers.[219;222;227;228;234] The 7DPAR has been used to assess physical activity levels of individuals participating in exercise-based cardiac rehabilitation. These studies found a significant increase in physical activity measured by the questionnaire after cardiac rehabilitation.[89;93;96] In addition, these changes were confirmed by a concomitant increase in exercise capacity.[89;93] In addition, physical activity intervention trials in the general population have found significant increases in physical activity measured by the questionnaire and accelerometers. Thus, the seven-day physical activity recall seems to be responsive to the effect of interventions to increase physical activity.

Cardiorespiratory Fitness

Cardiorespiratory fitness and the ability to improve fitness with exercise training has a sizeable hereditary component and can be affected by factors other than physical activity. Therefore, using fitness as an indicator of an individual's physical activity behaviour has its limitations. On the other hand, the improvement in aerobic capacity and submaximal endurance capacity with cardiac rehabilitation exercise training has been well documented. Furthermore, studies have shown that both physical activity and exercise capacity tend to decline in the long-term following completion of exercise-based cardiac rehabilitation. The aim of this study was to investigate the effect of an exercise consultation on maintenance of physical activity following completion of phase III exercise training, thus it was appropriate to assess cardiorespiratory fitness.

Exercise Capacity

Maximal oxygen uptake (VO_2max) is the criterion measure of exercise capacity.[83;235] VO_2max is the highest oxygen uptake attainable during a progressively increasing exercise test, as demonstrated by a plateau in oxygen uptake despite further increases in work rate. VO_2 max is equal to maximal cardiac output and maximal arteriovenous oxygen difference, thus represents the capacity of the cardiovascular system to supply oxygen to exercising skeletal muscles and the potential for oxygen extraction by the exercising muscle. However, most cardiac patients fail to achieve a "true" VO_2max as they are often limited by cardiac symptoms and may be reluctant to push themselves to their maximal capacity.[236] Thus, peak VO_2, the rate of oxygen uptake at the highest level of exercise, is more relevant when discussing the exercise capacity of individuals with CHD.

Accurate assessment of exercise capacity (i.e. peak VO_2) is achieved with direct analysis of expired gases during a progressively increasing exercise test. The type of exercise testing protocol used influences the measurement of peak oxygen uptake.[237] In clinical practice, the Bruce protocol (or modified Bruce) is the most commonly used exercise testing treadmill protocol for cardiac patients.[83;236;237] However, this protocol has been criticised because of its large and unequal increases in work rate every three minutes. It is thought that some patients will be unable to tolerate these large and unequal increases in exercise intensity, thus will terminate the exercise test prematurely.[83;236;237] In addition, large and unequal increments in work rate leads to difficulties in interpreting gas-exchange

measurements (e.g. lactate threshold) and causes inaccurate estimates of exercise capacity.[83] Protocols with smaller, more frequent uniform work rate increments are recommended, which are preferred and better tolerated by patients and produce an accurate assessment of peak VO_2.[83;236;237] The exercise testing protocol should also be individualised, as cardiac patients exhibit a wide range of exercise capacities.[237] For example, exercise testing protocols with small work rate increments to accommodate patients with poor exercise capacity result in very long tests for fitter patients, which may produce unrepresentative values for peak VO_2 as subjects may terminate the test prematurely due to boredom. The optimal exercise test duration is 8 to 12 minutes, thus the work load increments should be adjusted for each patient's capabilities to achieve this test duration.[83;236;237]

Submaximal Endurance Capacity

Estimating the lactate threshold, the exercise VO_2 at which the concentration of lactate in arterial blood increases above the resting value,[83] during a progressive exercise test can be used as an indicator of submaximal endurance capacity. A range of techniques have been used to estimate the lactate threshold, including both direct measurements and indirect estimation. Direct measurement of lactate requires serial blood sampling during exercise testing, which is invasive and uncomfortable for patients.[83] However, the lactate threshold can be estimated noninvasively as the increase in lactate during exercise causes profound alterations in ventilation and gas exchange. The V-slope and ventilatory equivalent techniques are common and reliable methods for indirectly detecting the lactate threshold.[83]

The V-slope method for estimating the lactate threshold is illustrated in Figure 4. As work rate increases during an incremental exercise test, there is a linear increase in oxygen uptake (VO_2) and carbon dioxide output (VCO_2). Above the lactate threshold, the increase in lactic acid production causes carbon dioxide output to increase more rapidly than oxygen uptake. When these VO_2 and VCO_2 are plotted against each other, the relationship is composed of two linear components, the lower of which has a slope slightly less than 1.0, whereas the upper component has a slope steeper than 1.0. The intercept of these two slopes defines the oxygen uptake above which VCO_2 increases faster than VO_2 and signals the increased concentration of lactic acid. The increase in VCO_2 in excess of that derived from aerobic metabolism is generated from the buffering of lactic acid.

The ventilatory equivalent method is illustrated in Figure 5. Above the lactate threshold, minute ventilation (V_E) increases proportionally with the increased CO_2 output. Thus, V_E retains a constant relationship with VCO_2 (VE/VCO_2 appears constant or decreases slightly), whereas V_E increases relative to VO_2 (VE/VO_2 increases). Similarly, the end tidal pressure of oxygen ($PETO_2$) increases, whereas the end tidal pressure of carbon dioxide ($PETCO_2$) remains constant at the lactate threshold. This method is frequently used to verify estimation of the lactate threshold by the V-Slope method. Beaver found that the values obtained by the V-slope method agreed closely with those obtained by direct measurement of the lactate threshold.[238]

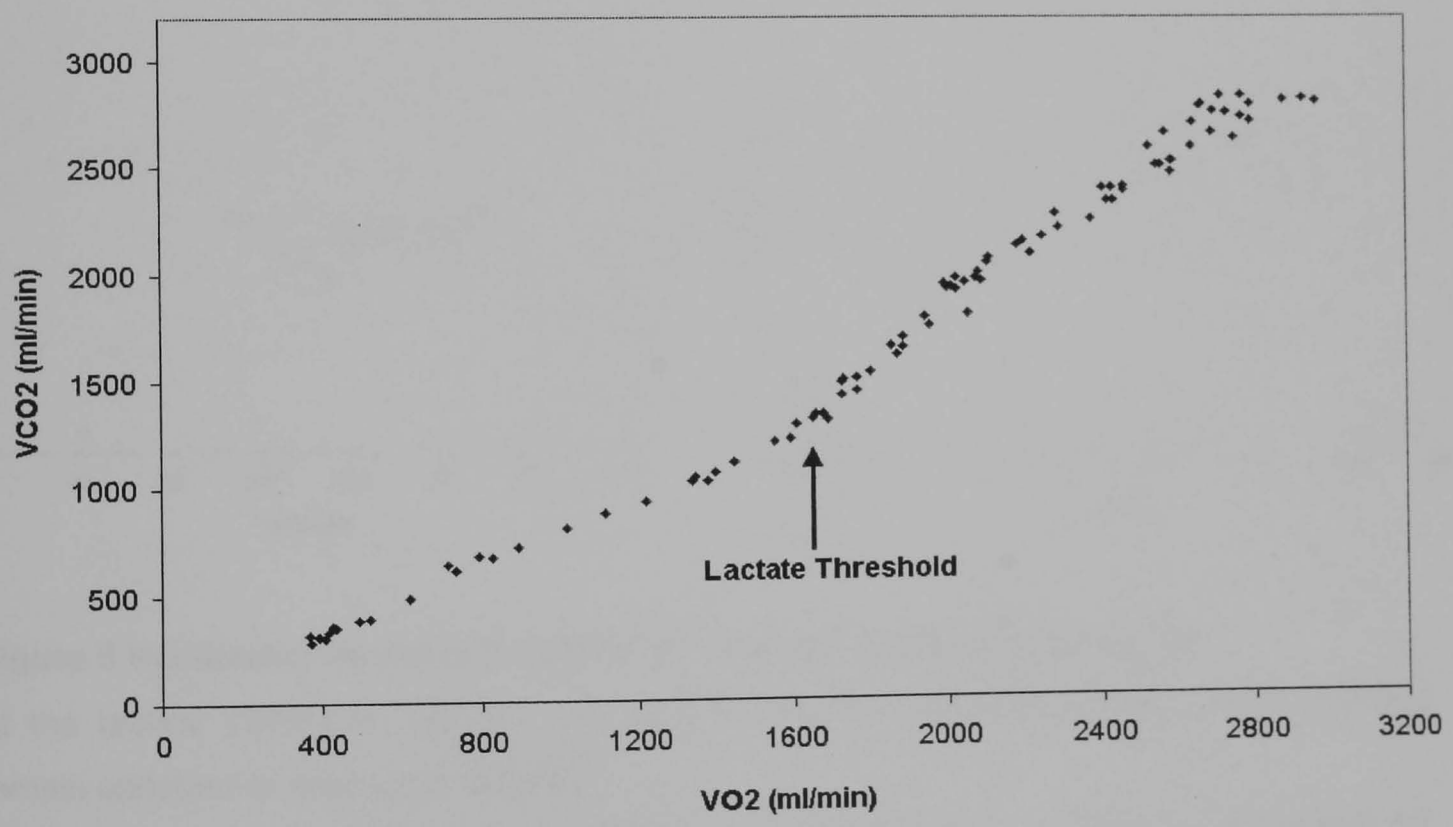
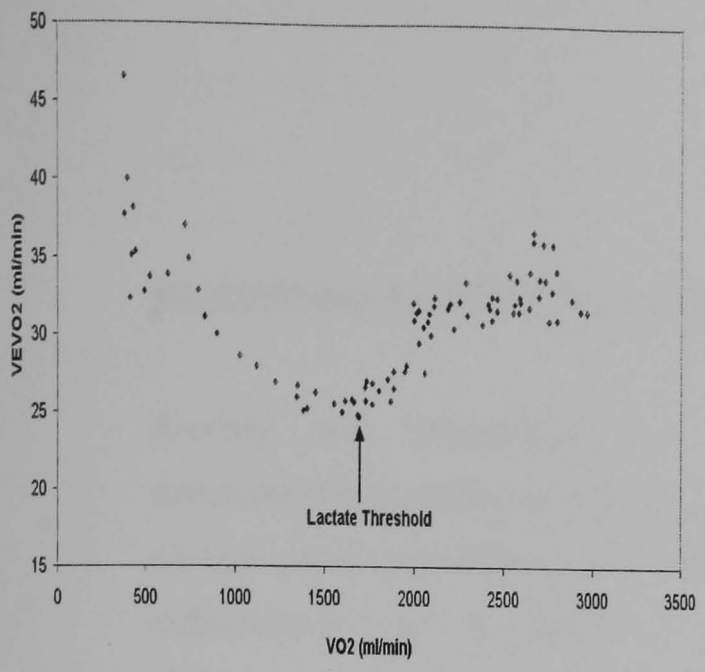
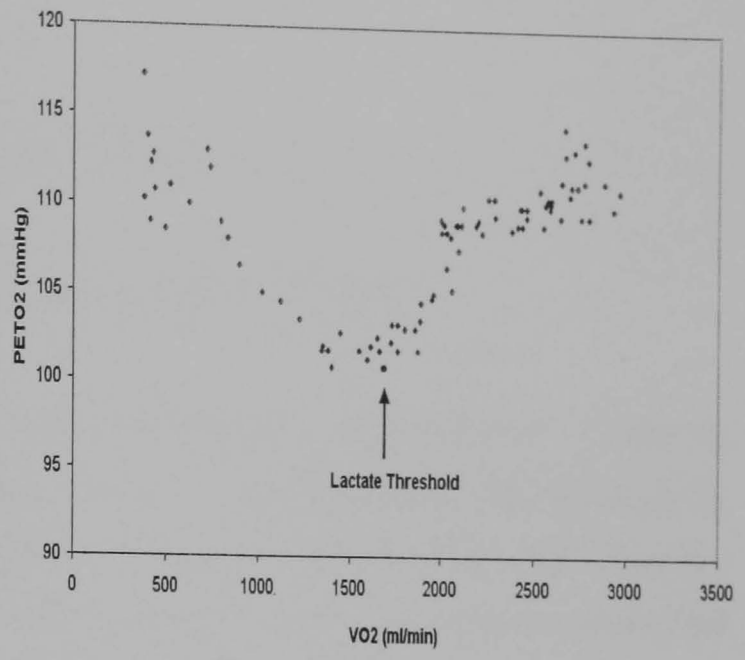
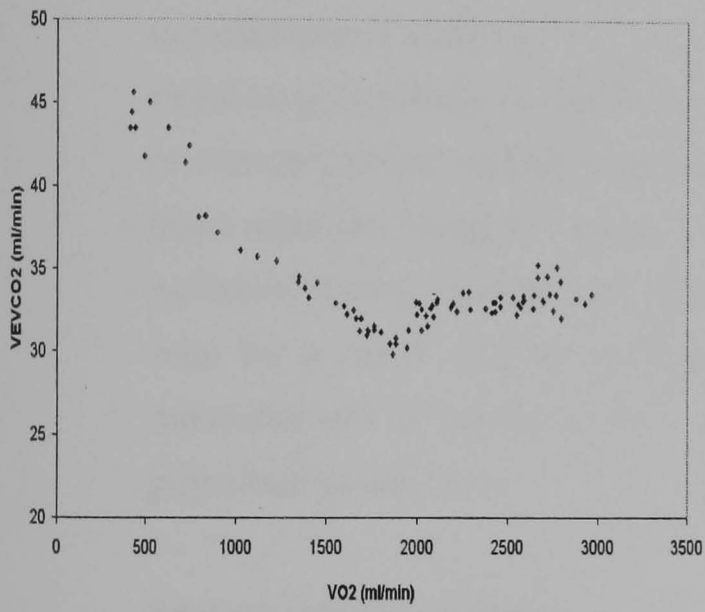


Figure 4 The V-Slope technique to estimate the lactate threshold.

Below the lactate threshold, there is a linear increase in VO_2 and VCO_2. Above the lactate threshold, VCO_2 increases more rapidly than VO_2 due to increased lactic acid. VO_2 = Oxygen uptake. VCO_2 = Carbon dioxide production.







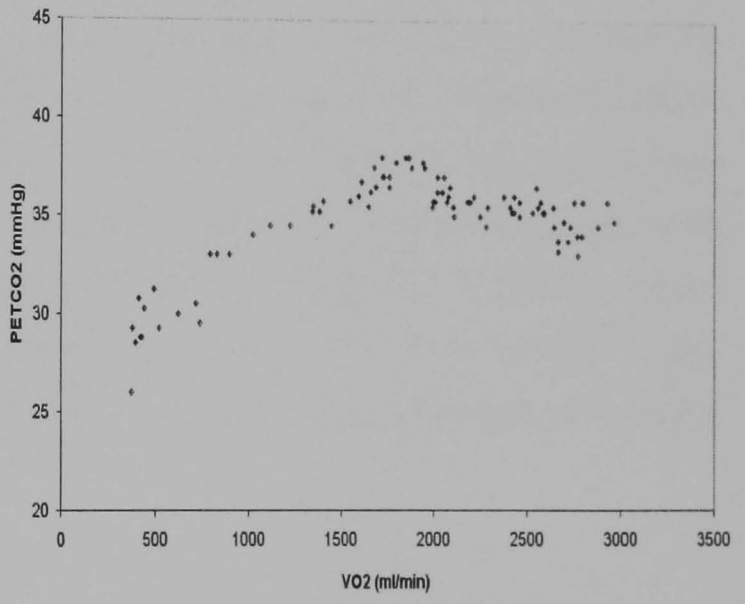


Figure 5 Ventilatory equivalent method to estimate the lactate threshold.

At the lactate threshold, VE/VO_2 and $PETO_2$ increase, whereas VE/VCO_2 and $PETCO_2$ remain constant or decreases slightly.

VE/VO_2 = ventilatory equivalent for oxygen uptake, VE/VCO_2 = ventilatory equivalent for carbon dioxide output. $PETO_2$ = End tidal O_2 partial pressure, $PETCO_2$ = End tidal VCO_2 partial pressure.

SECTION FOUR

MEASURING ANXIETY, DEPRESSION AND QUALITY OF LIFE

Anxiety and depression are commonly experienced psychological problems encountered by patients following cardiac events.[2] Evidence shows that moderate to severe depression following an MI is a significant predictor of cardiac mortality over the subsequent 6 to 18 months.[100;101] In addition, patients suffering from psychological distress have demonstrated poorer quality of life, increased severity of chest pain, poor compliance to lifestyle changes and increased use of health services compared to non-distressed patients.[102] Therefore, a goal of cardiac rehabilitation is to limit the psychological effects of cardiac illness and to enhance quality of life.[1] Beneficial effects of exercise-based cardiac rehabilitation on quality of life, anxiety and depression have been reported. However, some studies have found that improvements in quality of life achieved during cardiac rehabilitation diminish after programme completion, which may be a result of poor compliance to lifestyle changes in the long-term. Thus, measurement of quality of life, anxiety and depression were important secondary outcomes of this study.

Anxiety and Depression

National guidelines for cardiac rehabilitation[2] have recommended using the Hospital Anxiety and Depression Scale (HADS) to measure anxiety and depression. This instrument is easy to administer, simple to score and has been widely used in cardiac rehabilitation settings to screen patients for symptoms of psychological distress, and to evaluate psychological outcomes of cardiac rehabilitation.

The Hospital Anxiety and Depression Scale

The Hospital Anxiety and Depression Scale is a self-report instrument developed by Zigmond and Snaith (1983) to measure states of anxiety and depression in medical patients.[239] Symptoms relating to serious mental disorders were excluded from the instrument, which is thought to make the scale more sensitive to mild forms of psychological distress. Furthermore, the investigators omitted physical symptoms of psychological distress such as insomnia, loss of energy or weight loss, which may be a

direct consequence of a medical illness (e.g. MI) rather than related to depression or anxiety.[240] The instrument measures states of anxiety and depression over the past week, therefore it is sensitive to change but is not limited to an immediate stressful situation.[240]

The HADS is a reliable and valid instrument for measuring anxiety and depression in medical patients and patients with CHD.[241] Visser found that the anxiety and depression scales were very reliable over a two week period in a group of patients who had suffered an MI in the previous 6 to 24 months.[242] It is fundamental that the questionnaire items that compose a scale are related to one another, known as internal consistency. A Cronbach's alpha coefficient of 0.7 or above is taken as evidence of internal consistency. Johnston reported cronbach's alpha coefficients exceeding 0.80 for the HADS anxiety and depression scales at three time periods over 12 months following myocardial infarction.[243] The validity of a psychological instrument can be examined by comparing the questionnaire with relevant psychometric properties of other instruments (i.e. concurrent validity). A recent review observed moderate to high correlations ranging from 0.6 to 0.8 between the HADS anxiety and depression subscales and other self-assessment instruments.[244] The HADS is able to detect anxiety disorders and depression in medical populations compared to gold standard diagnostic instruments.[241;244] In addition, the prevalence of moderate anxiety and depression estimated using the HADS in a sample of the general adult UK population was similar to findings from epidemiology studies that employed well-validated classification systems.[245] Finally, it is expected that patients who have experienced a myocardial infarction will have higher levels of anxiety and depression compared to the general healthy population. Visser[242] reported higher scores on HADS anxiety and depression subscales for a group of post MI patients compared to healthy controls. Studies have shown that the HADS is responsive to the effects of cardiac rehabilitation for up to one year after the baseline assessment.[246;243;110;247]

Quality of Life

Evaluating the effect of cardiac rehabilitation on quality of life should include the assessment of physical function, psychological well-being and social functioning.[248] Instruments designed to measure quality of life can be divided into two categories: generic and disease-specific. Generic questionnaires provide a broad assessment of the health status of an individual and allow comparisons between groups of patients

with different conditions and general populations, however a main concern of these instruments is their lack of sensitivity to measure change over time.[248] In contrast, disease-specific instruments assess the effects of a particular disease on health status, which may improve the sensitivity of the measurement. However, a disadvantage of disease-specific instruments is that they may be less comprehensive in covering the multiple dimensions of health status. Due to the advantages and disadvantages of both types of questionnaire, ideally both disease-specific and generic instruments should be used to measure quality of life.[246] However, a number of methods were being used to measure the primary outcomes of this study, thus the addition of two quality of life questionnaires may have burdened the patients potentially affecting patient recruitment and compliance to the study. A generic questionnaire was selected to measure quality of life in this study for the reasons discussed below.

A variety of valid and reliable generic and disease-specific instruments have been used to measure quality of life in cardiac rehabilitation. In addition, a systematic review identified a number of instruments that were responsive to cardiac rehabilitation.[246] Thus, no agreement has been reached regarding the most appropriate questionnaire to measure quality of life in cardiac rehabilitation settings.[2] Recently, Hevey[247] examined the responsiveness of nine disease-specific and generic instruments, which are commonly used to assess quality of life in cardiac rehabilitation. The study sample was comprised of patients who had experienced a MI, CABG or PTCA. Results showed that most of the disease-specific questionnaires (i.e. Quality of Life after Myocardial Infarction, Quality of Life Index Cardiac Version III, and Cardiac Depression Scale) were not responsive to the effects cardiac rehabilitation. The exceptions were two subscales of the Heart Patient's Psychological Questionnaire: feelings of disability and despondency. However, the English version of this questionnaire has not been validated.[247] In contrast, a number of generic questionnaires were responsive to cardiac rehabilitation. These included the Global Mood Scale, the depression scale of the HADS and five out of eight subscales of the Short-Form 36.

Similarly, Smith[249] showed that the Short-Form 36 was more responsive to 6 weeks of cardiac rehabilitation than two disease-specific measures; the Quality of Life after Myocardial Infarction and the Quality of Life Cardiac Version III. There are other valid and reliable disease-specific instruments available, such as the Cardiac Quality of Life Inventory, and the Multidimensional Index to Life Quality, however their

responsiveness to cardiac rehabilitation has not been assessed. In contrast, the Seattle Angina Questionnaire is more responsive than the Short-Form 36,[250] however this disease-specific instrument does not provide a broad measure of quality of life. Another reason for choosing a generic questionnaire was the fact that many of the disease-specific questionnaires tend to focus on a specific cardiac condition (e.g. Quality of Life after MI, Angina Pectoris Quality of Life Questionnaire), and would not be appropriate for the heterogeneous population (i.e. MI, CABG and PTCA) included in this study. In addition, McGhee[246] suggested that if both a disease-specific and generic measure were shown to have equal responsiveness, then it may be preferential to use a generic measure as this allows comparison with other patient groups and normative data for the general population.

The most commonly used generic instruments to measure quality of life in coronary heart disease are the Nottingham Health Profile, the Short-Form 36 and the Sickness Impact Profile.[251] The Sickness Impact Profile was not feasible for use in this study because it takes patients approximately 20 minutes to complete the 136 questionnaire items.[242] Although, the Nottingham Health Profile has demonstrated acceptable reliability and validity in patients with heart disease,[252] major ceiling effects (maximum possible score) have been reported.[251;253] Dempster[251] found that 16% to 62% of MI patients achieved the maximum possible score on all NHP domains at the beginning of a cardiac rehabilitation programme. Thus, the Short-Form 36 (SF-36) was selected to measure quality of life in this study for the following reasons.

The Short-Form 36 (SF-36)

The SF-36 demonstrates good content validity, as it measures quality of life dimensions that are important in cardiac rehabilitation, for example physical function, social function and psychological wellbeing.[248] Secondly, the validity, internal consistency and test-retest reliability of the instrument has been established among patients with CHD. An acceptable level of internal consistency has been found for all SF-36 subscales in cardiac rehabilitation participants,[249;254] patients with stable coronary heart disease,[255] and patients who had suffered an MI in the previous 4 years.[253] In addition, Jette found that all SF-36 scales, except Role Emotional, were very reliable over a two week time interval in a group of cardiac rehabilitation participants.[254] High levels of agreement have been observed between SF-36 domains and relevant scales on the Nottingham Health Profile,[253] the Seattle Angina

Questionnaire, and the Chronic Heart Failure Questionnaire.[256] Dougherty reported a significant correlation between the physical limitations scale of the Seattle Angina Questionnaire and the physical function domain of the SF-36 in a sample of stable angina patients.[257] Furthermore, the disease perception scale of the Seattle Angina Questionnaire correlated significantly with both the energy and general health scales of the SF-36 in this group.

Studies have reported significantly poorer scores on all SF-36 subscales for post MI patients[258] and patients with stable coronary heart disease[256] compared to age and sex adjusted normative data for the general population. In addition, scores on all of the SF-36 scales were significantly poorer for cardiac patients who were experiencing symptoms of angina or breathlessness compared to those who were asymptomatic.[253;255] Among post MI patients, increasing frequency of chest pain was significantly associated with lower scores on all physical health domains, with no differences for role emotional or mental health. On the other hand, some studies have reported that all of the SF-36 subscales, except bodily pain[255] were unable to detect differences in the severity[255;257] and frequency[255] of angina in patients with stable coronary artery disease. However, similar findings were observed for two disease-specific instruments. In addition, floor and ceiling effects were reported for the Role Physical and Role Emotional SF-36 subscales among patients with stable coronary artery disease, post MI, and those at the beginning of cardiac rehabilitation.[253;255] Consequently, the SF-36 was revised to reduce the floor and ceiling effects of these subscales.[259] The authors reported that the Role Physical and Role Emotional subscales of the SF-36 version II demonstrated greater internal consistency and fewer floor and ceiling effects compared to the original questionnaire, which may improve the instrument's responsiveness.[259] Normative values for the SF-36 version II are available for a UK general population.[259]

CHAPTER TWO

AIMS

The benefits of exercise-based cardiac rehabilitation are well documented. However, evidence suggests that physical activity is not maintained in the long-term following completion of these programmes. Therefore, interventions are required to improve maintenance of physical activity after programme completion. Exercise consultation, based on the established behaviour change theories is effective in encouraging promotion and maintenance of physical activity in patients with Type II diabetes and the general population for 6 to 24 months. In a pilot study, the exercise consultation improved short-term maintenance of physical activity after completion of a phase III exercise programme.

Primary Aim

The primary aim of this randomised-controlled trial was to evaluate the effect of an exercise consultation (experimental condition) compared with standard exercise information (control condition) on maintenance of physical activity six and 12 months following completion of a phase III cardiac rehabilitation exercise programme.

Secondary Aim

The secondary aim was to determine the effect of an exercise consultation (experimental) compared with standard exercise information (control) on maintenance of cardiorespiratory fitness, the use of processes of exercise behaviour change, quality of life, anxiety and depression, blood lipids and clinical outcomes at six and 12 months in phase IV cardiac rehabilitation.

The Research Objectives are:

1) To describe the baseline demographic and clinical characteristics of the experimental and control group.

2) To describe the baseline physical activity behaviour of the study sample (n=70) using the CSA accelerometer,[190] the Stanford Seven-Day Physical Activity Recall[220] and the Stage of Exercise Behaviour Change.[260] As subjects had recently

completed a phase III exercise programme, it was hypothesised that they would be physically active at baseline.

3) To determine the effect of the exercise consultation (experimental) compared with standard exercise information (control) on the following:

   a) Maintenance of physical activity measured using the Stage of Exercise Behaviour Change,[260] the CSA accelerometer[190] and the Stanford Seven-Day Physical Activity Recall.[220]

   b) Maintenance of cardiorespiratory fitness,[83] including exercise capacity and submaximal endurance capacity.

   c) The use of the experiential and behavioural processes of exercise behaviour change.[160]

   d) Anthropometric variables including weight, body mass index and the proportion of patients who were overweight and obese.

   e) Blood lipids including total cholesterol, triglycerides, LDL cholesterol, HDL cholesterol and the ratio of total cholesterol to HDL cholesterol.

   f) Psychological functioning including anxiety and depression, and quality of life measured using the Hospital Anxiety and Depression Scale[240] and the Short-Form 36 version 2,[261] respectively.

4) To report the incidence of total and cardiovascular mortality, and the number of cardiac hospital readmissions (e.g. MI, UAP, CABG and PTCA) in the experimental and control group.

CHAPTER THREE

METHODS

Participants

Men and women who had experienced a myocardial infarction (MI), unstable angina (UAP) or had undergone coronary artery bypass grafting (CABG) or percutaneous transluminal coronary angioplasty (PTCA) and were attending a phase III hospital-based cardiac rehabilitation exercise programme were candidates for inclusion in the study. Patients were excluded if any of the following criteria were present: (1) on waiting list for cardiac investigation (i.e. angiography), (2) on waiting list for coronary artery bypass surgery (CABG) or percutaneous transluminal coronary angioplasty (PTCA), (3) attended less than 70% of the phase III exercise programme. Subjects were recruited from the phase III exercise programme from August 2000 to August 2001. Eligible subjects who were attending weeks 6 to 11 of the exercise programme were given information on the study. Subjects who agreed to participate in the trial received an appointment for a baseline assessment. Baseline assessments were carried out within one month of patients completing the phase III exercise programme. All assessments were conducted in the cardiology department at the Royal Alexandra Hospital. The study was approved by the Argyll and Clyde Research Ethics Committee (Appendix A) and all patients provided written informed consent before participation in the study (Appendix B).

Phase III Cardiac Rehabilitation Programme

The phase III cardiac rehabilitation programme was based at a district general hospital (Royal Alexandra Hospital) in the West of Scotland. The programme involved supervised exercise training, education sessions, medical evaluation, psychological support from specialist nurses and access to a clinical psychologist. The exercise programme involved two exercise classes each week for 11 weeks. Each class included a 15 minute warm-up with flexibility exercises, 20 minutes of circuit training, and a 10 minute cool down period with flexibility exercises. Participants were also encouraged to participate in physical activity at home on days they were not attending the exercise programme. Information booklets on physical activity and CHD were available in the gymnasium where the phase III classes took place.

Sample size

Physical activity measured using the CSA accelerometer[190] was used to estimate the number of participants needed to detect a true difference in this variable between the experimental and control groups. The following equation was used $n = 2(\alpha+ \beta)^2 s^2/d^2$, s = standard deviation and d = difference between the groups, and the values selected for alpha (α) and beta (β) were 5% and 10% respectively.[262]

No randomised controlled trials have evaluated the effect of exercise-based cardiac rehabilitation on physical activity measured objectively using accelerometers. However, a pilot study compared the effect of an exercise consultation with standard care on physical activity measured using CSA accelerometers in a group of people with type II diabetes.[263] Thus, the difference between the experimental and control group for the mean change in total activity counts/week measured with the CSA accelerometer was used to calculate the sample size (baseline mean = 1813893, mean difference between groups = 409780, SD = 444779). A minimum of 25 patients in each group was required to have a 90% power of detecting a true difference of 409780 counts/week (22%) with a significance level of 5%. However, 70 patients were recruited to allow for potential dropout from the study.

Baseline Assessment

Visit One: Total duration 60 minutes

Patients attended the first visit within one month of completing the phase III exercise programme. Written informed consent was obtained from participants. The following data were collected by self-report and reviewing medical records: demographics, socio-economic status[264] and occupation, medical history, medication and smoking status. Quality of life was measured using version 2 of the UK Short Form 36.[261] The Hospital Anxiety and Depression scale[240] was used to assess psychological distress. Weight (kg) and height (m) were recorded and used to determine body mass index (BMI). An incremental exercise test with expired gas analysis was performed to assess cardiorespiratory fitness.[83] Patients were given a Computer and Science Applications (CSA) accelerometer[190] to objectively measure their physical activity behaviour for a minimum of seven days. The monitor was attached to the subject's

right ankle. Individuals wore the accelerometer during all waking hours and recorded the time (see Appendix D) that the monitor was attached and removed each day until they returned for the second visit. Additionally, subjects were asked to record the activities they engaged in over the following week that were at least of a moderate intensity, defined as effort similar to a brisk walk. The physical activity recording sheet is shown in Appendix E.

Visit 2: Total duration 45 minutes

Approximately eight days after visit one, subjects returned for visit two. Patients were asked to fast for 12 hours prior to the visit and a blood sample was obtained to measure total cholesterol, triglycerides, HDL cholesterol and LDL cholesterol. The CSA accelerometer was returned and the Stanford Seven-Day Physical Activity Recall,[220] and the stages and processes of exercise behaviour change[260] questionnaires were completed. Thus, data collected with the accelerometer corresponded with information obtained from the seven-day physical activity recall. Participants were randomised (refer to randomisation procedure) to the experimental or control group. All participants received standard exercise information. Patients in the experimental group received an exercise consultation,[174] which was delivered either during the second visit or in a third visit depending on what was convenient for the patient and the researcher.

Six and 12 Month Assessment

All outcome measures recorded at baseline were repeated at six and 12 months. The experimental group received a further exercise consultation[174] at six months.

Randomisation

Participants were randomly allocated to the experimental or control group following the baseline assessment. Patients remained in the same allocation group throughout the study period. A blocked randomisation procedure was used to ensure two groups of equal size. Block sizes of 10 and 20 were used with an allocation ratio of one experimental to one control. For each block, a blinded research assistant generated a random sequence of numbers. This same individual then marked cards with "control" or "experimental" by allocating odd numbers to the control condition and even numbers to the experimental condition. These cards were placed in sequentially numbered opaque envelopes, which were then sealed. These envelopes were then allocated to

participants at visit two and were opened by the principal researcher after the baseline measures had been recorded.

Experimental Group

Patients in the experimental group received an exercise consultation[174] delivered after the baseline and 6 month assessments. In addition, patients received a support phone call three months and nine months after the baseline assessment.

Exercise Consultation at Baseline

All exercise consultations were conducted in accordance with guidelines published by Loughlan and Mutrie.[174] The researcher was trained in the exercise consultation process in a one-day course (BASES workshop, Dr Nanette Mutrie, 30th November 1998) and through the use of an Exercise Consultation video.[265] In addition, the researcher had previously conducted a pilot study to tailor the exercise consultation to cardiac patients who had recently completed a phase III exercise programme.[178] The consultation involved an individualised, one to one counselling session lasting between 20 and 30 minutes. Consultations were conducted in a private room containing two comfortable chairs. Excellent communication and counselling skills are required to effectively conduct a consultation. The researcher achieved this through an open posture (e.g. avoid crossing arms or legs), leaning towards the client, appropriate eye contact and a relaxed style to put the participant at ease and convey interest and attention. Counselling skills, including empathy (i.e. understanding the client's goals and needs), parroting (i.e. repeating the key words and phrases that the client used) and paraphrasing (i.e. summarising what the participant has said) were used to show the participant that the researcher had listened carefully and had understood what he or she had said. The exercise consultation is a client-centred approach, thus the researcher avoided preaching, lecturing or providing solutions for the client. However, the researcher could offer suggestions, such as how to overcome a certain barrier to activity, but watched for reactions to them, such as non-verbal clues.

As the participants had recently completed a phase III exercise programme, it was thought that participants would be either regularly physically active (i.e. in the action or maintenance stage of change) or doing some activity but not enough to meet current physical activity guidelines (i.e. preparation stage). Physical activity guidelines included accumulating at least 30 minutes of moderate intensity activity on most days of the

week[8] or engaging in a minimum of three 20 minute sessions of moderate to vigorous intensity exercise per week.[6] The aim of the consultation was to ensure that patients were regularly physically active and to encourage them to remain regularly active by preventing relapse.

The strategies used to increase and maintain physical activity were; previous activity history, decisional balance, problem solving barriers to physical activity, social support, goal setting and preventing relapse. A copy of the sheet that was used to guide the exercise consultation is included in Appendix C.

The consultation began by reviewing the participant's stage of exercise behaviour change and current physical activity behaviour using information collected during the baseline assessment (i.e. from the Stanford Seven-Day Physical Activity Recall and the Stage of Exercise Behaviour Change questionnaire). This was followed by a discussion on the participant's past and present activities to discover their likes and dislikes (e.g. if the patient enjoyed exercising in a group setting then they were encouraged to attend exercise classes in the community). Clients completed a decisional balance sheet, which involved comparing the perceived pros and cons of participating in physical activity. Patients were encouraged to identify the benefits they had gained by attending the phase III exercise programme. Examples of benefits included increased fitness, improved wellbeing, increased confidence and weight management. The researcher educated the patient on the importance of remaining active in phase IV to maintain these benefits. At this point, the researcher described the recommended amounts of physical activity required to improve and maintain health and fitness, and suggested ways to achieve these recommendations. Then participants thought of additional benefits that they would gain by remaining active in the long-term. Examples included preventing another heart attack, improved quality of life, living longer, and controlling weight. Patients were asked to identify possible barriers that may prevent them from remaining active in phase IV. For example, many patients' return to work after completing phase III exercise training, thus lack of time may be a potential barrier to activity in phase IV. The aim of the decision balance sheet was to help the participant realise that the benefits of increasing and maintaining activity outweighed the barriers. This is an effective technique for improving exercise adherence.[149] This followed with a discussion on ways to overcome potential barriers to activity in phase IV. Possible solutions to time constraints included using the stairs,

walking part or the entire journey to work or taking a brisk walk at lunchtime.

Social support is a major determinant of adherence to exercise in cardiac rehabilitation. Thus, an important part of the consultation was to encourage patients to seek support for their activity plan. For example, family or friends could engage in some activity with the patient or praise them for continuing with an active lifestyle. Alternatively, joining a group exercise programme provided a supportive environment for some individuals. Clients received information on local exercise opportunities that were suitable for phase IV cardiac patients. For example, patients could attend GP exercise referral schemes called "Living Plus" located in the surrounding leisure centres. These schemes involved group exercise classes and gym-based programmes lasting 10 weeks. Following completion of these programmes, maintenance exercise classes called "Fit for Life" were available. In addition, patients and their partners could attend a community exercise programme, which took place in the evening once a week. BACR phase IV maintenance exercise programmes were not available in the surrounding area until near the end of the study.

Relapse prevention training[150] was an important component of the exercise consultation process as patients had recently completed phase III. Participants were asked to identify high-risk situations that may cause a lapse from activity, for example bad weather or illness. These lapses can accumulate and may lead to a return to a sedentary lifestyle. Thus, the researcher helped the patient to acquire strategies to cope with these situations, for example having an alternative indoor activity in bad weather, thereby reducing the likelihood of a lapse in activity and an overall decline in physical activity. Finally, the client was encouraged to set short-term (1 month), intermediate (3 month) and long-term (6-month) specific activity goals. The researcher assisted the client with the goal setting to ensure that the goals were acceptable and achievable, met the client's needs and accounted for factors discussed during the consultation such as solutions to barriers, likes and dislikes, and relapse prevention strategies. Participants were given a copy of the activity goals to take away with them.

Exercise Consultation at Six Months

Information recorded during the baseline exercise consultation was reviewed in the six-month consultation. For example, participants were asked if they had achieved the activity goals set at baseline. If clients did not achieve their goals, then the reasons for

this were explored and new goals were set. Additionally, clients received feedback on their level of physical activity and cardiorespiratory fitness collected during the baseline and six-month assessments. Specifically, the researcher informed participants if they had improved, maintained or declined over the past 6 months in terms of their physical activity levels and fitness. If the patient's activity and fitness had declined from baseline to six months, then the reasons for this deterioration were explored. For example, clients were asked if they had encountered any barriers to activity or risky situations that had caused a lapse or relapse from activity over the past six months. Participants who had maintained or increased their activity from baseline to six months were praised by the researcher and instructed to "keep up the good work". However, barriers to activity, problem solving barriers, goal setting and relapse prevention strategies were briefly discussed with all patients to ensure they had acquired the necessary skills to help them remain active over the next six months.

Support Phone Calls

The exercise consultation also included two support phone calls, which were delivered three months after the baseline and six-month exercise consultations. The notes recorded during the exercise consultations were used to guide the phone calls. The phone calls lasted approximately 10 minutes and involved discussing any problems the patients were experiencing in achieving their activity goals, attending community exercise programmes, and remaining active.

Control Group

Subjects in the control group also received two phone calls, delivered three months after the baseline and six-month assessments, to maintain equal contact time between the experimental and control groups and to limit dropout from the study. The phone calls lasted approximately five to 10 minutes and the topics discussed were unrelated to exercise and included asking patients if they had attended their GP, any problems in relation to cardiac symptoms, medication or blood pressure. This is not standard management of patients in phase IV cardiac rehabilitation.

Standard Exercise Information

All study participants in the control condition and the experimental condition received a leaflet entitled Physical Activity and your Heart[266] published by the British Heart

Foundation after the baseline and six month measurements. Patients normally receive this booklet in phase I and on completion of phase III. The leaflet included information on the benefits of regular physical activity, CHD risk factors, physical activity recommendations and examples of how to become more active.

Primary Outcome Measures

Physical Activity

Stages of Exercise Behaviour Change

The stages of exercise behaviour change[156] are: Precontemplation (inactive and no intention to change), Contemplation (inactive, but intending to change in the next 6 months), Preparation (engaging in some activity, but not regularly), Action (regularly physically active, but only began in the past 6 months), and Maintenance (regularly active for more than six months).[156] Patients in action or maintenance are meeting guidelines[6;8] for regular physical activity.

The method used to assess the stages of exercise behaviour change has been validated against subjective and objective measures of physical activity.[96;144;157-159] Studies have reported an increase in self-reported physical activity and cardiorespiratory fitness with advancing stage of change in healthy adults and cardiac rehabilitation participants.[96;144;157-159] The original stages of exercise behaviour change questionnaire developed by Marcus[160] in 1992 defined regular physical activity as "exercising at least three time per week for 20 minutes." The reliability and validity of this questionnaire has been demonstrated.[157;267] However, Loughlan and Mutrie[260] modified this questionnaire to include the 1995 physical activity recommendation (i.e. 30 minutes of accumulated moderate activity on five days per week). The validity of the modified questionnaire has been demonstrated in the general population.[268] Lowther and Mutrie[268] found that subjects in the action and maintenance stages (i.e. meeting the definition of regular physical activity) reported significantly more physical activity compared to those in preparation and contemplation. In addition, those in the preparation stage participated in significantly more physical activity compared to subjects in the contemplation stage. Similarly, a recent study used both physical activity recommendations to assess the stage of exercise behaviour change of a group

of patients before and after participation in 12 weeks cardiac rehabilitation.[96] The modified stage of exercise behaviour change questionnaire,[260] which included the two physical activity recommendations was selected for use in this study.

The questionnaire[260] included a clear description of each stage of exercise behaviour change and regular physical activity was based on the current physical activity recommendations[6;8] (i.e. a minimum of 20 minutes of continuous, moderate to vigorous exercise on three days per week or accumulating a minimum of 30 minutes of moderate physical activity on five days per week. Based on this definition, subjects selected the stage of change that described their physical activity behaviour over the past **six** months. At the baseline assessment only, the stage of change questionnaire measured individuals physical activity behaviour over the past **two** months, as they had suffered a cardiac event in the preceding six months, but had attended cardiac rehabilitation classes in the past two months.

Stanford Seven-Day Physical Activity Recall

The reliability and validity of the Stanford Seven-Day Physical Activity Recall[220] (7DPAR) was discussed in Chapter One. The 7DPAR was administered in a structured interview format by the same researcher throughout the study period. A standardised interview process was followed[222] to limit the interviewer's bias, minimise guessing and over- or underestimating a participant's physical activity. Gross found that individuals trained in the standardised interview process reliably administered and scored the seven-day physical activity recall.[225] The researcher performed a number of practice sessions conducting and scoring the questionnaire with cardiac rehabilitation participants before commencing the study.

Subjects were asked to recall the duration spent sleeping and participating in moderate, hard and very hard intensity physical activity for each of the previous seven days. Participants were reminded that they had been wearing the CSA accelerometer, and were asked to recall the duration and intensity of their activities as accurately as possible and to report the activities they actually engaged in over the previous week instead of activities they would normally do. Information from the activity recording sheet (Appendix E) administered during visit one was used to assist patients with their recall. To further assist with recall, subjects were asked to recall activities in the morning, afternoon and evening for each of the seven days, beginning with the

previous day. After each day of recall and at the end of the interview, the participant was asked if there was any activity that they may have overlooked or forgotten. During the interview, prompts were used to help participants recall their activity. For example, general questions like "Did you do any housework or gardening that was similar in intensity as a brisk walk?" or "How do you get to and from work?" were asked. Classifying the intensity level of activities is the most difficult and ambiguous part of the interview process. Thus, subjects were instructed that moderate intensity activities are similar to a brisk walk, very hard intensity activities are equivalent to running and hard activities are in between brisk walking and running (e.g. a brisk walk uphill). Physiological symptoms, such as perceptions of sweating, heart rate, and breathing were not used to define intensity because they vary greatly by age, weather, fitness level, cardiac medication and condition. In addition, subjects were shown a list of activities in each intensity category. Patients were instructed that light intensity activities were not included in the questionnaire, such as desk work, standing, light housework (e.g. ironing, washing dishes etc), strolling, and walking for less than 5 minutes (e.g. shopping). Subjects were asked about the duration of each activity excluding breaks (e.g. being at the swimming pool for 1 hour but only swimming for 30 minutes was recorded as 30 minutes, not 1 hour). The information recorded by the seven-day recall was summarised as follows: the number of minutes each day spent in sleep, and moderate, hard and very hard intensity activity was totalled for the week. Minutes spent in light intensity activity per week was determined by subtracting the time in sleep, and moderate to very hard activity from 10080 minutes. Total physical activity per week was calculated by summing the duration of moderate, hard and very hard activities per week.

Total weekly energy expenditure can be estimated by multiplying the time spent in sleep (1 MET), light (1.5 METs), moderate (4 METs), hard (6 METs) and very hard (10 METs) activities per week by their corresponding MET value. However, total energy expenditure was not determined in this study for the following reasons. The fixed MET values for estimating energy expenditure were derived from young adults. Duncan found that actual MET values for moderate, hard and very hard intensity activity were 3.8, 5.3 and 6.2, respectively in a group of sedentary middle aged adults, which are lower than the fixed MET values used by the recall.[223] Furthermore, Duncan reported that the average aerobic capacity of the study group was 7.3 METs, thus these individuals would be unable to perform very hard activity (10 METs according to the

recall) as it would require 137% of their aerobic capacity. Therefore, fixed MET values used by the 7DPAR to estimate energy expenditure may not be appropriate for individuals with low exercise capacities. In addition, several studies reported that the seven-day recall significantly overestimated energy expenditure compared to DLW.[230-232] Thus, minutes per week in various physical activity intensities were used instead.

Computer Science and Applications Accelerometer

The Computer Science and Applications accelerometer (CSA) model 7164 (Shalimar, Florida) is small (5.1 x 3.8 x 1.5 cm), lightweight (42.6 g), measures dynamic movement in one direction and can be worn on the waist, ankle or wrist.[190] This instrument is illustrated in Figure 6. The CSA measures vertical movement using a piezoelectric bender, which generates a signal in response accelerations during bodily movement. The signal generated is proportional to the magnitude of the acceleration. Thus, greater accelerations produce higher signals. The acceleration signal is digitised, and these values are summed over a one-minute time interval (epoch). At the end of each epoch, the summed value is expressed as an activity count and is stored in the memory. Each activity count represents the sum of accelerations during one minute. Thus, the greater the intensity of activity (i.e. in terms of the magnitude and frequency of accelerations) the higher the activity count. The CSA measures accelerations ranging in magnitude from 0.05 to 2 G's in the vertical (z) direction. The instrument detects movements that occur within a frequency response from 0.25 to 2.5 hertz so that normal human movement is detected and nonphysiological motion is filtered out. The reliability and validity of the CSA for measuring physical activity was discussed in Chapter One. However, a pilot study[269] was conducted to examine the relationship between CSA accelerometer counts and VO_2 during daily living activities in 15 cardiac patients recruited from a phase III hospital-based programme. Patients walked at three self-selected speeds (slow, normal and brisk) and performed other daily living activities (i.e. carrying loaded shopping bags and pushing a loaded shopping trolley at the normal walking pace). During each activity, steady state VO_2 (ml/kg/min) was measured using a portable metabolic system (Cosmed K4b^2) and activity counts/minute were recorded using CSA accelerometers worn on the waist, ankle and wrist. This study found that VO_2 (ml/kg/min) and activity counts/minute significantly increased as walking speed increased. In addition, VO_2 (ml/kg/min) for carrying shopping bags and pushing shopping trolley were similar and significantly higher than normal walking. In contrast, activity counts/minute recorded during carrying

shopping bags and pushing shopping trolley were not significantly higher compared to normal walking. Similar results were obtained for CSA accelerometers worn on the wrist and ankle. Findings from this study suggest that changes in VO_2 during daily living activities are not always reflected by changes in accelerometer counts. Thus, CSA accelerometers do not accurately record all types of activity.

The CSA was operated according to the manufacturer's instructions.[190] The instrument was initialised to begin recording at visit one and the monitor was instructed to collect data in one-minute time intervals (i.e. epochs), thus output from the CSA was in activity counts per minute. The CSA was attached to the right ankle with a Velcro strap above the anklebone with the orientation of the notch toward the knee, as specified by the manufacturer. Although most studies using CSA accelerometers have attached the monitor to the waist, the ankle was chosen as the attachment site in this study. This was because during pilot studies,[263;269] subjects reported that monitors attached to a belt and worn on the waist frequently became loose and turned upside down. Participants were instructed to wear the monitor during all waking hours except during bathing or other water activities. Subjects recorded the time that the monitor was attached and removed each day, to ensure that the monitor was being worn during all waking hours. The recording sheet is shown in Appendix D. In addition, participants were asked to ensure that the orientation and placement of the monitor on the ankle was held constant from day to day in order to obtain accurate repeatable data. Participants wore the accelerometer for a minimum of seven days, so that the accelerometer data would correspond with the information obtained from the Stanford Seven Day Physical Activity Recall. The monitor was returned at visit 2 and data recorded by the accelerometer were downloaded to a desktop computer using a CSA reader interface unit connected to the PC serial port. Data were exported to excel and activity counts per minute for the seven-monitored days were summed to produce total activity counts per week. Activity counts were **not** used to estimate energy expenditure or the time spent in various intensity categories due to limitations of the equations used to predict these variables from accelerometer readings. Due to the small number of monitors available for use in the study, it was not possible to use the same CSA monitor on the same individual at each of the three measurement periods.

Figure 6 The Computer Science and Applications (CSA) accelerometer for measuring physical activity

Cardiorespiratory Fitness

Cardiorespiratory fitness, specifically exercise capacity and submaximal endurance capacity, was measured using a cardiopulmonary exercise test. This test was carried out on a motorised treadmill (Quinton 65) with gas exchange and ventilatory variables being analysed breath by breath using the Cosmed $K4b^2$ (Cosmed, Italy). All tests were performed in the same laboratory.

Cosmed $K4b^2$

The Cosmed $K4b^2$ (Cosmed, Italy) is a breath by breath portable metabolic system, illustrated in Figure 7. The Cosmed $K4b^2$ is valid for measuring gas exchange variables across a wide range of exercise intensities.[270;271] In addition, a recent study found no significant difference between the Cosmed $K4b^2$ and a stationary metabolic cart for measuring gas exchange during treadmill exercise in a group of cardiac patients attending phase III exercise programmes.[272] Although the reliability of the Cosmed $K4b^2$ has not been assessed, the earlier model (Cosmed K4) was shown to be reliable.[273] The portable unit of the Cosmed $K4b^2$ contains the oxygen and carbon dioxide analysers, sampling pump, UHF transmitter, barometric sensors and electronics and is powered by a rechargeable battery.[274] The total system weighs less than 1 kg. The oxygen analyser has a measurement range of 7-24% O_2, and accuracy to 0.02% O_2. The carbon dioxide analyser has a measurement range of 0 – 8% CO_2 and accuracy to 0.01% CO_2. The O_2 and CO_2 analysers have a rapid response time and are thermostated and compensated for variations in barometric pressure,

temperature, and environmental humidity. The flowmeter contains a bi-directional digital turbine that measures respiratory flow with a flow range capacity up to 20 litres/sec and a wide ventilation range between 0 to 300 l/min with accuracy to ±2%. The flowmeter is fixed to a soft facemask (Hans-Rudolph, Kansas City, MO), that covers the subject's mouth and nose. The masks are available in different sizes and have two inspiratory valves to reduce inspiratory resistance. A Permapure tube connected to the flowmeter and the portable unit is used to sample oxygen and carbon dioxide in expired air.

The manufacturer recommended that the portable unit (connected to the charger unit) was warmed up for at least 45 minutes before use.[274] Before each exercise test, the following calibrations were performed, according to the manufacturer's guidelines,[274] to ensure accurate and reliable readings. The system automatically performs a room air calibration by sampling room air to update the CO_2 analyser baseline and the O_2 analyser gain to match the readings with predicted atmospheric values (20.93% for O_2 and 0.03% for CO_2). The reference gas calibration involves sampling a gas with a known composition (i.e. 16% O_2 and 5% CO_2, balance N_2) from a calibration cylinder and updating the baseline and the gain of the analysers to match the measured readings with the predicted values. The output pressure from the calibration cylinder was set between 300 to 500 Kpa to ensure that room air was not mixed with the reference gas, which would affect the calibration. The delay calibration measures the time necessary for the gas sample to pass through the sampling line before being analysed. The turbine calibration consists of measuring the volume of a 3 litre calibration syringe and updating the gain of the flowmeter in order to match the predicted value. The calibration results were viewed to ensure that the measured values were within the acceptable range of the predicted values.

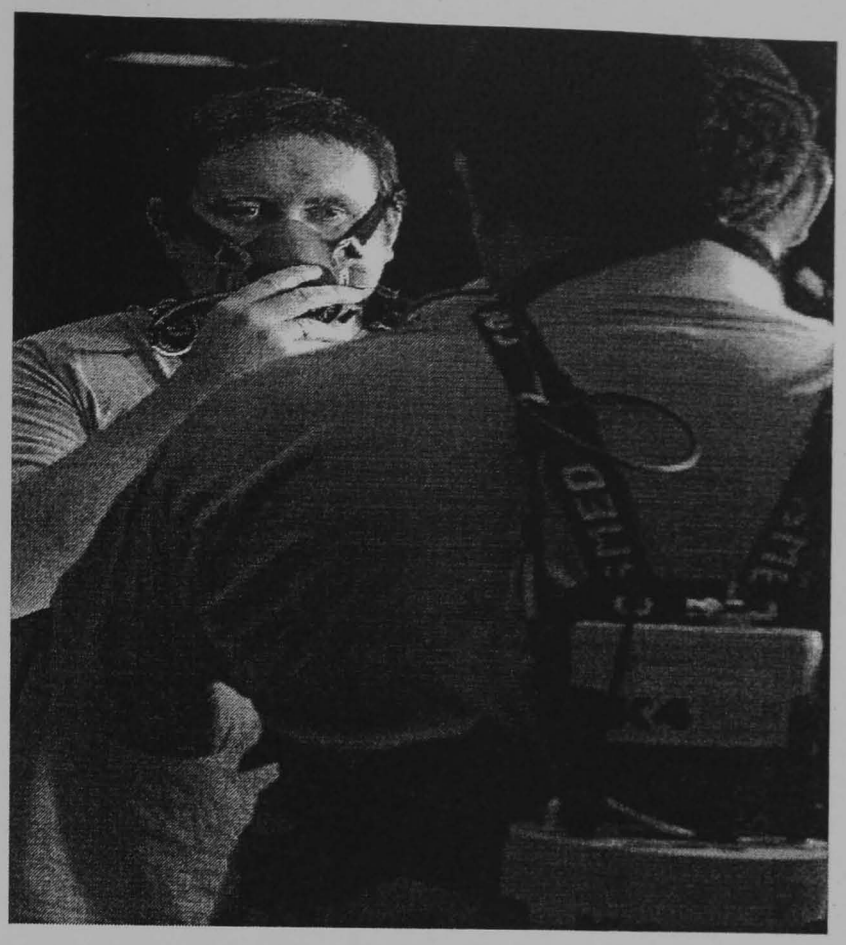


Figure 7 The Cosmed K4b^2 portable metabolic system

Exercise Testing Protocol

An individualised exercise testing protocol[275] was devised to accommodate each patient's capabilities and to achieve an optimal exercise test duration of 8 to 12 minutes (excluding the 3 minute warm-up phase) for reasons previously discussed in Chapter One. The protocol consisted of: 1) 2 minutes at rest; 2) 3 minute warm-up phase, which involved gradually increasing treadmill speed to achieve a brisk walking pace (range 1-3.3mph), at 0% gradient; and 3) incremental phase, which involved increasing treadmill gradient by 1%, 1.5% or 2% each minute until the test was terminated. However, if after five minutes of incremental exercise, the respiratory exchange ratio (RER) values were less than 0.90 and there were no ischaemic ECG changes, the treadmill speed was increased by 0.3 to 0.5mph per minute to achieve a fast walk or jog, (the gradient did not change). Once a fast walk or jog was achieved, the speed did not changed and the gradient was increased by 1.5% to 2% per minute until the test was terminated.

Exercise Testing Procedure

An established procedure for conducting cardiopulmonary exercise tests was followed.[83] Patients were advised to wear comfortable clothes and shoes, to adhere to his or her usual medical regimen, to eat a light meal no less than 2 hours before the test, and to avoid cigarettes and caffeine for at least 2 hours. A full explanation of the

exercise test procedure was given to standardise the response to the test and to reduce the patient's anxiety.

A cardiac technician fitted each patient with a 12 lead ECG and an automated blood pressure cuff. A resting 12 lead ECG and a blood pressure reading was obtained with the subject in a seated position. A heart rate belt was fixed to the patient's thorax and the heart rate probe was attached to the harness. The $K4b^2$ portable unit and the battery were fixed to the back plates of the harness worn by the patient. The facemask was attached to the participant with a nylon mesh hairnet and velcro straps. The researcher ensured that the facemask fitted well and was fixed securely to prevent air leakage. The ambient humidity was determined using a hygrometer and entered into the portable unit along with physical characteristics of the participant (age, weight, height, and gender). After performing a room air calibration, the portable unit began recording gas exchange data. The data recorded by the portable unit was transmitted breath by breath to a laptop allowing the data to be displayed in table and graphic formats.

Patients performed a practice walk on the treadmill to familiarise themselves with the treadmill and the equipment. Then subjects were seated and remained in this position until values for ventilation and RER returned to resting levels (i.e. ventilation below 10 l/min and RER less than 0.85) to ensure that the patient was not hyperventilating, which may affect the estimation of the lactate threshold. During the exercise test patients received standardised encouragement from the cardiac technician, who was blinded to group allocation, to maximise exercise performance. Throughout the test, a 12 lead ECG was monitored continuously and blood pressure readings were taken every 3 minutes. The patient could terminate the test by symptoms of chest pain, fatigue, and breathlessness. The supervising cardiac technician could also stop the test in the event of significant ECG changes (≥2mm ST depression), significant arrhythmia or a fall in blood pressure. The following data were obtained breath by breath: oxygen uptake (VO_2 ml/min), VO_2 per kg (VO_2 ml/kg/min), carbon dioxide output (CO_2 ml/min), respiratory frequency (Rf breath/min), tidal volume (VT litres), heart rate (HR beats/min), ventilation (VE l/min), respiratory exchange ratio [RER (VCO_2/VO_2)] ventilatory equivalent for O_2 (VE/VO_2), ventilatory equivalent for CO_2 (VE/VCO_2), oxygen pulse (VO_2/HR ml/beat), end tidal partial pressures of O_2 ($PETO_2$) and CO_2 ($PETCO_2$). On test completion, data were downloaded from the portable unit

to the laptop and then exported to an excel spreadsheet for data management.

Data Analysis

Data were managed in excel in the following way. Graphs were drawn for VO_2, VCO_2 and VE versus time. Outlying points caused by the patient talking, coughing or abnormal breathing patterns (e.g. patient taking a deep breath) were deleted from the data sheet as they could affect the detection of the lactate threshold. The edited data was then used to determine exercise capacity and submaximal endurance capacity.

Exercise Capacity

Exercise capacity was determined by calculating the following variables at peak exercise:

1. Peak exercise duration (excluding the three minute warm-up phase)
2. Peak VO_2 ml/min (average VO_2 for the last 30 secs of the exercise test)
3. Peak VO_2 ml/kg/min (average VO_2 for the last 30 secs of the exercise test).
4. Peak oxygen pulse (VO_2/HR ml/beat) was calculated from peak VO_2 divided by peak heart rate (highest heart rate measured by the K4b^2 and ECG). This variable measures the amount of oxygen carried in each stroke volume, and gives an index of cardiac function.
5. Peak respiratory exchange ratio (RER) is the ratio of VCO_2/VO_2 at peak exercise and was used as an indicator of exercise effort. An RER greater than 1.15 at peak exercise indicates a maximal effort.[83]

Submaximal Endurance Capacity

Submaximal endurance capacity was determined by estimating the lactate threshold using the V-slope and ventilatory equivalent methods, which were previously described in Chapter One. The V-slope and ventilatory equivalent methods were carried out using an established procedure.[276] Breath by breath values for VO_2, VCO_2, VE/VO_2, $VEVCO_2$, $PETO_2$, and $PETCO_2$ were averaged (i.e. 4, 6 and 8 breath averages), which makes it easier to estimate the threshold. For the V-slope method, a graph of VCO_2 versus VO_2 graphs was drawn and the lactate threshold was estimated by visual inspection of the VO_2 at which a breakpoint in the VCO_2/VO_2 relationship is first evident. For the ventilatory equivalent method, graphs of VO_2 versus VE/VO_2, VE/VCO_2, $PETO_2$, and $PETCO_2$ were drawn. The lactate threshold was determined by the VO_2 at which an increase in V_E/VO_2 and $PETO_2$ occurs with no change or a

decrease in V_E/VCO_2 and $PETCO_2$. These methods are demonstrated in Chapter One.

Secondary Outcome Measures

Psychological Functioning

Quality of Life

Quality of life was assessed using version 2 of the UK Short Form-36.[261] It is a self-administered generic instrument containing 36 items that measure the following 8 dimensions of general health: physical functioning (PF), social functioning (SF), role limitations due to physical problems (RLP), role limitations due to emotional problems (RLE), pain (P), energy/vitality (EV), mental health (MH) and general health perception (GHP). Scores for each dimension are obtained by reverse scoring, where necessary and summing responses. Raw scores can then be transformed on to a scale from 0 (worst possible health) to 100 (best possible health).

The validity, reliability and responsiveness of the SF-36 to assess quality of life in cardiac patients has been demonstrated (Chapter One). In 1999, version 2 of this questionnaire was developed to overcome some of the problems with the original instrument.[259] The wording and layout of the SF-36 was modified and the responses to the two role functioning domains were changed from dichotomous scales (yes/no) to five point response categories to reduce ceiling and floor effects for these dimensions. In addition, minor modifications to the wording of six items were made to make the questionnaire acceptable in the British context. The UK SF-36 version 2 demonstrated greater internal consistency and fewer floor and ceiling effects compared to the original questionnaire, which may improve the responsiveness of the instrument.[259] Normative values for this version are available for a UK general population.[259]

Anxiety and Depression

Anxiety and depression was assessed by the Hospital Anxiety and Depression Scale.[240] The validity, reliability and responsiveness of the HADS for measuring anxiety and depression in medical patients, including individuals with CHD has been discussed (Chapter One). The Hospital Anxiety and Depression Scale (HADS) is a brief, self-report questionnaire that measures the states of anxiety and depression over the past week. The questionnaire is divided into anxiety and depression subscales both containing 7 items, each item is rated on a 4 point scale from 0 to 3, giving total scores ranging from 0 to 21 for each subscale. The HADS scores can be used to classify

individuals into normal (0-7), mild (8-10), moderate (11-14) and severe (15 or above) categories for anxiety and depression.[245] In addition, it has been suggested that a HADS score of ≥ 8 for both subscales identifies possible cases of psychological distress in the general population and medical patients.[239]

Processes of Exercise Behaviour Change

The processes of change for exercise behaviour[160] are strategies that people use when changing their exercise behaviour. There are five experiential (consciousness raising, social liberation, dramatic relief, environmental re-evaluation, self re-evaluation) and five behavioural processes (helping relationships, stimulus control, counter conditioning, self-liberation, reinforcement management), see Table 6 for definitions. The ten processes were assessed using a 40 item questionnaire that asked participants to indicate on a 5 point likert scale ranging from 1 (never) to 5 (repeatedly) how frequently in the past month they had used or thought about each process.[160] The internal consistency for each process and the validity of the processes of change questionnaire has been demonstrated.[160;277] Several studies have used the questionnaire to measure process use in cardiac rehabilitation populations.[96;144;161] Recently, Bock reported significant increases in the use of behavioural processes following participation in a 3 month cardiac rehabilitation exercise programme.[96]

Lipids

A 12 hour fasting blood sample was obtained and analysed to determine total cholesterol, high-density cholesterol (HDL-C), low-density cholesterol (LDL-C) and triglycerides. The blood samples were analysed by the biochemistry laboratory at the Royal Alexandra Hospital. The concentration of total cholesterol, tryglycerides and HDL cholesterol in serum was directly quantified by enzymatic methods using an automated clinical chemistry analyser (Hitachi 917, Roche Diagnostics Ltd). LDL cholesterol was calculated using the Friedewald equation.[278] The accuracy and reliability of the biochemistry laboratory for measuring total cholesterol, triglycerides, HDL cholesterol and LDL cholesterol is tested throughout each day by analysing control samples and monthly by analysing reference samples supplied by a UK-CDC Network Laboratory at the Glasgow Royal Infirmary. The difference between values measured by the laboratory and target values were within acceptable limits (i.e. ± 5%).

Smoking Status at Baseline

Smoking status at baseline was assessed by asking patients if they were a current smoker, an ex smoker or a non-smoker.

Social Class at Baseline

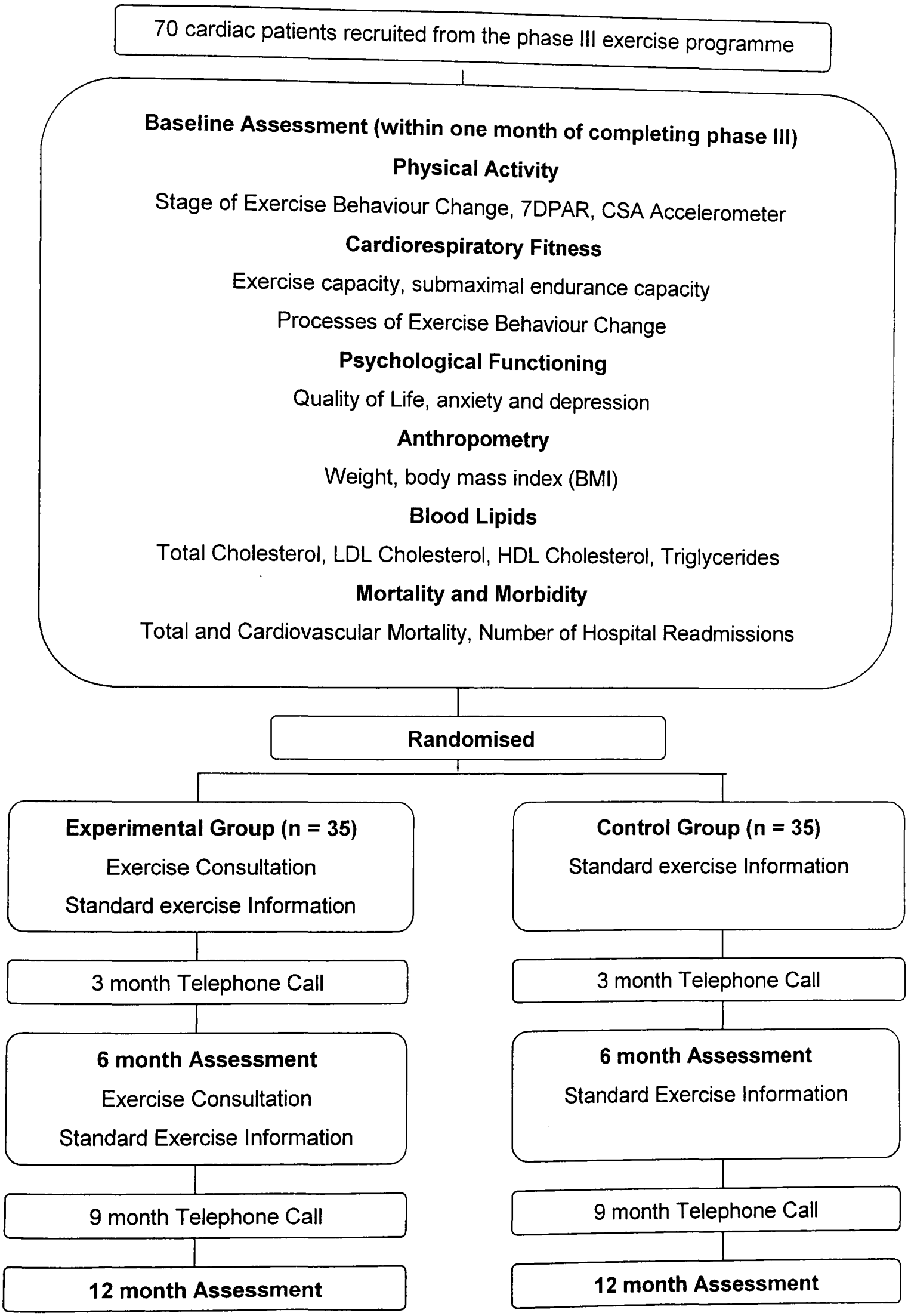
Social class at baseline was determined using the new National Statistics Socio-Economic Classification[264] (NS-SEC), which replaces the two previous classifications; Social Class based on Occupation (SC) and Socio-economic Groups (SEG). The SEC has been thoroughly tested and validated using the Labour Force Survey and a variety of national datasets (e.g. health, mortality and employment).[264] The SEC has a full version, which involves 14 functional classes based on occupation and employment status. In addition, the full version can be collapsed into 9, 8, 5 or 3 socio-economic classes for analytical purposes. The full version of the SEC is very similar to the SEG, whereas the collapsed versions of the SEC resemble the SC. Thus, all three methods are closely related.

The new classification (SEC) groups individuals according to both occupation and employment status.[264] Therefore, information was collected on the patient's occupation and employment status (i.e. self-employed with 25 plus employees, self-employed with less than 25 employees, self-employed with no employees, and employee). In addition, information was collected on employees with managerial or supervisory status (i.e. manager with 25 plus employees, manager with less than 25 employees, supervisor/foremen and employee). Unemployed and retired individuals were classed using their last main job. In the five and three class versions, individuals who have never worked were allocated to working class. Women who had never worked and were married were classified using their husbands' occupation and employment status. The three-class version, which categorises individuals into Managerial/Professional, Intermediate and Working Class, was used to determine social class in this study.

Mortality and Morbidity

Total and cardiovascular mortality was recorded by reviewing patient's medical records. The number of cardiac hospital re-admissions (e.g. MI, UAP, receiving PTCA or CABG) was recorded by reviewing medical records and asking patients at the six and 12-month follow-up visits.

Figure 8 Summary of Study Design



Statistical Analysis

Analyses were performed using computer software packages Minitab version 13.30 and SPSS version 11.0. Normality of all data were assessed by examining histograms, boxplots and normal probability plots of the data. In addition, comparisons of descriptive statistics were used to assess the normality of the data; such as mean and median, minimum and maximum values, and upper and lower interquartile ranges. Normally distributed data are expressed as mean ± standard deviation (SD); median and interquartile range (IQR) are used for data that are not normally distributed. The General Linear Model (GLM) repeated measures statistical test was considered to determine the effect of the exercise consultation (experimental) compared with standard exercise information (control) on the dependent variables (e.g. physical activity) from baseline to six and 12 months. The GLM test is based on the assumption that the data come from normal distributions within the groups and that the variances of these distributions are the same. Normal probability plots of the residuals, plots of residuals versus fitted values and the Bartletts test of equal variance were used to check these assumptions. However, results of these tests suggested that the assumption of normality and equal variance were not met for the following dependent variables (i.e. physical activity, lipids, quality of life, processes of change, anxiety and depression). In addition, performing normality and equal variance tests on log and square root transformed data suggested that the transformed data did not meet these assumptions. Therefore, using GLM repeated measures to analyse the data was rejected.

The following statistical tests were performed instead. Normally distributed data were analysed using paired student's t tests for within group analyses and two sample t tests for between group analyses. Data that was not normally distributed were analysed using Wilcoxon signed rank tests for within group differences and Mann Whitney tests for differences between groups. Ninety eight percent confidence intervals were used to quantify the between and within group differences. Ninety eight percent confidence limits were used to control for Type 1 errors due to the number of comparisons being conducted. Categorical data are reported as number of patients and proportions and analysed using chi square tests and Fisher's exact tests. Dr Lilian Murray, Statistician, University of Glasgow provided statistical advice.

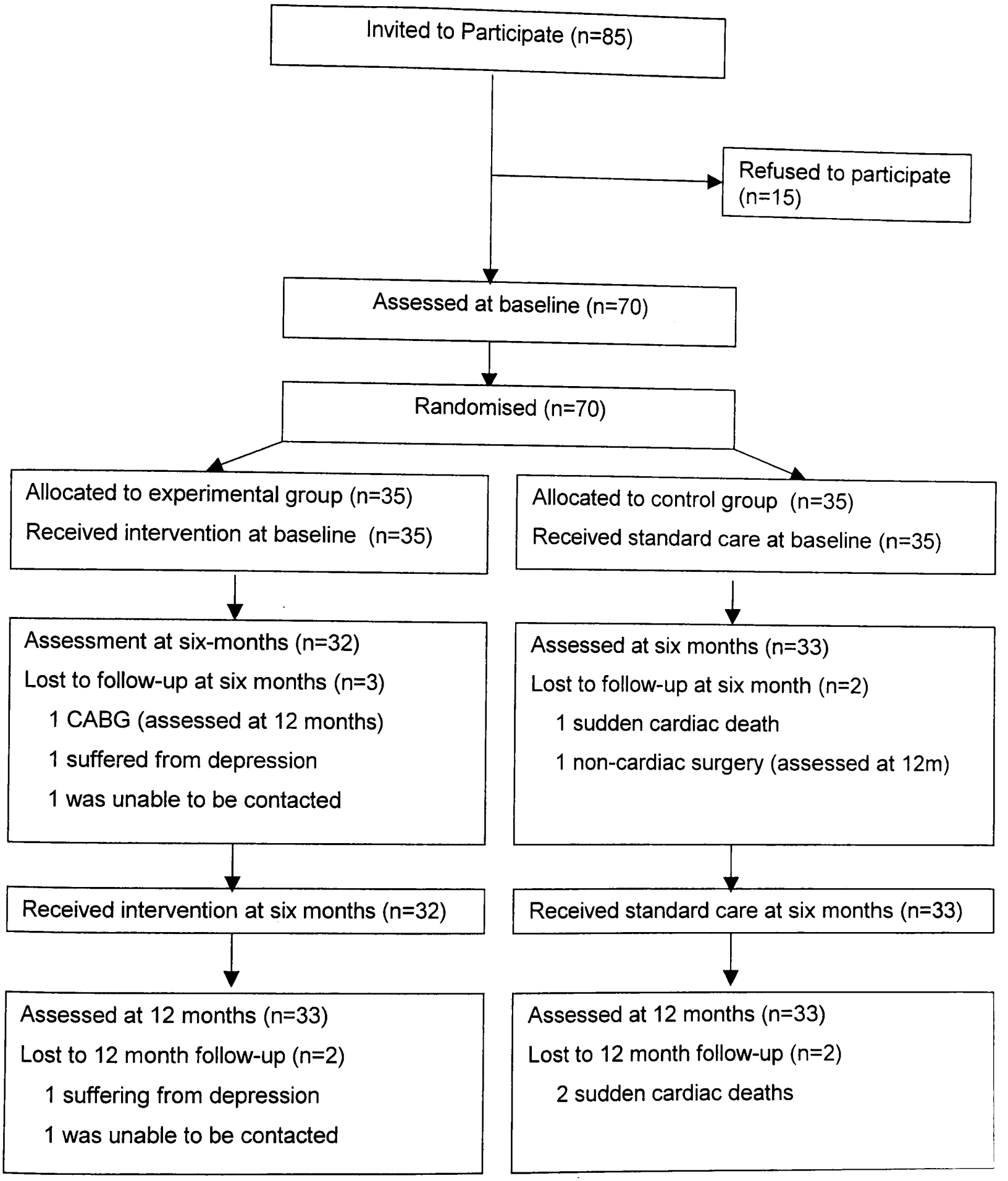
CHAPTER FOUR

RESULTS

Flow of Participants Through the Study

Figure 9 demonstrates the flow of participants through each stage of the study. In total, 85 patients were invited to participate in the study, and 15 patients refused to take part. Therefore, 70 patients were assessed at baseline and then randomised to the experimental or control group. At baseline, all patients received the allocated intervention or standard care. Ninety three percent (65/70) of the sample were assessed at six months (n=32 experimentals and n=33 controls). Two control patients were lost to follow-up at six months; one patient died and one patient received non-cardiac surgery. Three experimental patients were lost to the six month follow-up; one patient received CABG, one patient could not be contacted and one patient was suffering from depression. Ninety four percent (66/70) of the sample were assessed at 12 months (n=33 experimentals and n=33 controls). In the control group, two patients had died at 12 months. In the experimental group, one patient could not be contacted and one patient was suffering from depression at 12 months. In addition, the two patients who received surgery at six months, thus were lost to the six-month follow-up, returned for the 12 month assessment.

Figure 9 Flow of participants through the randomised controlled trial



Baseline Demographic and Clinical Characteristics

Table 7 displays the baseline demographic and clinical characteristics of the experimental and control groups. The experimental and control groups were comparable for age (95%CI -3.1, 7), and gender (χ^2=1.4, p=0.23). However, mean weight (kg) of the experimental group was significantly greater compared to the control group (95%CI -13.8, -0.1). In addition, there was trend for a greater mean body mass index in the experimental group compared to the control group (95%CI -3.1, 0.2). As shown in Table 7, the majority of patients in both groups were male and had suffered a myocardial infarction. A small number of experimental and control patients were current smokers, had suffered a previous MI or were diagnosed with type II diabetes mellitus. Most of the patients in each group were married or cohabiting. The majority of patients in the experimental and control groups were either employed or retired. Within each group, a comparable number of patients were employed or retired. In the experimental group, 46% (16/35) were in the managerial/professional social class and 37% (13/35) were working class. Similarly, 43% (15/35) of the control group were in the managerial/professional social class and 29% (10/35) were working class.

Table 7 Baseline Demographic and Clinical Characteristics of Study Groups

Characteristic	Experimental (n = 35)	Control (n = 35)
Mean age ± SD, y	59.0 ± 9.7	60.9 ± 11.3
Males, n (%)	30 (86)	26 (74)
Mean weight ± SD, kg	86.0 ± 13.9	79.1 ± 14.7
Mean BMI ± SD, kg/m^2	28.5 ± 3.5	27.0 ± 3.5
Cardiac diagnosis, n (%)		
MI	22 (62.9)	24 (68.6)
CABG	8 (22.9)	6 (17.1)
PTCA	4 (11.4)	3 (8.6)
UAP	1 (2.9)	2 (5.7)
Smoking status, n (%)		
Current Smoker	2 (5.7)	4 (11.4)
Previous Smoker	22 (62.9)	21 (60.0)
Non Smoker	11 (31.4)	10 (28.6)
Previous MI, n (%)	7 (20.0)	6 (17.1)
Diabetes Mellitus, n (%)	4 (11.5)	5 (14.3)
Marital status, n (%)		
Married/cohabiting	29 (82.9)	28 (80.0)
Widowed/divorced/single	6 (17.1)	7 (20.0)
Employment status, n (%)		
Employed	16 (45.7)	16 (45.7)
Retired	16 (45.7)	15 (42.9)
Unemployed	3 (8.6)	1 (2.9)
Unable to work due to illness	0	3 (8.5)
Socio-economic class, n (%)		
Managerial/Professional	16 (45.7)	15 (42.9)
Intermediate	6 (17.1)	10 (28.6)
Working	13 (37.1)	10 (28.6)

BMI = body mass index; MI = myocardial infarction; CABG = coronary artery bypass graft; PTCA = percutaneous transluminal coronary angioplasty; UAP = unstable angina pectoris.

A similar number of experimental and control subjects were taking the cardiac medications described in Table 8. As shown in Table 8, 74% (26/35) of patients in each group were taking beta-blocker medication. Only one patient from the sample population was not prescribed anti-platelet medication, this patient was taking anti-coagulent therapy instead. In addition, 89% (31/35) of patients in the control group and 100% of those in the experimental group were taking lipid-lowering medication. Forty percent (14/35) of the control group and 26% (9/35) of the experimental group were prescribed oral nitrates or nitrate patches for angina.

Table 8 Proportion of Patients Taking Cardiac Medication at Baseline

Cardiac Medication	Experimental (n = 35)	Control (n = 35)
Beta blocker	26 (74.3)	26 (74.3)
Lipid lowering	35 (100)	31 (88.6)
Anti-platelet	34 (97.1)	35 (100)
ACE inhibitor	19 (54.3)	19 (54.3)
Nitrate[a]	9 (25.7)	14 (40.0)
Calcium channel blocker	12 (34.3)	10 (28.6)
Anti-coagulent	1 (2.9)	0
Diuretic	6 (17.1)	6 (17.1)

[a] Nitrate includes oral nitrate and nitrate patch, nitrate spray is not included.

Note. Values represent number of patients and percentages in parentheses.

Physical Activity at Baseline

This section describes the baseline physical activity level of the total study sample (n=70).

Physical activity was measured objectively using the CSA accelerometer,[190] and subjectively using the Stanford Seven-Day Physical Activity Recall[220] and the Stage of Exercise Behaviour Change.[220] The Seven-Day Recall measures the duration of sleep, and light, moderate, hard, very hard and total physical activity in minutes per week. Total physical activity is the sum of moderate, hard and very hard intensity physical activity. The stages of exercise behaviour change are: Precontemplation (inactive and no intention to change), Contemplation (inactive, but intending to change in the next 6

months), Preparation (engaging in some activity, but not regularly), Action (regularly physically active, but only began in the past 6 months), and Maintenance (regularly active for more than six months). Patients selected the stage of exercise behaviour change that described their physical activity behaviour over the previous two months at baseline. Patients in the action or maintenance stages considered themselves to be regularly physically active.

Stage of Exercise Behaviour Change

There were no study patients in the precontemplation or contemplation stages of change at baseline. Results revealed that 17.1% (12/70) of the study sample were in preparation, 45.7% (32/70) were in action and 37.1% (26/70) were in maintenance at baseline. Thus, 83% of the sample population were regularly physically active at baseline. This is because physical activity was measured within one month of the study sample completing an 11-week phase III exercise programme.

Computer Science and Applications (CSA) Accelerometer

CSA accelerometer counts/week at baseline were not normally distributed, thus median and interquartile range values are reported. In the total group, median accelerometer counts/week at baseline were 4025432 (3034626, 5095810).

Stanford Seven-Day Physical Activity Recall

Baseline physical activity in minutes per week measured by the Stanford Seven-Day Recall were not normally distributed, thus median and interquartile range values are stated. Only two patients reported participating in very hard intensity physical activity, thus hard and very hard intensity categories were combined to produce a vigorous intensity activity category. Table 9 reports median values for total, moderate, vigorous and light intensity physical activity in minutes/week at baseline for the study group. Subjects participated in 248 minutes/week of moderate intensity activity and 26 minutes/week of vigorous intensity activity. Total physical activity, which was the sum of moderate and vigorous intensity activity, was 288 minutes/week at baseline.

Table 9 Baseline Physical Activity (mins/week) Measured by the Seven-Day Physical Activity Recall

Physical Activity Category	Median (IQ Range)
Total[a]	288 (192, 460)
Moderate	248 (142, 381)
Vigorous	26 (0, 82)
Light	6029 (5560, 6282)

[a] Total activity is the sum of moderate and vigorous intensity activity.

Note. Values represent median and interquartile (IQ) range.

Effect of an Exercise Consultation on Physical Activity

Stage of Exercise Behaviour Change

There were no experimental or control patients in the precontemplation or contemplation stages at baseline, six and 12 months. Table 10 shows the proportion of patients in the preparation, action and maintenance stages of exercise behaviour change at each time period by group. Subjects in the action and maintenance stages are considered to be regularly physically active. Therefore, the action and maintenance stages were combined to produce an action/maintenance category. Chi square tests and Fisher's exact tests were used to compare the number of patients in the preparation stage versus the action/maintenance category between and within the experimental and control groups.

Table 10 shows that 88% of the experimental group and 77% of the control group were in action/maintenance at baseline, chi squared analysis reveals that this difference was not significant (χ^2=1.6, *df*=1, p=0.2). Thus, the majority of patients in both groups were regularly active at baseline. At six months, 84% of the experimental group and 69% of the control group were in the action/maintenance stage, this difference was not significant (χ^2=2.2, *df*=1, p=0.1). At 12 months, a greater proportion of the experimental group (85%) were in the action/maintenance category compared to the control group (67%), chi square analysis shows that this difference was not statistically significant (χ^2=3.0, *df*=1, p=0.08).

Chi square analysis revealed that the number of control patients who were in the action/maintenance category did not significantly change at six (χ^2=0.6, *df*=1, p=0.4) and 12 months (χ^2=0.9, *df*=1, p=0.3) compared with baseline. Similarly, Fisher's exact tests found that the number of experimental patients in the action/maintenance category was similar at six months (p=0.73) and 12 months (p=0.73) compared with baseline. Thus, the number of regularly active patients in the experimental and control group did not significantly change at six and 12 months compared to baseline.

Table 10 Proportion of Patients in the Preparation, Action and Maintenance Stages of Change at Baseline, Six and 12 Months by Group

Stage of Change	Experimental Group	Control Group
Baseline		
Preparation	11% (4/35)	23% (8/35)
Action	51% (18/35)	40% (14/35)
Maintenance	37% (13/35)	37% (13/35)
Six months		
Preparation	16% (5/32)	31 (10/32)
Action	19% (6/32)	7% (2/32)
Maintenance	65% (21/32)	62% (20/32)
12 months		
Preparation	15% (5/33)	33% (11/33)
Action	6% (2/33)	3% (1/33)
Maintenance	79% (26/33)	64% (21/33)

Note. Values in parentheses represent number of patients.

Change in Stage of Exercise Behaviour from Baseline to Follow-up

The change in stage of exercise behaviour from baseline to six and 12 months in each group was determined by the following method. Patients were categorised into four groups: progression (progressed from preparation to action or maintenance), regression (regressed from action or maintenance to preparation), stable active (remained in action or maintenance) and stable inactive (remained in preparation). Table 11 shows the number of patients in each category at six and 12 months by group.

Table 11 Change in Stage of Exercise Behaviour from Baseline to Follow-up by Group

Category	0 – 6M		0 – 12M		6-12M	
	Experimental n = 32	Control n = 32	Experimental n = 33	Control n = 33	Experimental n = 32	Control n = 32
Progression	1	3	2	4	3	4
Regression	3	5	3	7	2	6
Stable Active	26	19	26	18	25	17
Stable Inactive	2	5	2	4	2	5

Note. Values represent number of patients.

Table 11 shows that few patients in both groups were in the progression, regression and stable inactive categories. Therefore, the progression and stable active categories were combined, as these patients were regularly physically active, in order to conduct chi-square analysis. Similarly, the stable inactive and regression categories were combined, as these patients were not regularly active. Figure 10 shows the proportion of patients in each group who were regularly physically active at follow-up compared to baseline. From baseline to six months, 69% of the control group and 84% of the experimental group had progressed to action/maintenance or remained in action/maintenance (Figure 10), thus were regularly active (χ^2=2.2, *df*=1, p=0.1). From baseline to 12 months, a greater number of patients in the experimental group compared to the control group had remained regularly active or become regularly active, see Figure 10 (χ^2=3.0, *df*=1, p=0.08). From six to 12 months, a significantly greater proportion of experimental patients had remained in action/maintenance or progressed to action/maintenance than controls, see Figure 10 (χ^2=4.3, *df*=1, p=0.04).

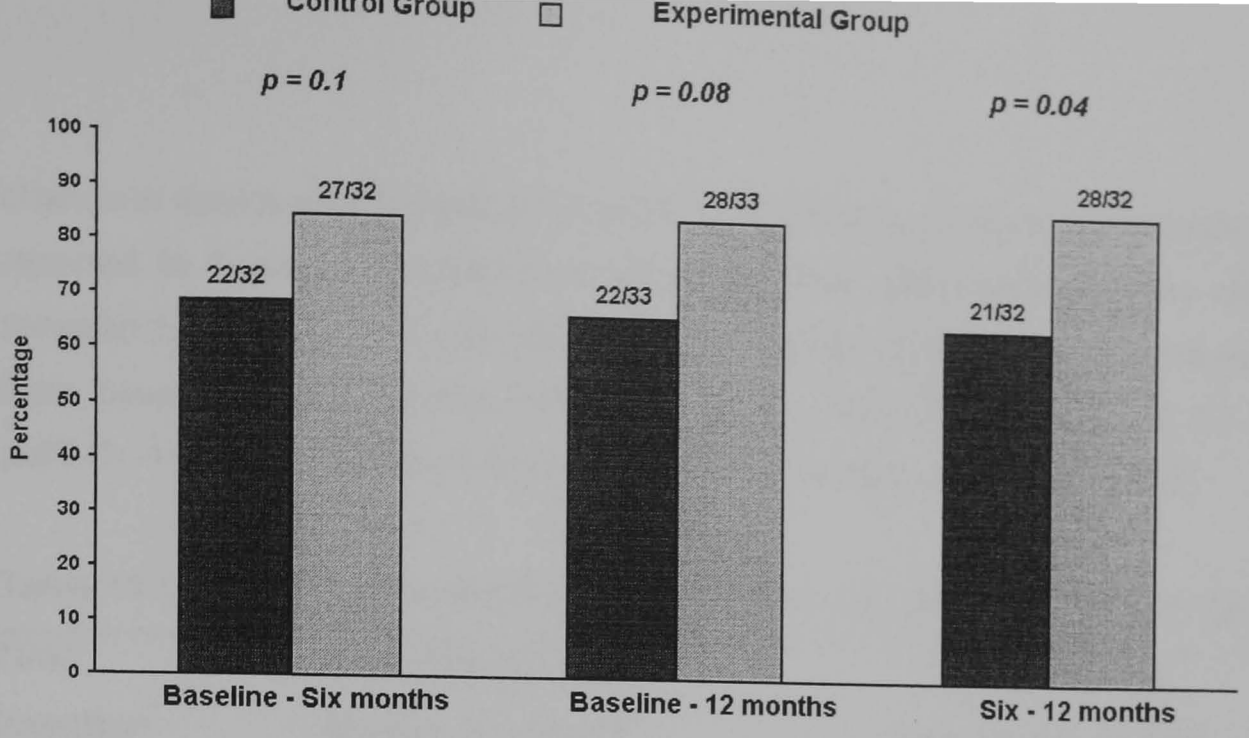


Figure 10 Change in stage of exercise behaviour from baseline to six and 12 months by group. Bars represent the proportion of patients remaining in action/maintenance stage or progressing from preparation to action/maintenance.

Computer Science and Applications (CSA) Accelerometer

Two control patients refused to wear the accelerometer at 12 months. In the experimental group, one accelerometer malfunctioned and one patient refused to wear the accelerometer at six months. At 12 months, one accelerometer malfunctioned in the experimental group.

CSA accelerometer data were not normally distributed, thus non-parametric tests were used to analyse the data. Table 12 reports medians and interquartile ranges for weekly CSA accelerometer counts at baseline, six and 12 months in the experimental and control group. Figure 11 and Table 13 demonstrates the median change in weekly CSA accelerometer counts from baseline to follow-up in the two groups.

A Mann Whitney test revealed that CSA accelerometer counts per week at baseline were similar between the experimental and control group (98%CI –856002, 889959). In the experimental group, accelerometer counts increased by 1.9% (76431/3999309) from baseline to six months, 0.8% (33619/4257833) from six to 12 months, and 2.2% (86575/3999309) from baseline to 12 months. However, Wilcoxon signed rank tests revealed that the change in weekly accelerometer counts was not significant from baseline to follow-up in the experimental group (Table 13). In the control group, accelerometer counts decreased by 5.2% (-215141/4105603) from baseline to six months, 3% (-109691/3621195) from six to 12 months, and 8% (-328116/4105603) from baseline to 12 months. Similar to the experimental group, there was no significant

change in weekly accelerometer counts from baseline to follow-up in the control group, reported in Table 13. Furthermore, Mann Whitney tests found that the difference between the groups for the change in weekly accelerometer counts was not significant from baseline to six months (98%CI -1143720, 607430), baseline to 12 months (98%CI -1131128, 366473) and six to 12 months (98%CI -861968, 647527).

Table 12 CSA Accelerometer Counts at Baseline, Six and 12 Months by Group

Time (months)	Experimental group Median (IQ Range)	Control group Median (IQ Range)
0	3999309 (2652408, 5105019)	4105603 (3089611, 4779881)
6	4257833 (3074216, 5556053)	3621195 (3008800, 5063930)
12	4221539 (2640305, 5600396)	3674479 (2746242, 5035637)

Note. The values represent median and interquartile (IQ) range.

Experimental group: n=35 at baseline, n=33 at six months, n=31 at 12 months. Control group: n=35 at baseline, n=31 at six months, n=31 at 12 months.

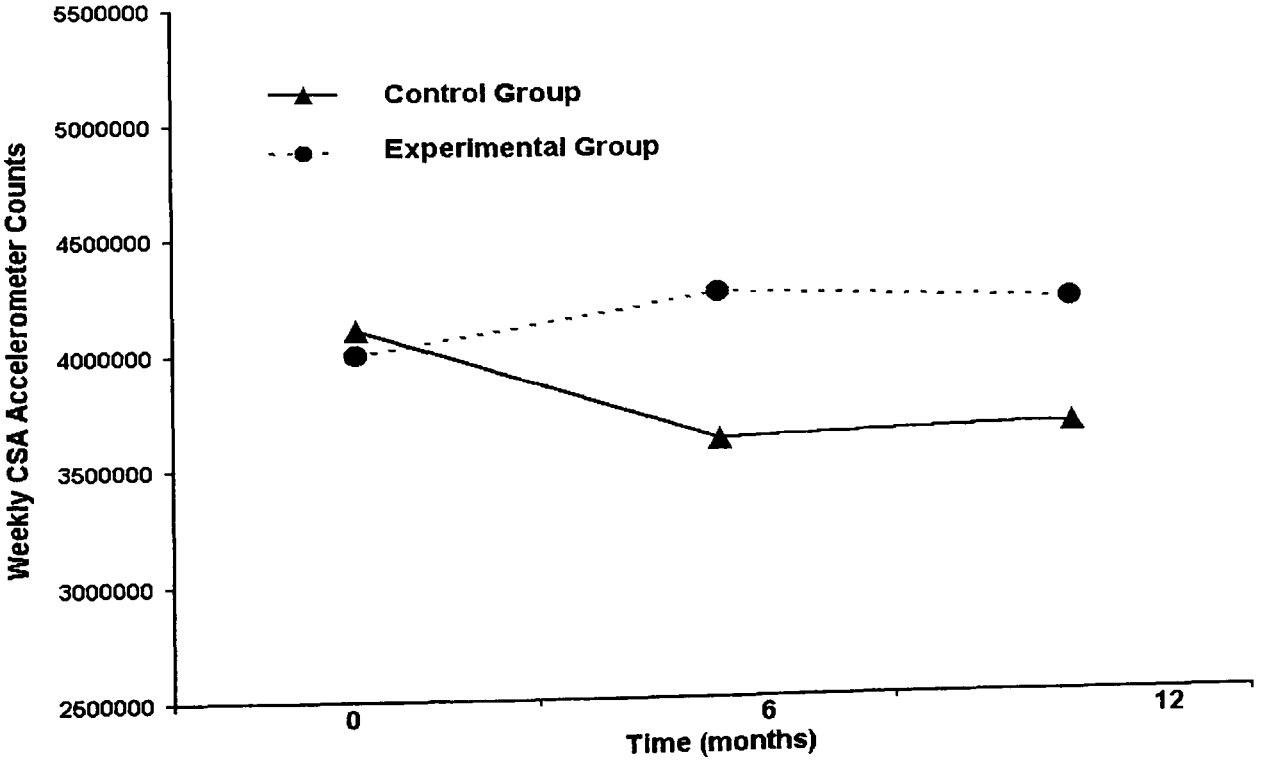


Figure 11 Median values for CSA accelerometer counts/week at baseline, six and 12 months in the experimental and control groups.

Table 13 Change in CSA Accelerometer Counts from Baseline to Follow-Up

Time Period	Experimental group	Control group
Baseline – 6 months	(-705643, 860599)	(-680366, 349212)
Baseline – 12 months	(-381927, 504719)	(-906564, 268168)
Six months – 12 months	(-737184, 671240)	(-514984, 303570)

Note. Values represent 98% confidence intervals for the change

Stanford Seven Day Physical Activity Recall

The Stanford Seven Day Physical Activity Recall measures minutes of sleep, and light, moderate, hard, very hard and total physical activity over the previous week. Total activity is the sum of moderate, hard and very hard intensity physical activity.

Physical activity data was not collected at six months for one patient in the control group. Physical activity data was not normally distributed, therefore non-parametric tests were used to detect between and within group differences for each physical activity variable. Table 14 reports median values for total physical activity, vigorous, moderate and light intensity activity, and sleep in minutes/week at baseline, six and 12 months by group.

Table 14 Physical Activity (mins/week) by Group at Baseline and Follow-Up

PA[a] (mins/week)	Baseline	6 months	12 months
Total[b]			
Experimental	300 (195, 470)	330 (188, 664)	360 (208, 590)
Control	275 (175, 460)	235 (110, 369)	205 (68, 310)
Vigorous			
Experimental	56 (5, 100)	73.5 (16, 250)	65 (0, 178)
Control	10 (0, 40)	0 (0, 44)	0 (0, 0)
Moderate			
Experimental	250 (115, 382)	235 (120, 489)	215 (98, 371)
Control	240 (145, 375)	222 (110, 311)	190 (68, 282)
Light			
Experimental	6120 (5615, 6460)	6028 (5629, 6402)	5940 (5535, 6478)
Control	5885 (5584, 6240)	6000 (5671, 6390)	6082 (5875, 6410)

Sleep			
Experimental	3645 (3315, 3870)	3660 (3454, 3870)	3720 (3383, 4095)
Control	3825 (3645, 4230)	3720 (3428, 4035)	3750 (3473, 3968)

[a] PA = Physical Activity [b] Total = moderate + vigorous intensity PA.

Note. Values represent median and interquartile (IQ) range.

Experimental group: n=35 baseline, n=32 at six months, n=33 at 12 months. Control group: n=35 baseline, n=32 at six months, n=33 at 12 months.

Total Physical Activity

Table 14 shows that total physical activity at baseline was 300 minutes/week in the experimental group and 275 minutes/week in the control group, there was no significant between group difference (98%CI –120, 100). The change in total activity (minutes/week) from baseline to six and 12 months in the experimental and control group is shown in Figure 12 and Table 15. Wilcoxon signed rank tests found that there was no significant change in total activity from baseline to six and 12 months in the experimental group, thus total activity was maintained over the study period in this group (Figure 12, Table 15). In the control group, total activity did not significantly change from baseline to six months. In contrast, total activity significantly decreased by 115 minutes/week from baseline to 12 months and 63 minutes/week from 6 to 12 months in the control group. In addition, the between group difference for the change in total activity was borderline significant from baseline to 6 months (108 mins/week; 98%CI -272, 25), was significant from baseline to 12 months (130 mins/week; 98%CI –295, -20) and was not significant from 6 to 12 months (75 mins/week; 98%CI –178, 40).

Figures 11 and 12 show that the change in CSA accelerometer counts/week and total physical activity (minutes/week) from baseline to six months appear to track each other in both groups. In contrast, from six to 12 months Figures 11 and 12 illustrate a further increase in total physical activity (minutes/week) in the experimental group and a further decrease in the control group, whereas CSA accelerometer counts/week showed no further change from six to 12 months in either group. Thus, the change in total activity and weekly accelerometer counts do not track each other from six to 12 months in either group.

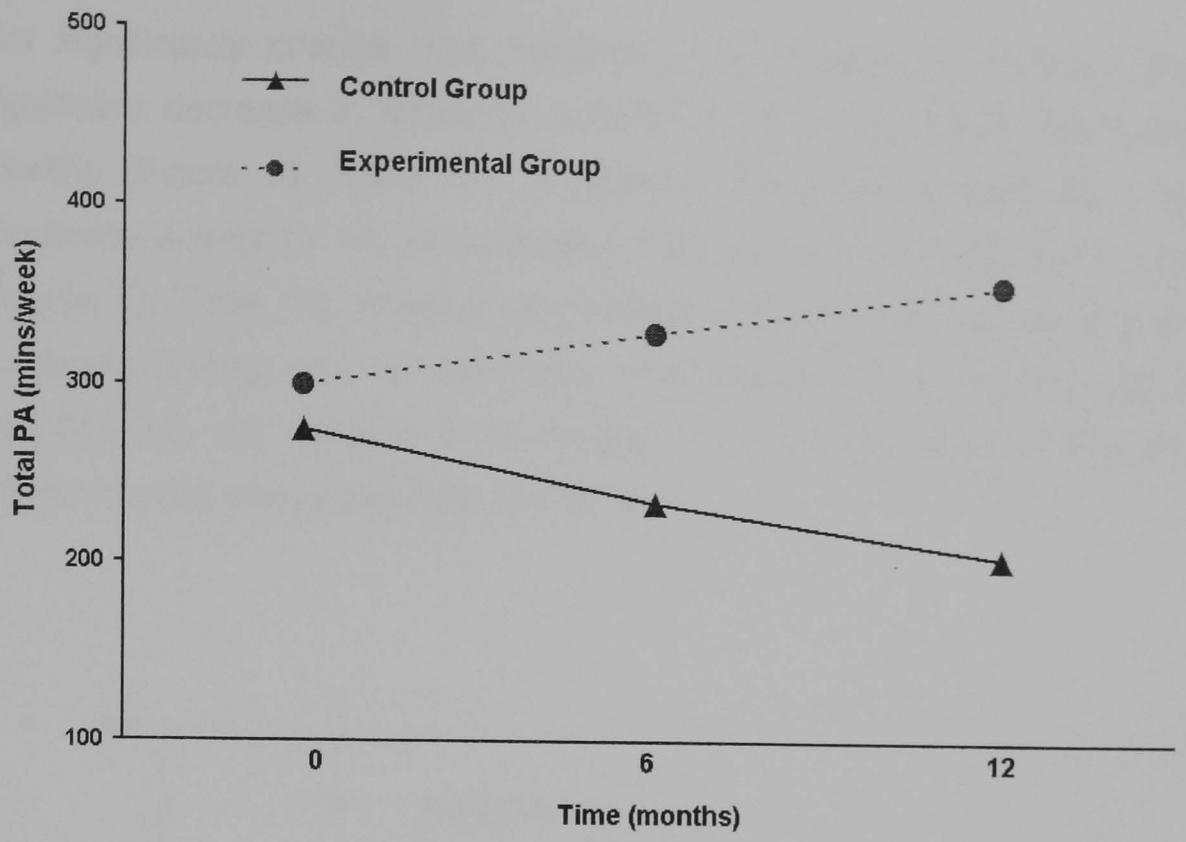


Figure 12 Total physical activity (PA) in minutes/week at baseline, six and 12 months in the experimental and control group

Table 15 Median Change in Total Physical Activity (mins/week) from Baseline to Six and 12 Months by Group

Total PA[a] (mins/week)	Experimental Group	Control Group
Baseline – 6 months	47 (-43, 191)	-55 (-178, 45)
Baseline – 12 months	23 (-63, 154)	*-115 (-228, -28)*
6 months – 12 months	3 (-128, 95)	*-63 (-126, -5)*

[a] PA = Physical Activity. *Note*. Values represent median change and 98% confidence interval for the change. Significant changes are represented in *italics*.

Moderate Intensity Physical Activity

Moderate intensity activity at baseline was similar (98%CI –85, 110) between the experimental group (250mins/week) and the control group (240 mins/week), as shown in Table 10. The change in moderate intensity activity (minutes/week) from baseline to follow-up in the experimental and control group is displayed in Figure 13 and Table 16. In the experimental group, there was no significant change in moderate activity from baseline to six and 12 months, therefore moderate intensity activity was maintained over the study period (Figure 13, Table 16). In the control group, moderate activity did

not significantly change from baseline to six months. In contrast, there was a significant decrease in moderate activity by 83 minutes/week from baseline to 12 months (Figure 13, Table 16). In addition, there was a trend for a decrease in moderate activity by 48 minutes/week from six to 12 months in the control group (Figure 13, Table 16). However, the difference between the groups for the change in moderate activity was not significant from baseline to six months (60 mins/week; 98%CI –190, 39), baseline to 12 months (75 mins/week; 98%CI –198, 30) and six to 12 months (22 mins/week; 98%CI –99, 68).

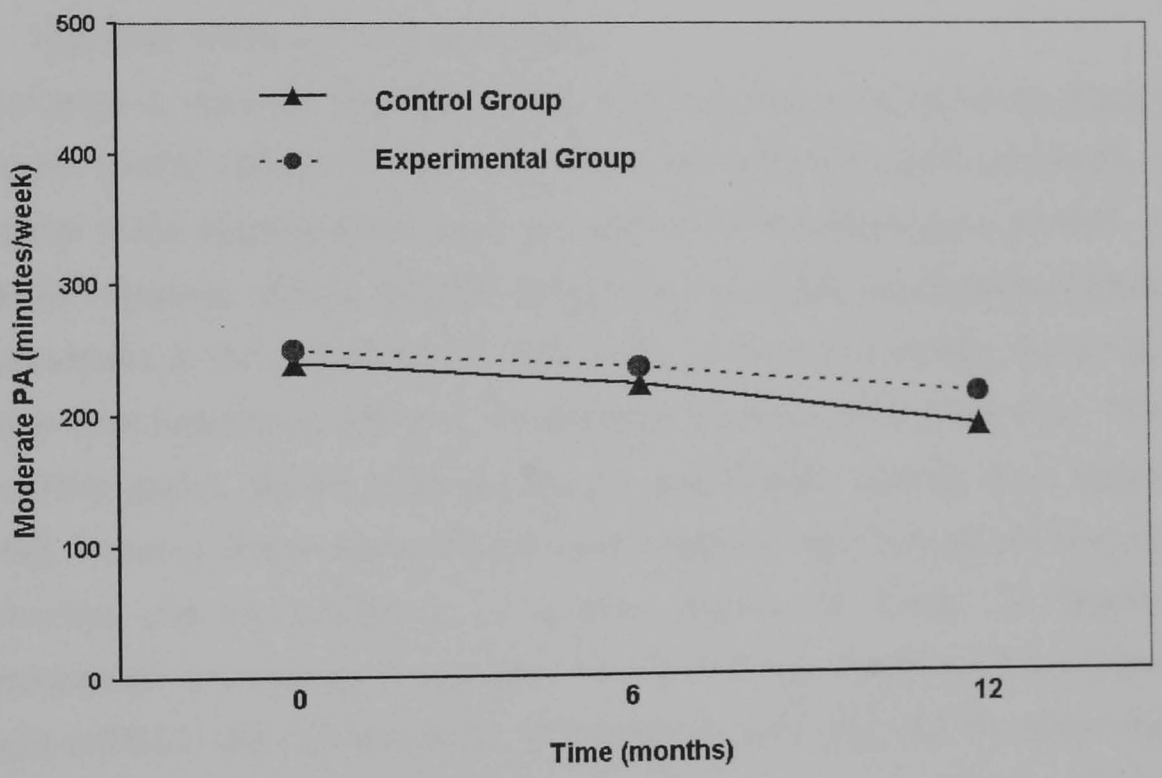


Figure 13 Moderate intensity physical activity (PA) in minutes/week at baseline, six and 12 months in the experimental and control group

Table 16 Median Change in Moderate Physical Activity (mins/week) from Baseline to Six and 12 Months by Group

Moderate PA[a] (mins/week)	Experimental Group	Control Group
Baseline – 6 months	35 (-60, 113)	-47 (-149, 49)
Baseline – 12 months	-10 (-90, 83)	-83 (-175, -14)
6 months – 12 months	-30 (-120, 39)	-48 (-92, 2)

[a] PA = Physical Activity. *Note*. Values are median change and 98% confidence interval for the change. Significant changes are represented in red text.

Vigorous Intensity Physical Activity

The change in vigorous intensity activity from baseline to follow-up by group is shown in Figure 14 and Table 17. There was a trend for baseline vigorous intensity activity to be higher in the experimental group compared to the control group (98%CI –61, 0). At baseline, vigorous activity was 56 minutes/week in the experimental group and 10 minutes/week in the control group, see Table 14. Vigorous activity did not significantly change from baseline to follow-up in the experimental group (Figure 14, Table 17). In the control group, vigorous activity did not significantly change from baseline to six months, however there was a trend for a decrease in vigorous activity from baseline to 12 months and six months to 12 months (Figure 14, Table 17). Between group differences for the change in vigorous activity was non-significant from baseline to six months (98%CI –90, 20) and six to 12 months (98%CI –90, 30). However, there was a bordeline significant difference between the groups for the change in vigorous activity from baseline to 12 months (45 mins/week; 98%CI -110, 0).

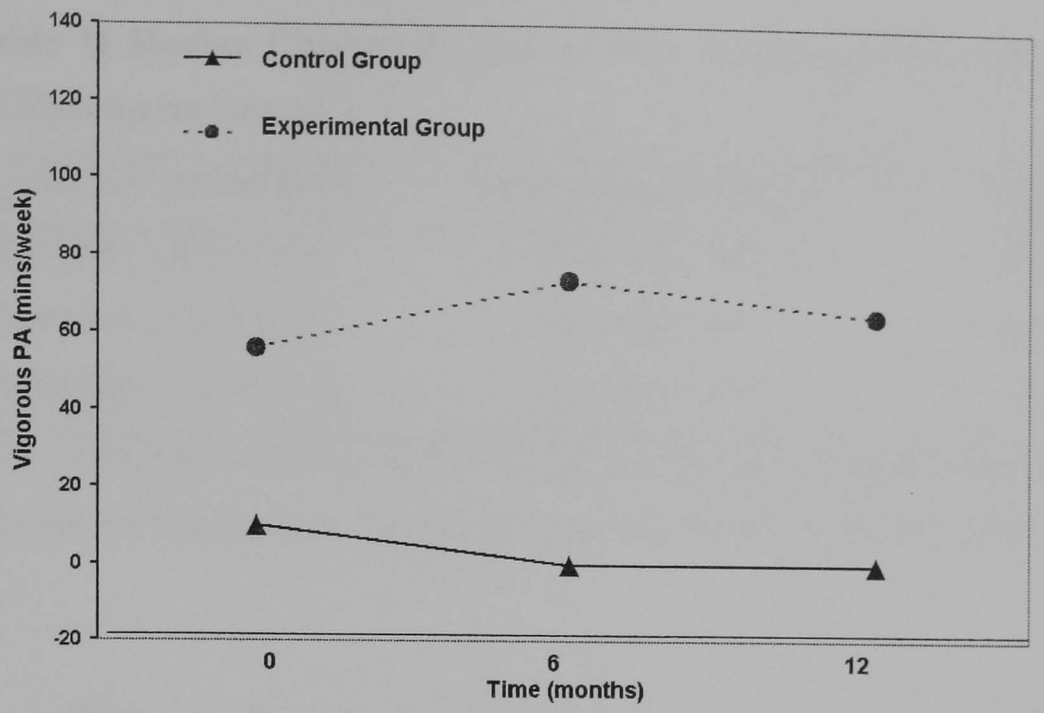


Figure 14 Vigorous physical activity (PA) in mins/week at baseline, six and 12 months by group

Table 17 Median Change in Vigorous Activity (mins/week) from Baseline to Six and 12 Months by Group

Vigorous PA[a] (mins/week)	Experimental Group	Control Group
Baseline – 6 months	31 (-21, 117)	0 (-20, 22)
Baseline – 12 months	35 (-15, 112)	-8 (-40, 0)
6 months – 12 months	0 (-68, 65)	-5 (-40, 0)

[a] PA = Physical Activity. *Note*. Values represent median change and 98% confidence interval.

Light Intensity Physical Activity

Light intensity activity per week at baseline was not significantly different between the experimental and control group (98%CI –485, 65). There was no significant change in light intensity physical activity from baseline to six and 12 months follow-up in the experimental group (Figure 15, Table 18). In the control group, light intensity activity did not significantly change from baseline to six months, and six to 12 months (Table 18, Figure 15). In contrast, light activity significantly increased by 205 minutes/week from baseline to 12 months in the control group. Furthermore, there was a significant difference between the groups for the change in light activity from baseline to 12 months (290 minutes/week; 98%CI 50, 550).

Table 18 Median Change in Light Activity (mins/week) from Baseline to Six and 12 Months by Group

Light PA[a] (mins/week)	Experimental Group	Control Group
Baseline – 6 months	-20 (-249, 185)	95 (-95, 347)
Baseline – 12 months	-70 (-308, 104)	*205 (48, 388)*
6 months – 12 months	-21 (-185, 113)	71 (-90, 248)

[a] PA = Physical Activity. *Note*. Values represent median change and 98% confidence interval for the change. Significant changes are represented *italics*.

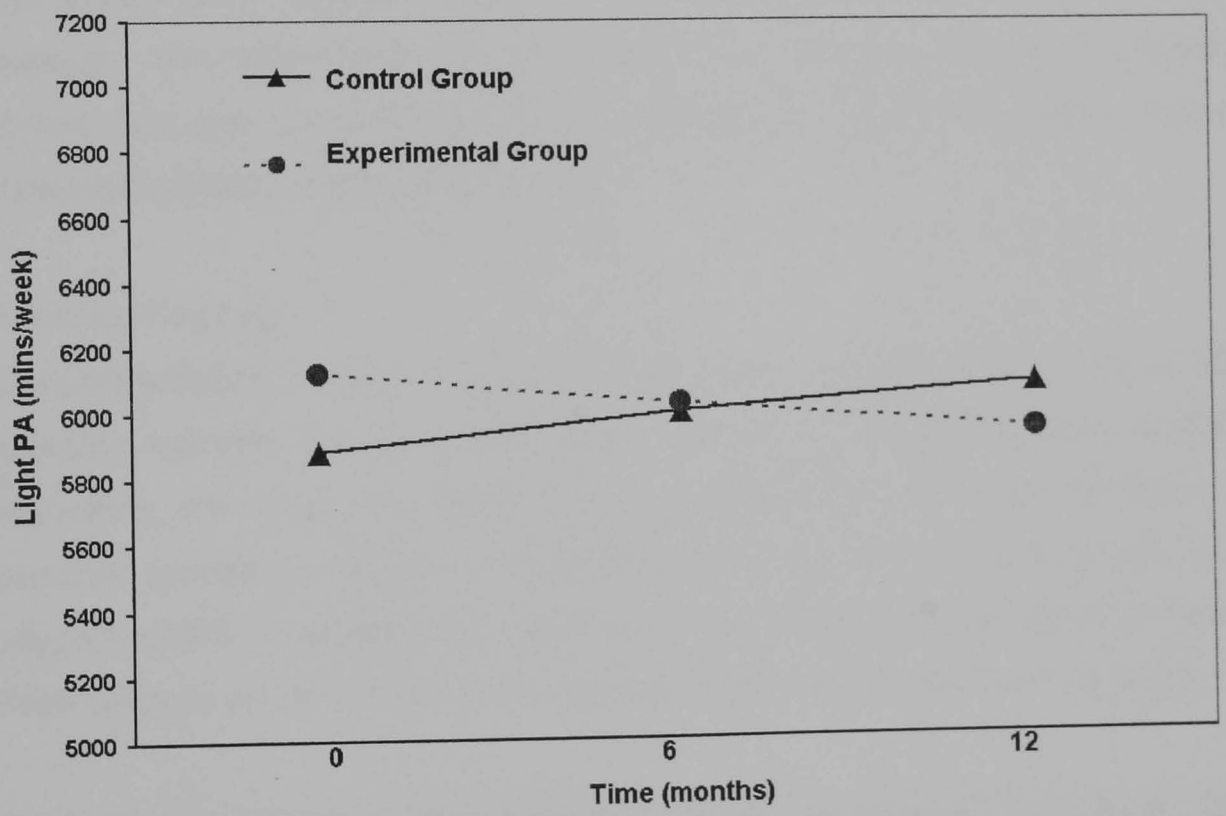


Figure 15 Light intensity physical activity (PA) in minutes/week week at baseline, six and 12 months in the experimental and control groups

Sleep

Baseline minutes of sleep per week was higher in the control group compared to the experimental group, this difference was not statistically significant (98%CI –30, 450). Sleep did not significantly change from baseline to follow-up in the either group (Table 19).

Table 19 Median Change in Sleep (mins/week) from Baseline to Six and 12 Months by Group

Sleep (minutes/week)	Experimental Group	Control Group
Baseline – 6 months	-44 (-233, 158)	-45 (-210, 75)
Baseline – 12 months	53 (-105, 248)	-90 (-233, 53)
6 months – 12 months	60 (-38, 278)	0 (-143, 113)

Note. Values represent median change and 98% confidence interval.

Effect of an Exercise Consultation on Cardiorespiratory Fitness

Cardiorespiratory fitness, specifically exercise capacity and submaximal endurance capacity, was determined by cardiopulmonary exercise testing. Cardiorespiratory fitness data was normally distributed, therefore parametric tests were used to detect differences between and within groups.

Exercise Capacity

Cardiorespiratory variables measured at peak exercise were used to determine exercise capacity. In the control group, values for oxygen uptake, ventilation and respiratory exchange ratio (RER) were unavailable for one patient at baseline as the portable metabolic system malfunctioned. In the experimental group, values for oxygen uptake, ventilation and RER were unavailable for one patient at baseline and three patients at six months as the portable metabolic system malfunctioned.

Table 20 reports mean values for exercise capacity variables at baseline, six and 12 months in each group. The mean change in exercise capacity parameters from baseline to follow-up by group is demonstrated in Table 21.

Table 20 Exercise Capacity Variables by Group at Baseline, Six and 12 Months

	Baseline		6 months		12 months	
	n	Mean ± SD	n	Mean ± SD	n	Mean ± SD
Peak VO_2 ml/min						
Experimental	34	2287 ± 579	29	2129 ± 561	33	2118 ± 584
Control	34	1950 ± 643	33	1817 ± 641	33	1774 ± 690
Peak VO_2 ml/kg/min						
Experimental	34	26.9 ± 6.6	28	26.1 ± 6.9	33	25.1 ± 6.2
Control	34	24.4 ± 6.5	33	22.5 ± 6.2	32	22.0 ± 6.1
Exercise Time (secs)						
Experimental	31	513 ± 155	27	508 ± 166	29	532 ± 179
Control	33	470 ± 226	32	481 ± 281	32	484 ± 264
Peak VE						
Experimental	34	69.9 ± 17.8	29	68.6 ± 18.7	33	69.9 ± 20.9
Control	34	61.1 ± 20.0	33	57.9 ± 18.9	33	56.6 ± 19.3
RER						
Experimental	34	0.94 ± 0.09	29	1.02 ± 0.12	33	1.04 ± 0.10
Control	34	0.93 ± 0.08	33	0.97 ± 0.11	33	0.98 ± 0.12
Peak HR						
Experimental	34	126.6±22.8	28	128.8±26.6	33	126.2±24.8
Control	34	121.8±20.2	33	116.5±22.4	32	114.2±20.1
Peak VO_2/HR						
Experimental	34	17.9±3.5	28	16.9±3.7	33	16.9±3.7
Control	34	16.1±4.8	33	15.6±4.7	32	15.4±5.3

VO_2 = oxygen uptake; VE = ventilation; RER = respiratory exchange ratio, HR = heart rate; VO_2/HR = oxygen pulse.

Table 21 Mean Change in Exercise Capacity Variables from Baseline to Six and 12 Months by Group

	0 – 6 months	0 – 12 months	6 - 12 months
Peak VO_2 ml/min			
Experimental	-82 (-169, 5)	*-136 (-267, -5)*	- 57 (-180, 65)
Control	-121 (-244, 2)	*-147 (-270, -23)*	-23 (-121, 75)
Peak VO_2 ml/kg/min			
Experimental	-1.0 (-2.2, 0.2)	*-1.8 (-3.2, -0.3)*	-1.1 (-2.5, 0.3)
Control	*-1.6 (-3.0, -0.1)*	*-2.3 (-3.8, -0.8)*	-0.6 (-2.0, 0.7)
Exercise Time (secs)			
Experimental	-1.2 (-52, 50)	16 (-42, 75)	22 (-25, 70)
Control	24 (-44, 91)	25 (-44, 94)	-5 (-54, 44)
Peak VE (l/min)			
Experimental	0.9 (-3.1, 4.9)	1.4 (-2.8, 5.7)	-0.1 (-4.0, 3.8)
Control	-1.9 (-6.7, 3.0)	-1.7 (-6.6, 3.2)	-0.05 (-3.2, 3.1)
RER			
Experimental	*0.07 (0.03, 0.11)*	*0.09 (0.06, 0.13)*	0.02 (-0.01, 0.06)
Control	*0.05 (0.01, 0.10)*	*0.07 (0.03, 0.11)*	0.01 (-0.03, 0.06)
Peak HR			
Experimental	0.5 (-4.5, 5.4)	-0.3 (-5.0, 4.5)	-1.6 (-6.3, 3.1)
Control	-4.4 (-9.6, 0.82)	*-5.6 (-10.6, -0.7)*	-1.8 (-4.8, 1.3)
Peak VO_2/HR			
Experimental	-0.6 (-1.5, 0.3)	-1.0 (-1.9, 0)	-0.3 (-1.4, 0.9)
Control	-0.5 (-1.5, 0.5)	-0.5 (-1.3, 0.3)	0 (-0.9, 1.0)

Note. Values represent median change and 98% confidence interval for the change. VO_2 = oxygen uptake; VE = ventilation; RER = respiratory exchange ratio, HR = heart rate; VO_2/HR = oxygen pulse. Significant changes are represented in *italics*.

Baseline mean peak VO_2 (ml/min) was higher in the experimental group compared to the control group, this between group difference was not statistically significant (98%CI –691, 17), see Table 20. Peak VO_2 in ml/min decreased by 3.6% (82/2287) in the experimental group and 6.2% (121/1950) in the control group from baseline to six months, this difference was near significant (Table 21). Furthermore, there was a significant decrease in peak VO_2 ml/min by 5.9% (136/2287) in the experimental group

and 7.5% (147/1950) in the control group from baseline to 12 months (Table 21). In contrast, peak VO_2 ml/min did not significantly change from six to 12 months in the experimental group [2.7% (57/2129)] and the control group [1.3% (23/1817)], as displayed in Table 21. Furthermore, the difference between the groups for the change in peak VO_2 ml/min was not significant from baseline to six months (98%CI –185.3, 108) and 12 months (98%CI -186.6, 164.5), and six to 12 months (98%CI -118.8, 187.0). Thus, the decrease in peak VO_2 from baseline to follow-up was similar in the experimental and control group.

Table 20 shows that baseline peak VO_2 corrected for body weight was 26.9 (SD 6.6) ml/kg/min in the experimental group and 24.4 (SD 6.5) ml/kg/min in the control group, this difference was not significant (98%CI –6.24, 1.30). Peak VO_2 declined from baseline to six months by 1 ml/kg/min (3.7%) in the experimental group and 1.6 ml/kg/min (6.5%) in the control group, this decline was borderline significant in the experimental group and was significant in the control group (Table 21). From baseline to 12 months, exercise capacity significantly decreased by 1.8 ml/kg/min (6.7%) in the experimental group and 2.3 ml/kg/min (9.4%) in the control group (Table 21). From six to 12 months, peak VO_2 decreased by 1.1 ml/kg/min (4.2%) in the experimental group and 0.6 ml/kg/min (2.7%) in the control group. This decrease in peak VO_2 (ml/kg/min) from six to 12 months was borderline significant in the experimental group. Two sample t tests revealed that the decline in peak VO_2 ml/kg/min was similar in the experimental and control groups from baseline to six (98%CI –2.4, 1.2) and 12 months (98%CI –2.5, 1.5) and six to 12 months (98%CI -1.4, 2.4). Figure 16 illustrates the decline in peak VO_2 ml/kg/min from baseline to follow-up in the experimental and control groups.

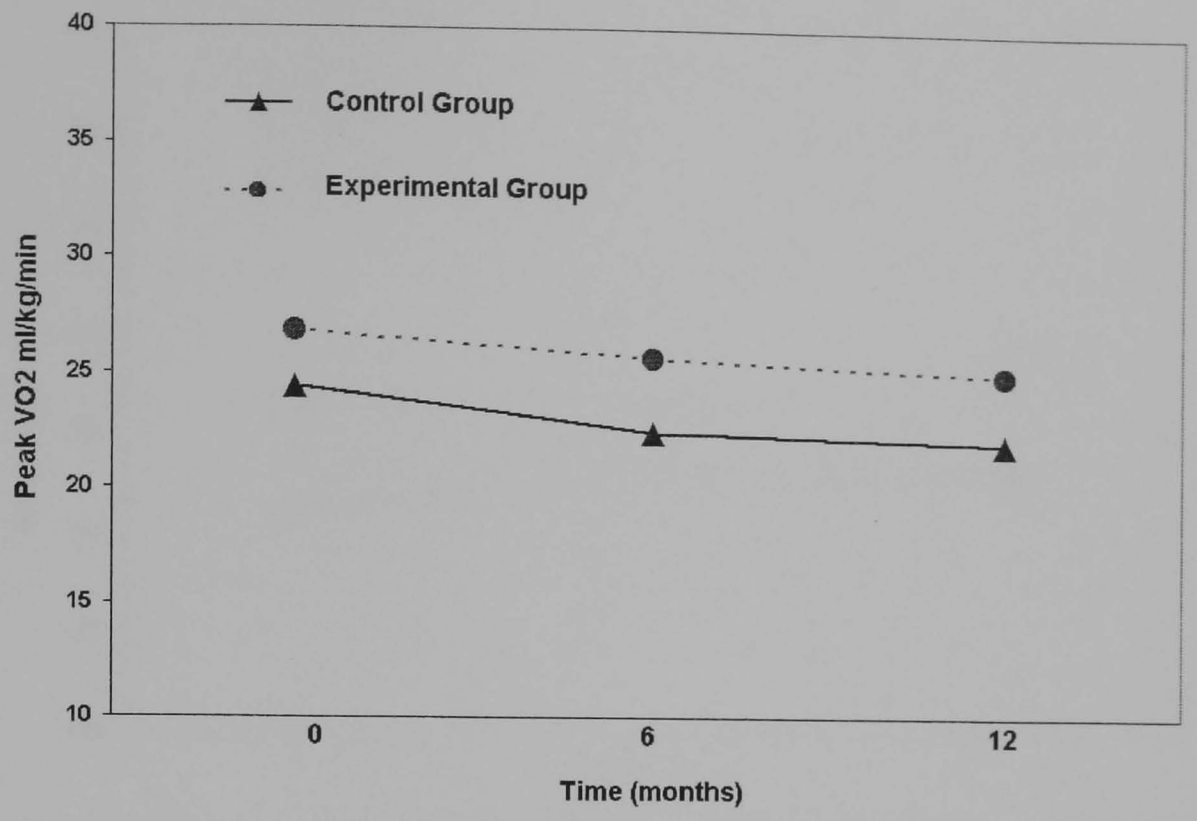


Figure 16 Mean peak VO_2 (ml/kg/min) at baseline, six and 12 months follow-up by group.

Exercise duration (seconds) at baseline was similar between the groups (98%CI -158.2, 72.7), shown in Table 20. Exercise duration did not significantly change from baseline to follow-up in the experimental group or the control group, as shown in Table 21 and Figure 17. Furthermore, there was no significant difference between the groups for the change in exercise duration from baseline to six months (98%CI –58, 107), baseline to 12 months (98%CI –79, 96), and six to 12 months (98%CI –93, 39). Exercise duration at each time period by group is shown in Figure 17.

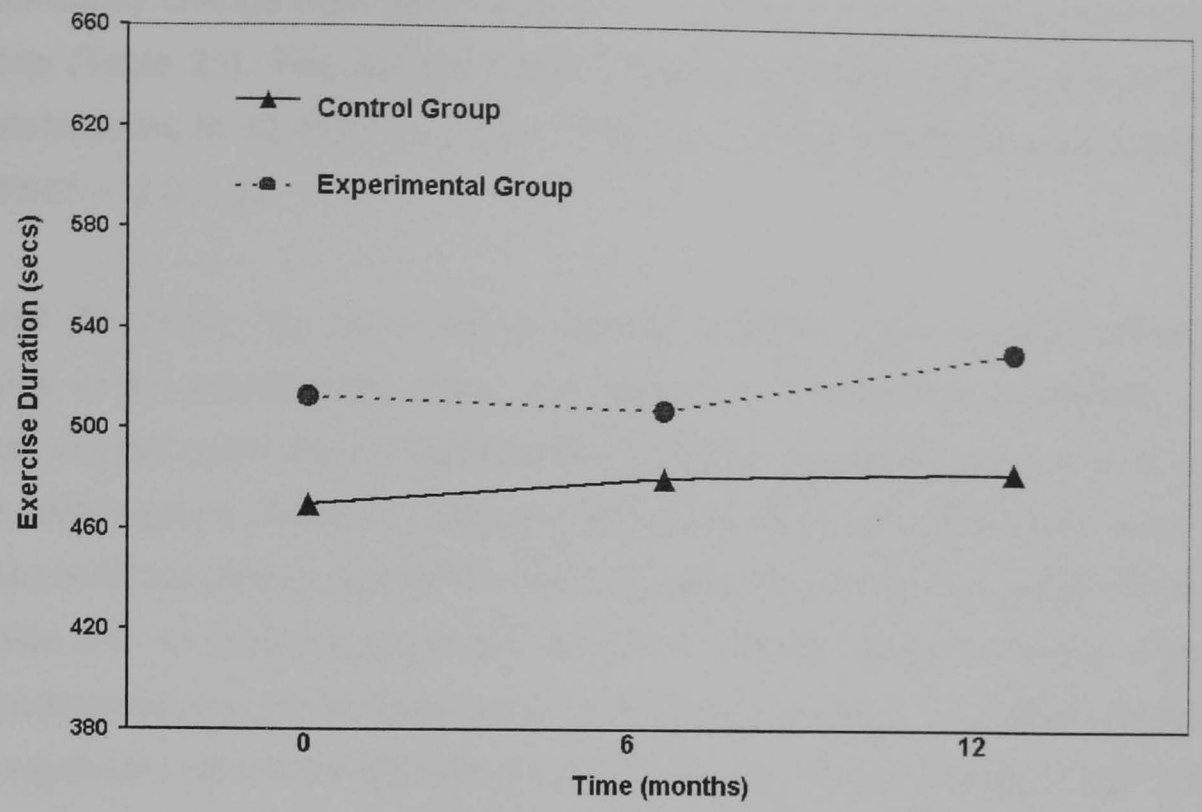


Figure 17 Mean exercise duration (secs) at baseline, six and 12 months follow-up by group.

Peak ventilation at baseline was higher in the experimental group compared to the control group (Table 20), the between group difference was not statistically significant (98%CI –19.8, 2.1). Table 21 shows that peak ventilation did not significantly change from baseline to six and 12 months follow-up in either group. Similarly, the between group difference for the change in peak ventilation was not significant from baseline to six months (98%CI –8.8, 3.4), baseline to 12 months (98%CI –9.4, 3.2) and six to 12 months (98%CI –4.8, 4.9).

As reported in Table 20, baseline respiratory exchange ratio (RER) at peak exercise was 0.93 in the control group and 0.94 in the experimental group, the between group difference was not significant (98%CI –0.06, 0.04). There were similar significant increases in peak RER from baseline to six and 12 months in both groups (Table 21). In contrast, RER did not significantly change from six to 12 months in either group.

Peak heart rate at baseline was 126.6 bpm (SD 22.8) in the experimental group and 121.8 bpm (SD 20.2) in the control group (98%CI –17.7, 8.1), see Table 20. In the control group, there was a decline in peak heart rate from baseline to six months by 4.4 bpm (98%CI -9.6, 0.82) and a significant decrease from baseline to 12 months by 5.6 bpm (98%CI -10.6, -0.7), as shown in Table 21. In contrast, peak heart rate did not

significantly change from baseline to six and 12 months follow-up in the experimental group (Table 21). Two sample t tests revealed a greater decline in peak heart rate from baseline to 12 months in the control group compared to the experimental group (98%CI –12.0, 1.3).

Table 20 shows that there was a trend for baseline peak VO_2/HR (ml/beat) to be higher in the experimental group compared to the control group (98%CI –4.3, 0.7). Peak oxygen pulse did not significantly change from baseline to six and 12 months in the control group (Table 21). Similarly, there was no significant change in peak oxygen pulse from baseline to six months and from six to 12 months in the experimental group (Table 21). In contrast, there was a trend for decline in peak oxygen pulse in the experimental group from baseline to 12 months. However, two sample t tests revealed no significant difference between the groups for the change in peak oxygen pulse from baseline to six (98%CI –1.2, 1.4), 12 months (98%CI -0.7, 1.6) and six to 12 months (98%CI –1.1, 1.8). Peak oxygen pulse at baseline, six and 12 months by group is illustrated in Figure 18.

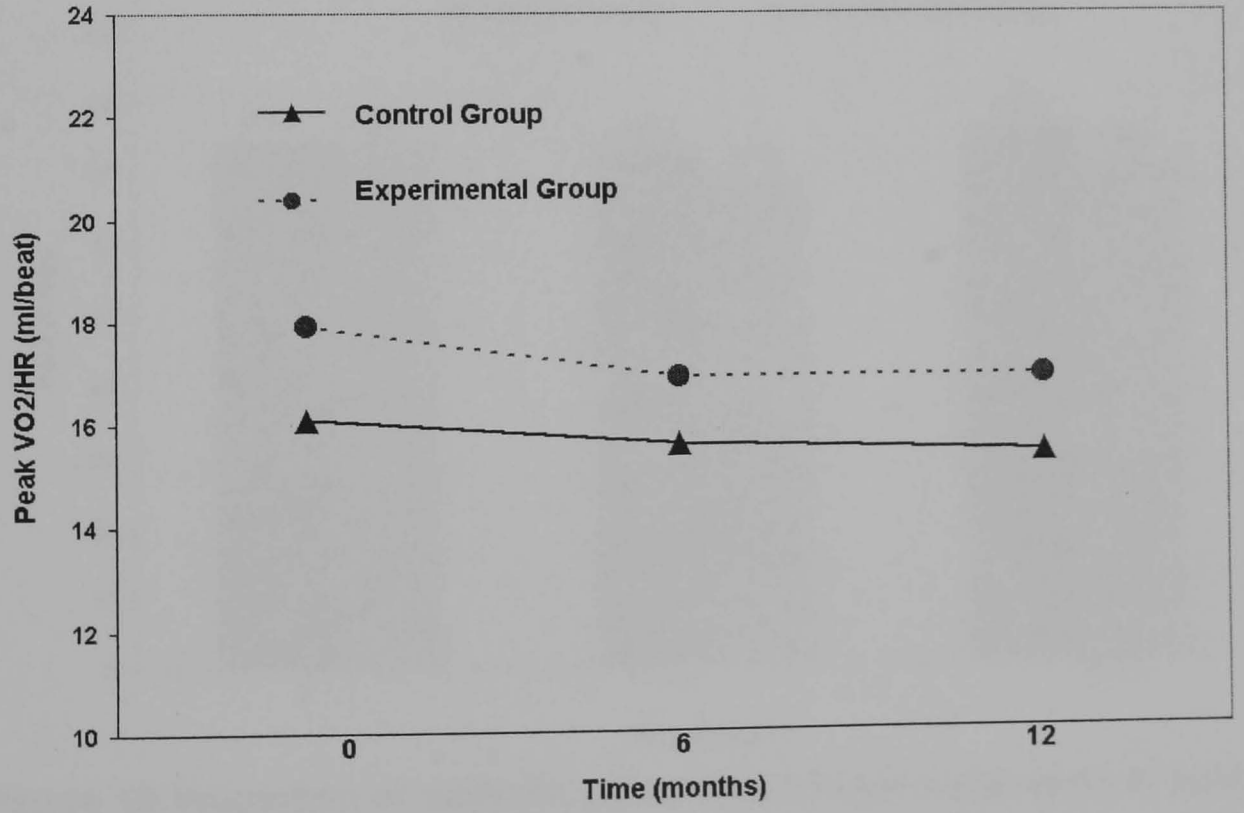


Figure 18 Peak oxygen pulse (VO_2/HR) in ml/beat at baseline, six and 12 months in the experimental and control groups

Proportion of Patients Taking Beta-blocker Medication

Beta-blocker medication can affect exercise capacity in terms of peak VO_2 and peak heart rate. Therefore, the change in beta-blocker medication from baseline to follow-up was examined to determine if alterations in medication could have influenced the outcome of the exercise capacity variables.

Figure 19 displays the proportion of patients taking beta-blocker medication at baseline, six and 12 months in each group. At baseline, 74.3% (26/35) of patients in the control group and the experimental group were taking beta-blocker medication. Figure 19 shows that the proportion of patients taking beta-blocker medication did not markedly change at six and 12 months in either group. Four control patients and two experimental patients had their dose of medication changed at six months compared to baseline. In addition, three controls and five experimental patients had their medication dose altered at 12 months compared to baseline.

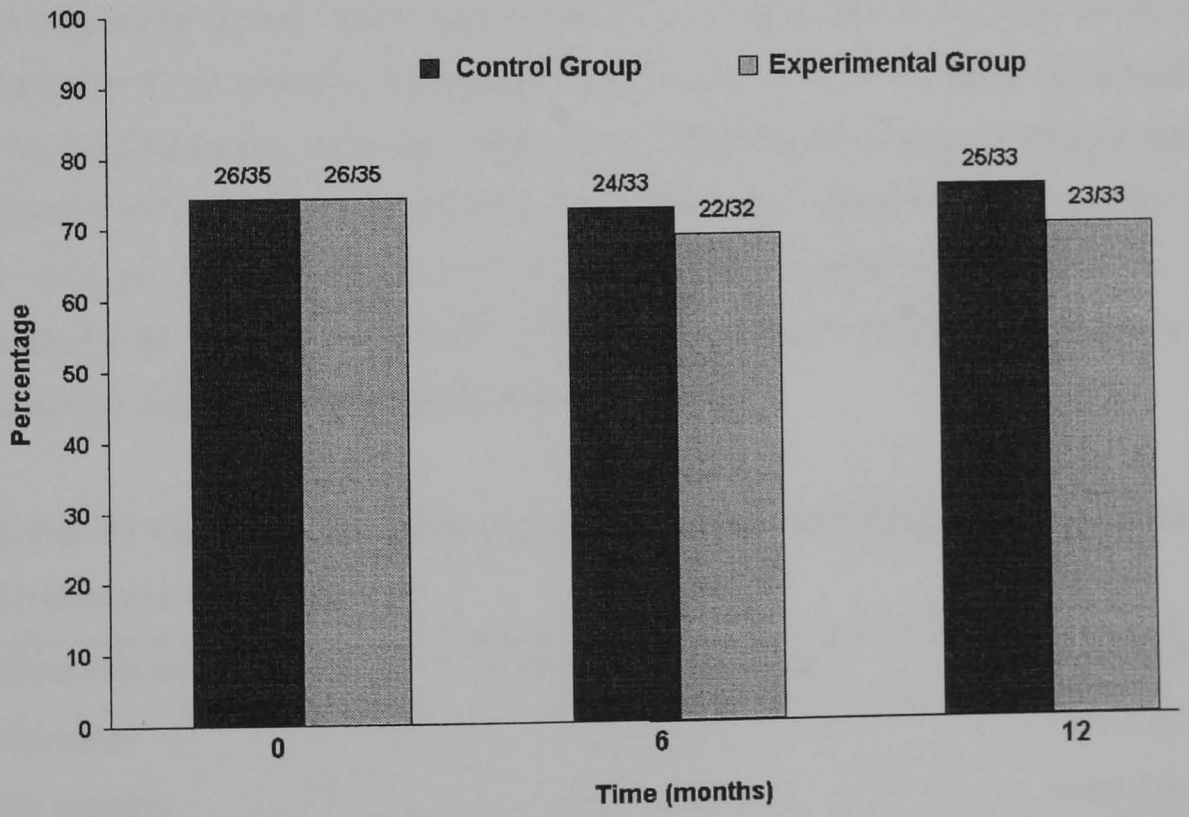


Figure 19 Proportion of patients taking beta-blocker medication at baseline, six and 12 months in both groups.

Submaximal Endurance Capacity

Submaximal endurance capacity was determined by estimating the oxygen uptake in ml/min at the lactate threshold (VO_2 LT). The lactate threshold could not be detected at each time period for five control patients and three experimental patients. In addition, data was unavailable for two experimental patients at 6 months as the portable metabolic system malfunctioned.

Table 22 reports mean values for oxygen uptake (ml/min) at the lactate threshold at baseline, six and 12 months in each group. The mean change in VO_2 LT from baseline to follow-up by group is demonstrated in Table 23.

Oxygen uptake (ml/min) at the lactate threshold (VO_2 LT) at baseline was similar between the experimental and control group (98%CI –257.0, 108.8). In the control group, there was a trend for a decrease in VO_2 LT by 52 ml/min from baseline to six months (Table 23). However, there was no further significant change in VO_2 LT from baseline to 12 months, and six to 12 months in the control group (Table 23). In the experimental group, there was a trend for an increase in VO_2 LT by 47ml/min from baseline to six months and a significant increase by 81 ml/min from baseline to 12 months (Table 23). Although, there was no significant change in VO_2 LT from six to 12 months in the experimental group. The difference between the groups for the change in VO_2 LT was significant from baseline to 6 months (98%CI –185.4, -12.6) and baseline to 12 months (98%CI –216.7, -34.6). Figure 20 demonstrates the VO_2 LT at baseline, six and 12 months by group.

Table 22 Oxygen Uptake at the Lactate Threshold (VO_2 LT) at Baseline, Six and 12 Months by Group

Time Period	Experimental Group	Control Group
Baseline	1251±273	1177±311
Six months	1274±267	1140±285
12 months	1332±278	1132±316

Note. Values represent mean ± SD.

Experimental group: n=30 baseline, n=27 at six months, n=30 at 12 months. Control group: n=29 baseline, n=28 at six months, n=29 at 12 months.

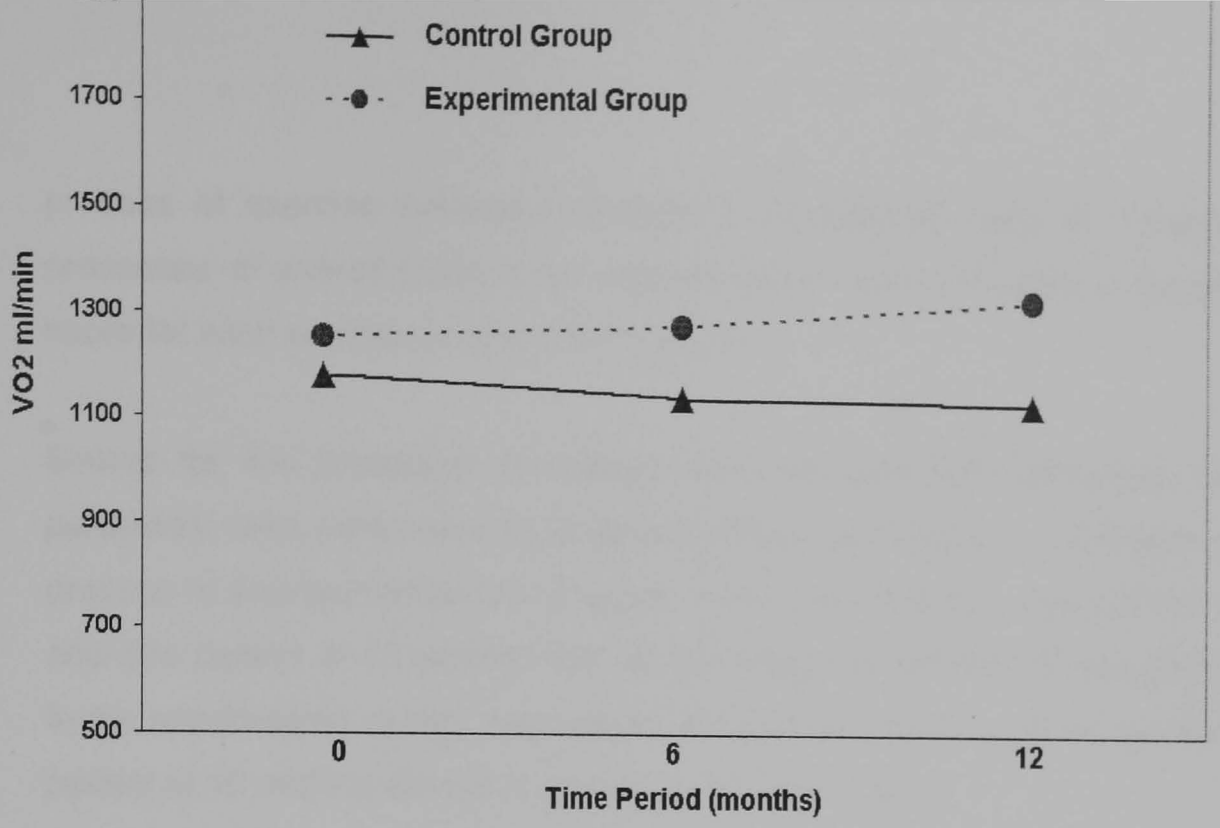


Figure 20 Oxygen uptake (ml/min) at lactate threshold in the experimental and control groups at baseline, six and 12 months

Table 23 Mean Change in Oxygen Uptake (ml/min) at the Lactate Threshold from Baseline to Six and 12 Months by Group

Time Period	Experimental Group	Control Group
Baseline - Six months	47 (-14, 109)	-52 (-116, 12)
Baseline - 12 months	*81 (22, 140)*	-45 (-118, 28)
Six months - 12 months	43 (-28, 113)	7 (-43, 57)

Note. Values represent median change and 98% confidence interval for the change. Significant changes are represented in *italics*.

Effect of an Exercise Consultation on the Processes of Exercise Behaviour Change

The processes of change are strategies that individuals use when changing their exercise behaviour.[160] The five experiential processes of exercise behaviour change are consciousness raising (CR), social liberation (SL), dramatic relief (DR), environmental re-evaluation (ER) and self re-evaluation (SR). The five behavioural processes are: helping relationships (HR), stimulus control (SC), counter conditioning (CC), self-liberation (SO), reinforcement management (RM). A description of each

process of exercise behaviour change is provided in Table 6, Chapter One. The processes of exercise behaviour change were measured using a questionnaire, the score for each process ranges from 4 to 20.

Scores for the processes of change were not normally distributed, therefore non-parametric tests were used to analyse between and within group differences for each process of exercise behaviour change. In the control group, one patient at six months and one patient at 12 months did not complete the process of change questionnaire. In the experimental group, one patient at baseline, two patients at six months and one patient at 12 months did not complete the questionnaire.

Experiential Processes

The median values for the use of the experiential processes at baseline and follow-up in the experimental and control groups. The use of the five experiential processes at baseline was similar between the experimental and control groups (Table 24). The median change in the use of each experiential process from baseline to six and 12 months follow-up is described in Table 25. There was a borderline decrease in consciousness raising from baseline to 12 months in both groups. Social liberation did not significantly change from baseline to follow-up in either group. Dramatic relief did not significantly change from baseline to six and 12 months in the experimental group, whereas there was a significant decrease in the use of this process from baseline to 12 months in the control group. Environmental re-evaluation did not significantly change from baseline to follow-up in the control group, whereas this process significantly decreased from baseline to 12 months in the experimental group. There was a trend for self re-evaluation to decrease from baseline to 12 months in both groups. In addition, there was a trend for a decrease in self re-evaluation from baseline to six months in the experimental group. However, the difference between the experimental and control group for the change experiential process use from baseline to six and 12 months follow-up was not significant.

Table 24 Experiential Processes by Group at Baseline, Six and 12 Months

Experiential Processes	Baseline	6 months	12 months
CR			
Experimental	11 (9, 13.2)	12 (8, 13)	10 (8, 12)
Control	11 (7, 14)	9.5 (7.2, 14.5)	10 (7.2, 12.8)
SL			
Experimental	15 (12, 17)	14.5 (11.8, 16)	14 (11, 17.8)
Control	13 (11, 16)	13.5 (11, 16)	13 (10, 15)
DR			
Experimental	7.5 (5, 11)	7 (5, 9.2)	6.5 (5, 8)
Control	8 (6, 12)	7 (5, 10)	6 (5, 8)
ER			
Experimental	12 (8, 13)	10 (8, 12)	9 (7, 10)
Control	10 (7, 13)	8.5 (6.2, 12)	8.5 (7, 11.8)
SR			
Experimental	15 (11.5, 16)	13 (10.8, 15.2)	12 (10.2, 15.8)
Control	13 (9, 16)	12 (9, 15)	11 (9, 14)

Note. Values represent median and interquartile range.

CR = consciousness raising, SL = social liberation, DR = dramatic relief, SR = self re-evaluation, ER = environmental re-evaluation.

Experimental group: n=34 baseline, n=30 at six months, n=32 at 12 months. Control group: n=35 baseline, n=32 at six months, n=32 at 12 months.

Table 25 Median Change in Experiential Processes by Group from Baseline to Six and 12 Months

Experiential Processes	0 – 6 months	0 – 12 months	6 - 12 months
CR			
Experimental	-0.5 (-1.5, 0.5)	-1.5 (-3, 0)	-1.0 (-2, 0.5)
Control	-0.5 (-1.5, 0.5)	-1.0 (-2, 0)	-0.5 (-1.5, 0.5)
SL			
Experimental	-0.5 (-2.5, 0.5)	-1 (-2.5, 1)	-0.5 (-1.5, 1)
Control	0 (-1.5, 1.5)	-0.5 (-2, 1)	0 (-1, 1)
DR			
Experimental	0 (-1, 1)	-0.5 (-2, 1)	-1 (-2, 0.5)
Control	-0.5 (-1.5, 0.5)	*-1.5 (-2.5, -0.5)*	-0.5 (-1.5, 0.5)
ER			
Experimental	-1 (-2.5, 0)	*-2 (-3, -0.5)*	-0.5 (-2, 0.5)
Control	0 (-1.5, 1)	-0.5 (-2, 1)	-0.5 (-2, 1)
SR			
Experimental	-1.5 (-3, 0)	-1.5 (-3, 0)	0 (-1, 1)
Control	0 (-1.5, 1)	-1 (-3, 0)	-0.5 (-2, 0.5)

Note. Values represent median change and 98% confidence interval for the change. CR = consciousness raising, SL = social liberation, DR = dramatic relief, SR = self re-evaluation, ER = environmental re-evaluation.
Significant changes are shown in *italics*.

Behavioural Processes

Table 26 demonstrates the median values for the use of the behavioural processes of change at baseline and follow-up in the experimental and control groups. The use of four of the five behavioural processes of change at baseline were similar between the experimental and control group (Table 26). In contrast, there was a trend for the experimental group to use counterconditioning more often than the control group (98%CI –4, 0). The change in the use of each behavioural process from baseline to six and 12 months follow-up is described in Table 27. There was a trend for a decrease in helping relationships from baseline to six in the control group. In addition, there was a trend for helping relationships to decrease from baseline to 12 months in both groups. Stimulus control and counter conditioning did not significantly change from baseline to

six and 12 months follow-up in the control group. However, there was a trend for a decrease in the use of both these processes from baseline to 12 months in the experimental group. There was no significant change in self-liberation and reinforcement management from baseline to six and 12 months in either group. Mann Whitney tests found that difference between the groups for the change in the use of the five behavioural processes was not significant from baseline to six and 12 months.

Table 26 Behavioural Processes by Group at Baseline, Six and 12 Months

Behavioural Processes	Baseline	6 months	12 months
HR			
Experimental	8 (6, 11)	6.5 (4, 10)	7 (5, 9.8)
Control	9 (6, 12)	7.5 (5, 11.8)	6.5 (4, 11.8)
SC			
Experimental	6 (5, 9)	6.5 (5, 9)	6.5 (4.2, 8)
Control	7 (4, 9)	6.5 (5.2, 8.8)	7 (5, 8)
CC			
Experimental	13.5 (10, 15.20	12.5 (9.8, 15)	11.5 (9, 14)
Control	11 (9, 13)	12 (10, 14)	11 (9, 13)
SO			
Experimental	10 (8, 12.2)	8.5 (7, 13)	8 (7.2, 11)
Control	10 (7, 13)	9 (7, 11)	9 (7, 11.8)
RM			
Experimental	10 (8, 12)	10 (7.8, 12)	9 (7, 11.8)
Control	9 (8, 11)	10 (8, 12)	10 (7, 12)

Note. Values represent median and interquartile range.

HR = helping relationships, SC = stimulus control, CC = counter conditioning, SO = self-liberation, RM = reinforcement management.

Experimental group: n=34 baseline, n=30 at six months, n=32 at 12 months. Control group: n=35 baseline, n=32 at six months, n=32 at 12 months.

Table 27 Median Change in Behavioural Processes by Group from Baseline to Six and 12 Months

Behavioural Processes	0 – 6 months	0 – 12 months	6 - 12 months
HR			
Experimental	-1 (-2, 0.5)	-1.5 (-2.5, 0)	0 (-1.5, 1)
Control	-1 (-2.5, 0)	-1.5 (-3, 0)	-0.5 (-2, 1.5)
SC			
Experimental	-0.5 (-2, 0.5)	-0.5 (-1.5, 0)	-0.5 (-1, 0.5)
Control	0 (-1, 1)	-0.5 (-1.5, 0.5)	0 (-1, 0.5)
CC			
Experimental	-0.5 (-2.5, 0.5)	-1.5 (-2.5, 0)	-1 (-2, 0.5)
Control	0.5 (-1, 1.5)	-1 (-2, 0.5)	-0.5 (-2, 0.5)
SO			
Experimental	-0.5 (-2, 1)	-1 (-2, 0.5)	0 (-1.5, 1)
Control	-0.5 (-2, 0.5)	-1 (-2, 0.5)	0 (-1.5, 1.5)
RM			
Experimental	-0.5 (-2, 0.5)	-0.5 (-2, 0.5)	0 (-1, 1)
Control	0 (-1, 1)	-0.5 (-1.5, 0.5)	-0.5 (-1.5, 1)

Note. Values represent median change and 98% confidence interval for the change. HR = helping relationships, SC = stimulus control, CC = counter- conditioning, SO = self-liberation, RM = reinforcement management.

Effect of an Exercise Consultation on Anthropometric Variables

Weight and Body Mass Index

Anthropometric data was normally distributed. Therefore, parametric tests were used to determine between and within group differences for weight and body mass index (BMI). Weight was not recorded for one control patient at 12 months, and two experimental patients (one subject at six months and one subject at 12 months).

Mean values for weight and BMI at baseline, six and 12 months by group are displayed in Figure 21 and Figure 22, respectively. The mean change in weight and BMI from baseline to six and 12 months are reported in Table 28.

There was trend for mean weight at baseline to be higher in the experimental group compared to the control group (98%CI –15.1, 1.2), see Figure 21. Figure 21 and Table 28 shows that mean weight did not significantly change from baseline to six and 12 months in either group.

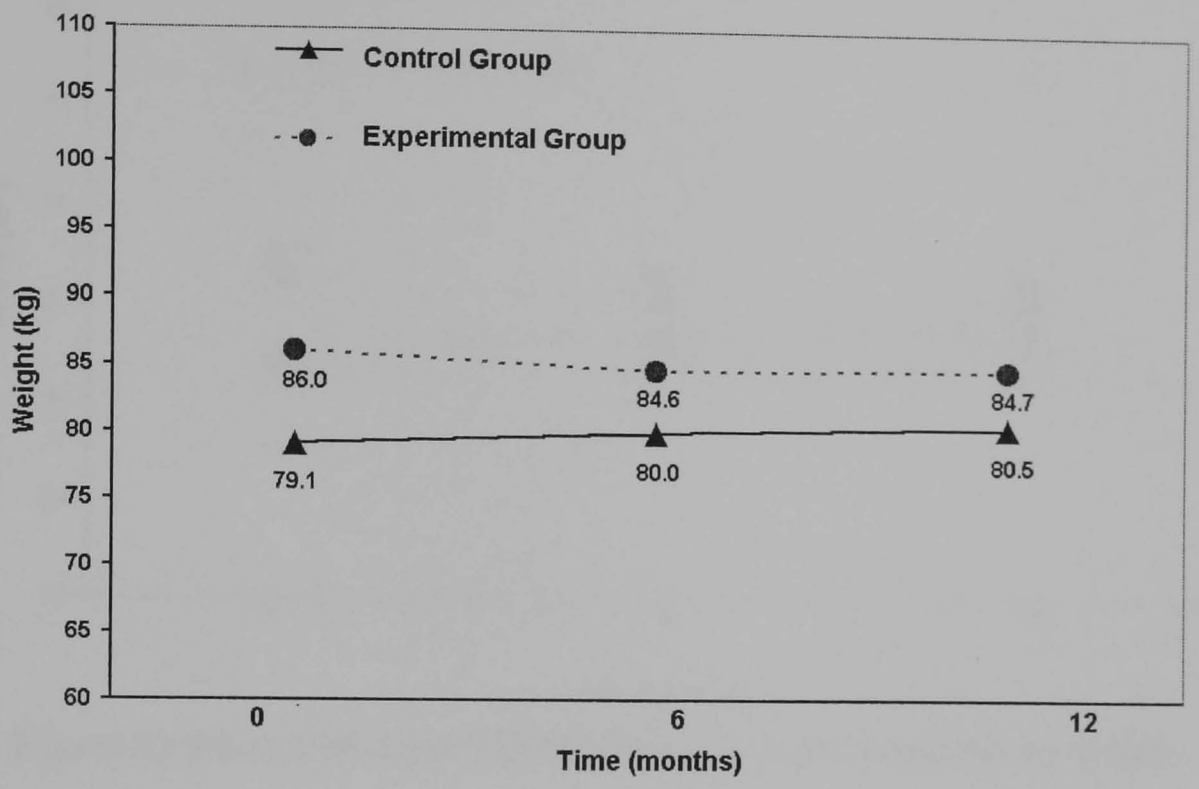


Figure 21 Mean weight (kg) at baseline, six and 12 months by group.

Experimental group: n=35 baseline, n=31 at 6 months, n=32 at 12 months. Control group: n=35 baseline, n=33 at 6 months, n=32 at 12 months.

Table 28 Mean Change in Weight and BMI from Baseline to Follow-Up by Group

Variable	0 – 6 months	0 – 12 months	6 - 12 months
Weight (kg)			
Experimental	0.06 (-1.35, 1.48)	0.06 (-1.78, 1.90)	0.10 (-1.26, 1.45)
Control	0.27 (-1.48, 2.02)	0.75 (-1.32, 2.82)	0.55 (-0.57, 1.67)
BMI			
Experimental	0.01 (-0.48, 0.50)	0.01 (-0.60, 0.62)	0.03 (-0.39, 0.46)
Control	0.14 (-0.46, 0.50)	0.28 (-0.44, 1.0)	0.16 (-0.22, 0.55)

Note. Values represent mean change and 98% confidence interval for the change.

Mean BMI at baseline was 27.01 kg/m^2 in the control group and 28.48 kg/m^2 in the experimental group, the between group difference was borderline significant (98%CI –

3.47, 0.52). Mean BMI did not significantly change from baseline to six and 12 months in either group, as shown in Figure 22 and Table 28.

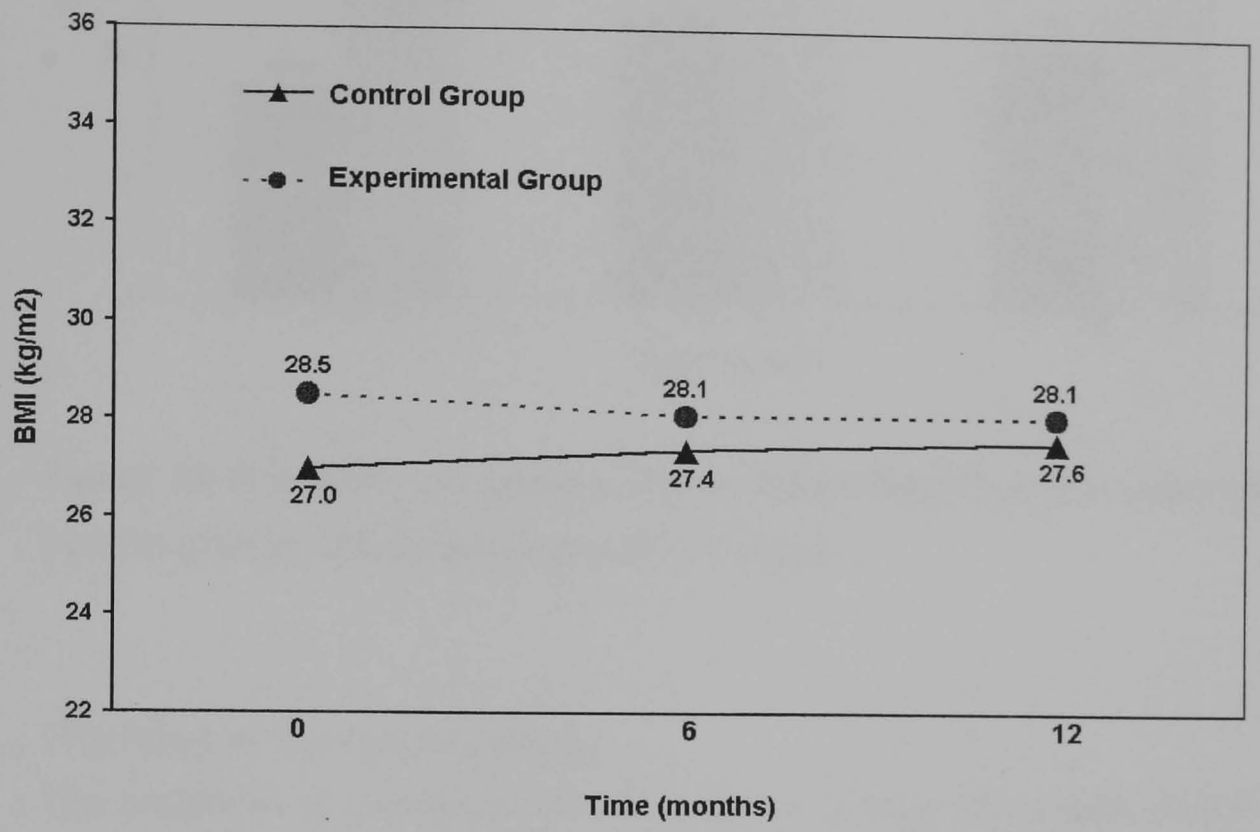


Figure 22 Mean BMI (kg/m^2) at baseline, six and 12 months by group.

Proportion of Obese Patients

The proportion of obese patients, defined as those with a BMI>30kg/m^2, in each group at baseline, six and 12 months was calculated and is displayed in Figure 23. Chi square analysis found that the number of obese patients were similar between the experimental group and the control group at baseline (χ^2=1.30; *df* =1, p=0.26), six months (χ^2=0.97; *df* =1, p=0.32) and 12 months (χ^2=0.72; *df* =1, p=0.40). At baseline, 28.5% (10/35) of the experimental group and 17.1% (6/35) of the control group were obese, as shown in Figure 23. The number of obese patients in the control group did not significantly change from baseline to six (χ^2=0.52; *df* =1, p=0.47) and 12 months (χ^2=0.24; *df* =1, p=0.63). Similarly, in the experimental group there was no significant change in the number of obese patients from baseline to six months (χ^2=0.36; *df* =1, p=0.55) and 12 months (χ^2=0.06; *df=1*, p=0.81).

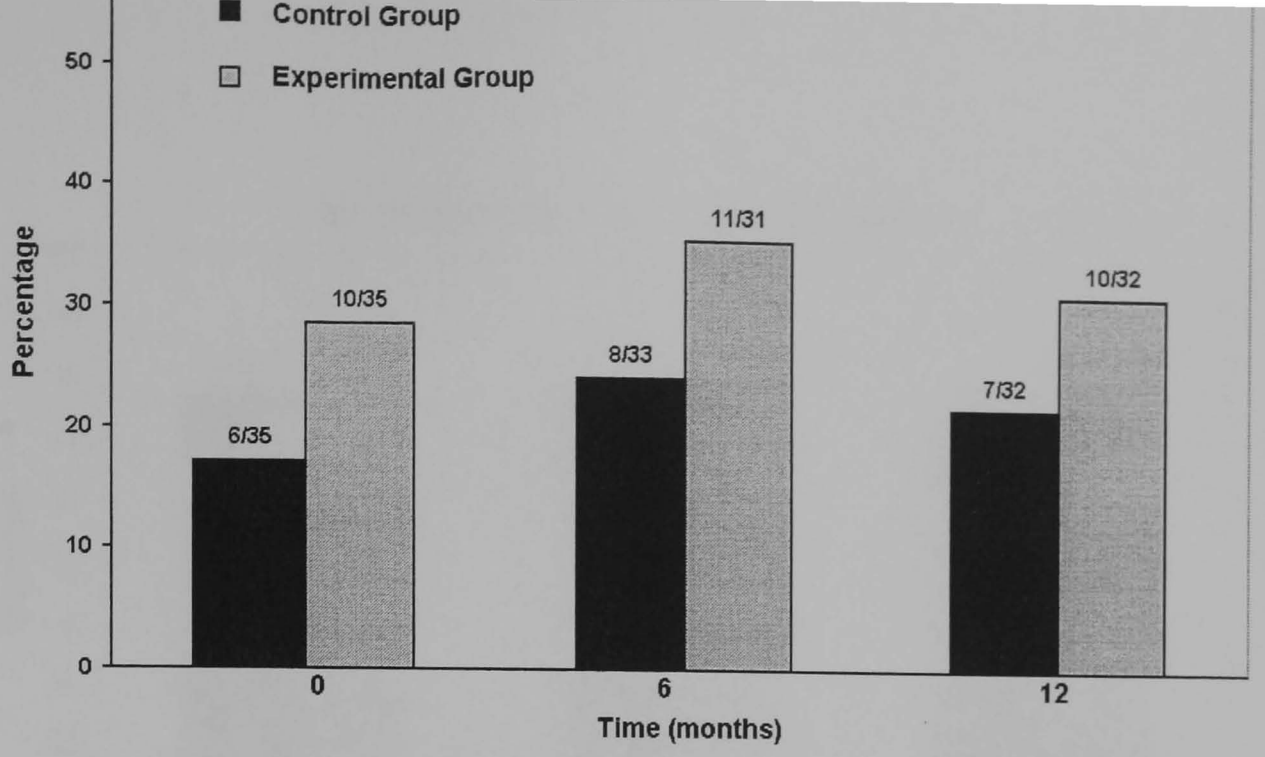


Figure 23 Proportion of obese patients (BMI>30kg/m^2) in the experimental and control groups at baseline, six and 12 months.

Proportion of Overweight Patients

The proportion of overweight patients, defined as those with a BMI>25kg/m^2, in each group at baseline, six and 12 months was calculated and the results are shown in Figure 24. A similar number of patients in the experimental group and the control group were overweight at baseline (χ^2=0.36; *df* =1, p=0.55), six months (χ^2=0.27; *df* =1, p=0.60) and 12 months (χ^2=1.64; *df* =1, p=0.20), see Figure 24. At baseline, 82.9% (29/35) of the experimental group and 77.1% (27/35) of the control group were overweight. In the control group, the number of overweight patients did not significantly change from baseline to six months (χ^2=0.03; *df* =1, p=0.87) and 12 months (χ^2=0.04; *df* =1, p=0.83). Similarly, there was no significant change in the number of overweight patients in the experimental group from baseline to six months (χ^2=0.01; *df* =1, p=0.91) and 12 months (χ^2=0.28; *df=1*, p=0.60).

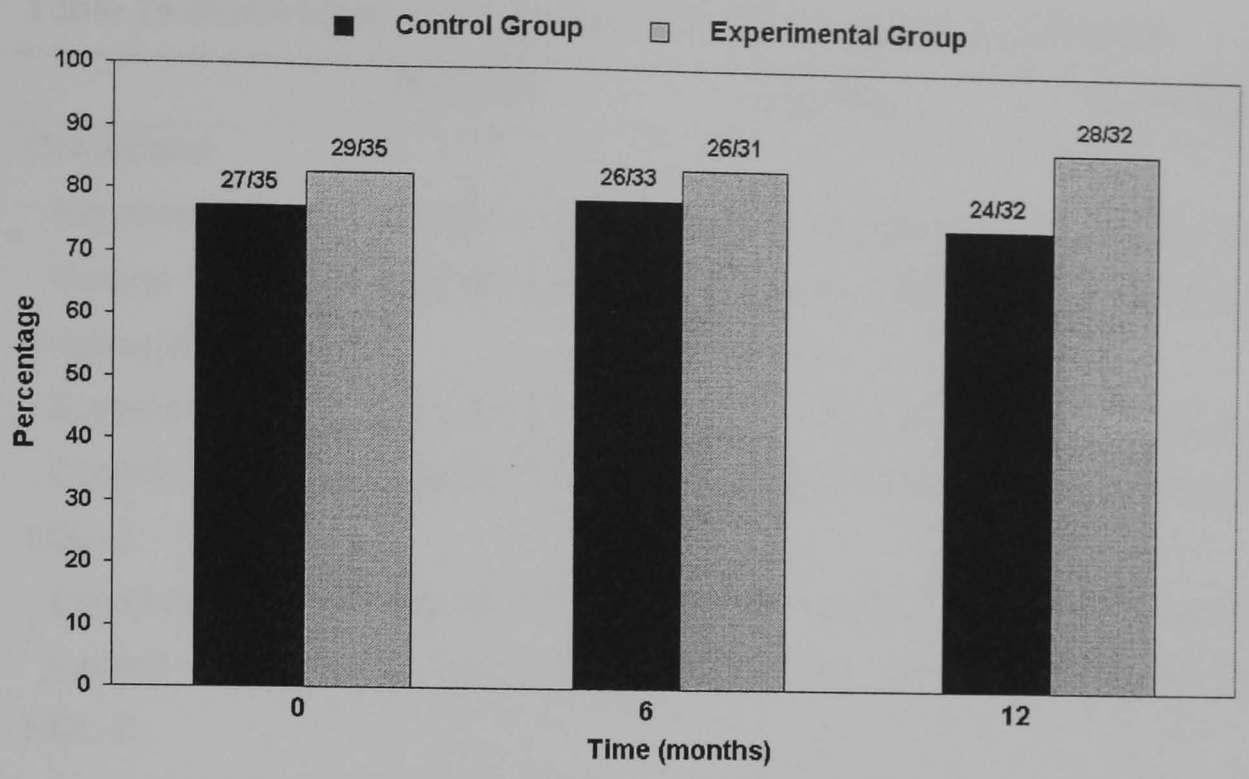


Figure 24 Proportion of overweight patients (BMI>25kg/m^2) in the experimental and control groups at baseline, six and 12 months.

Effect of an Exercise Consultation on Blood Lipids

Non-parametric tests were used to analyse the between and within group differences, as the lipid data were not normally distributed. Cholesterol and triglyceride values were not recorded for one control patient and one experimental patient at six months. LDL and HDL cholesterol values were not recorded at baseline for two control patients and two experimental patients. Median values for cholesterol, triglycerides, LDL cholesterol, HDL cholesterol and cholesterol/HDL cholesterol ratio at baseline, six and 12 months in the experimental group and the control group are displayed in Table 29. The median change in lipid values from baseline to six and 12 months by group are shown in Table 30.

Table 29 Blood Lipid Levels by Group at Baseline, 6 and 12 Months

	Baseline	6 months	12 months
Cholesterol			
Experimental	4.34 (3.81, 4.79)	4.31 (3.69, 4.93)	4.51 (3.80, 5.97)
Control	4.19 (3.86, 4.89)	4.50 (3.83, 4.82)	4.29 (3.98, 5.05)
Triglycerides			
Experimental	1.66 (1.23, 2.28)	1.65 (1.12, 2.06)	1.66 (1.26, 2.26)
Control	1.50 (0.95, 1.98)	1.28 (0.97, 2.28)	1.44 (1.00, 2.21)
LDL-C			
Experimental	2.51 (2.04, 2.76)	2.29 (1.96, 2.76)	2.31 (1.90, 2.77)
Control	2.30 (1.96, 2.89)	2.44 (1.89, 2.90)	2.39 (1.90, 2.83)
HDL-C			
Experimental	1.10 (1.00, 1.30)	1.20 (1.09, 1.40)	1.25 (1.08, 1.48)
Control	1.20 (1.00, 1.40)	1.24 (1.08, 1.52)	1.24 (1.10, 1.56)
Chol/HDL-C			
Experimental	3.68 (3.08, 4.58)	3.49 (2.80, 3.98)	3.32 (2.94, 4.13)
Control	3.42 (3.22, 4.49)	3.46 (3.11, 4.10)	3.47 (3.02, 4.09)

Note. Values represent median and interquartile (IQ) range.

Table 30 Median Change in Blood Lipid Levels from Baseline to Six and 12 Months by Group

	0 – 6 months	0 – 12 months	6 - 12 months
Cholesterol			
Experimental	0.07 (-0.20, 0.30)	0.14 (-0.11, 0.35)	0.08 (-0.09, 0.24)
Control	0.03 (-0.30, 0.28)	0.08 (-0.35, 0.34)	-0.04 (-0.23, 0.16)
Triglycerides			
Experimental	0.04 (–0.20, 0.28)	0.09 (-0.08, 0.30)	0.06 (-0.19, 0.36)
Control	-0.08 (–0.33, 0.22)	0.07 (–0.14, 0.28)	0.08 (–0.09, 0.22)
LDL-C			
Experimental	-0.04 (–0.25, 0.14)	-0.10 (–0.35, 0.11)	0.02 (–0.20, 0.21)
Control	0.01 (–0.23, 0.20)	-0.02 (–0.34, 0.21)	-0.02 (–0.22, 0.17)
HDL-C			
Experimental	0.08 (–0.01, 0.19)	0.11 (0.04, 0.18)	0.02 (–0.05, 0.09)
Control	0.05 (–0.10, 0.12)	0.06 (–0.02, 0.14)	-0.01 (–0.06, 0.06)
Chol/HDL-C			
Experimental	-0.21 (–0.72, 0.06)	-0.21(–0.44, -0.02)	0.04 (–0.21, 0.32)
Control	-0.16 (–0.46, 0.12)	-0.11 (–0.40, 0.11)	0.03 (–0.18, 0.23)

Note. Values represent median change and 98% confidence interval for the change.

Total Cholesterol

Baseline cholesterol values were 4.34 mmol/l in the experimental group and 4.19 mmol/l in the control group (Table 29), which are below the level recommended by national guidelines (i.e. < 5 mmol/l). A Mann Whitney test revealed that baseline cholesterol levels were similar between the groups (98%CI –0.44, 0.51). Table 30 demonstrates that cholesterol levels did not significantly change from baseline to six and 12 months in the experimental group or the control group. Similarly, there was no significant difference between the groups for the change in cholesterol from baseline to six months (98%CI –0.40, 0.32), baseline to 12 months (98%CI –0.41, 0.28) and six to 12 months (98%CI –0.37, 0.14).

Triglycerides

Table 29 shows that baseline triglyceride concentrations were 1.66 mmol/l in the experimental group and 1.50 mmol/l in the control group, which are within the normal

range (i.e. <5.2 mmol/l). Mann Whitney test revealed that baseline triglyceride levels were similar between the groups (98%CI –0.58, 0.18). Triglyceride levels did not significantly change from baseline to follow-up in either group (Table 30). Similarly, there was no significant difference between the groups for the change in triglycerides from baseline to six months (98%CI –0.45, 0.23), baseline to 12 months (98%CI –0.29, 0.24) and six to 12 months (98%CI –0.31, 0.30).

LDL Cholesterol

Table 29 shows that median values for LDL cholesterol were similar in the experimental and control groups at baseline (98%CI –0.39, 0.47), and were lower than recommended targets (i.e. < 3 mmol/l) at baseline, six and 12 months in both groups. LDL cholesterol concentrations did not significantly change from baseline to six and 12 months in either group (Table 30). Similarly, there was no significant difference between the groups for the change in LDL-C from baseline to six months (98%CI –0.22, 0.29), 12 months (98%CI –0.24, 0.40) and six to 12 months (98%CI –0.29, 0.24).

HDL Cholesterol

As shown in Table 29 and Figure 25, baseline HDL cholesterol concentrations were similar (98%CI –0.1, 0.2) in the experimental group (1.10 mmol/l) and the control group (1.20 mmol/l) and were within the normal range (i.e. >0.91 mmol/l). In the experimental group, there was a trend for an increase in HDL cholesterol from baseline by 0.08 at six months mmol/l and a significant increase by 0.11 mmol/l at 12 months (Table 30). In contrast, HDL cholesterol did not significantly change from six to 12 months. In the control group, there was a trend for an increase in HDL cholesterol from baseline by 0.05 mmol/l at six months and 0.06 mmol/l at 12 months, with no significant change from six to 12 months (Table 30). The difference between the groups for the change in HDL cholesterol was not significant from baseline to six months (98%CI –0.14, 0.09), 12 months (98%CI –0.15, 0.06) and six to 12 months (98%CI –0.12, 0.07). The change in HDL-C over the study period in each group is shown in Figure 25.

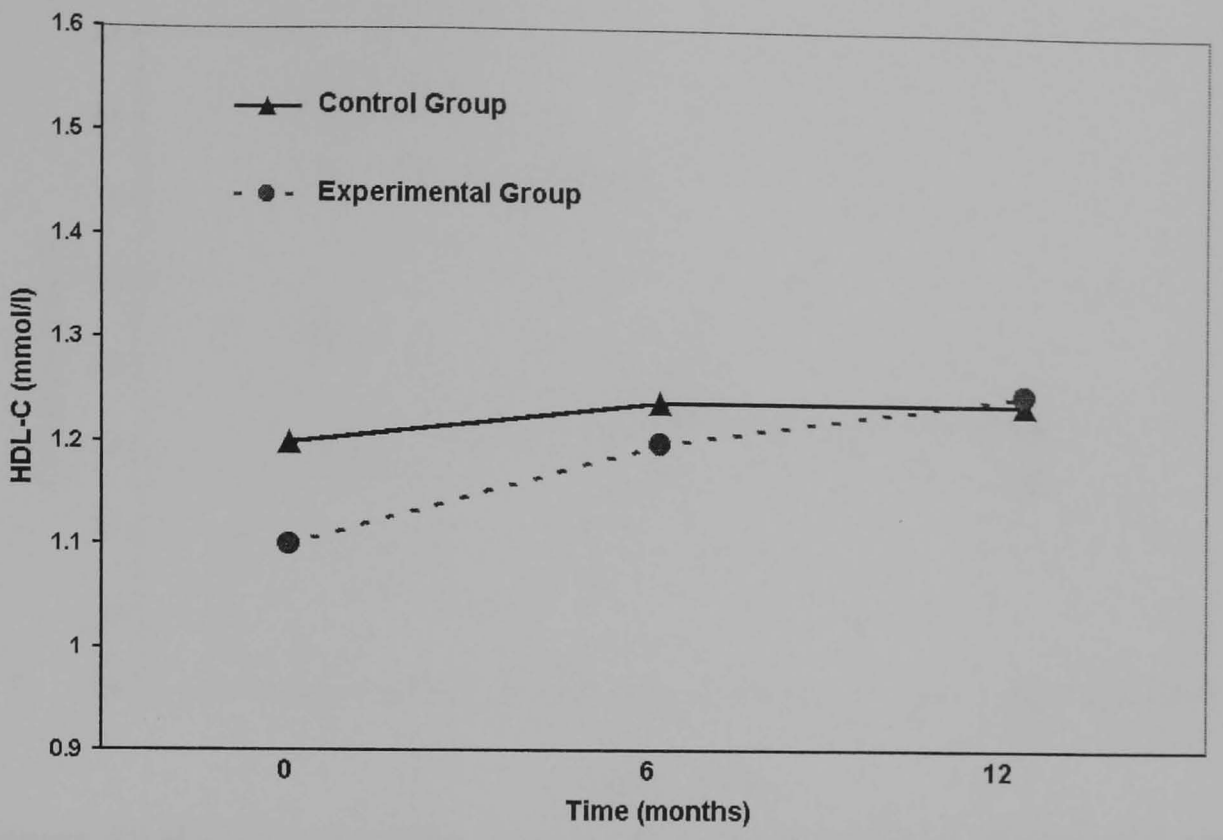


Figure 25 Median values for HDL cholesterol at baseline, six and 12 by group

Total Cholesterol/HDL Cholesterol Ratio

Table 29 and Figure 26 shows that the median values for the cholesterol/HDL cholesterol ratio at baseline and follow-up were below the recommended target of 5. A Mann Whitney test revealed that baseline values were similar between the groups (98%CI –0.59, 0.42). Table 30 indicates that the cholesterol/HDL cholesterol ratio did not significantly change from baseline in the control group. In contrast, the ratio of cholesterol to HDL significantly improved from baseline to six and 12 months in the experimental group, with no significant change from six to 12 months (Table 30). There was no significant between group difference for the change in cholesterol/HDL cholesterol ratio from baseline to six months (98%CI –0.33, 0.48), 12 months (98%CI –0.16, 0.41) and six to 12 months (98%CI –0.36, 0.32). Figure 26 displays the change in cholesterol/HDL-C ratio over the study period in each group.

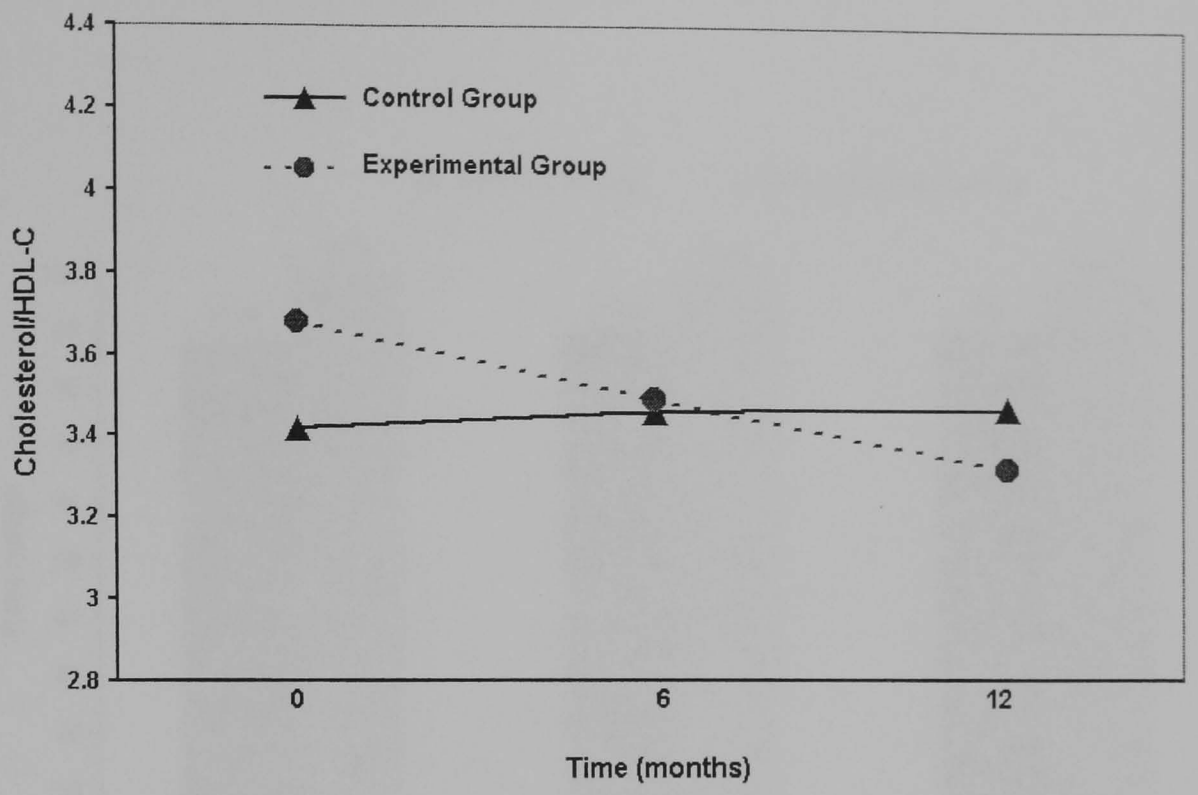


Figure 26 Median values for ratio of cholesterol to HDL-C at baseline, six and 12 by group

Proportion of Patients Taking Lipid Lowering Medication

Figure 27 displays the proportion of patients taking lipid-lowering medication at baseline, six and 12 months in each group. At baseline, 88.6% of the control group and 100% of the experimental group were taking lipid-lowering medication. The number of patients taking lipid-lowering medication did not change at six and 12 months in either group, see Figure 27. Three control patients and five experimental patients had their dose of medication changed at six months compared to baseline. Furthermore, three controls and seven experimental patients had their medication dose altered at 12 months compared with baseline.

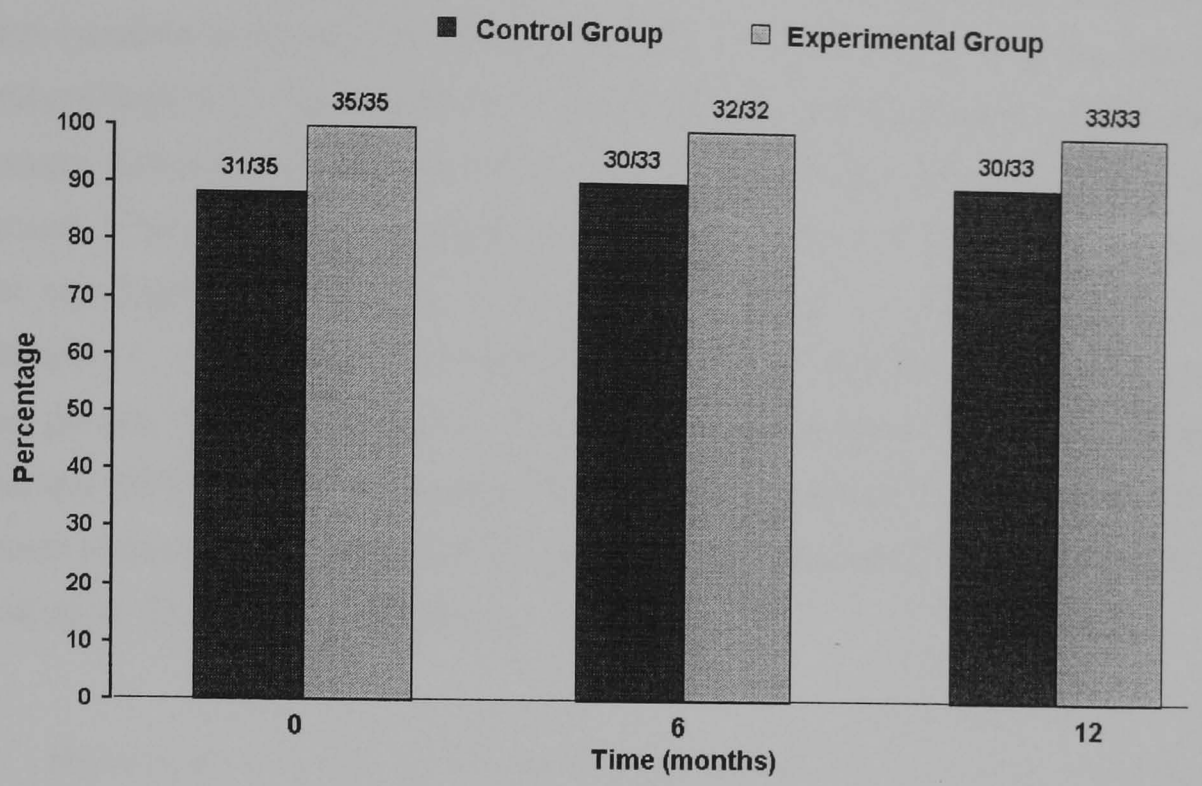


Figure 27 Proportion of patients taking lipid lowering medication at baseline, six and 12 months in the experimental and control group.

Effect of an Exercise Consultation on Psychological Function

Anxiety and Depression

Anxiety and depression was measured using the Hospital Anxiety and Depression Scale. The total score for the anxiety and depression subscales ranges from 0 to 21. The scores for the anxiety and depression subscales can be used to classify individuals into normal (0-7), mild (8-10), moderate (11-14) and severe (≥15) categories. In addition, it has been suggested that individuals with HADS scores ≥ 8 on the anxiety or depression subscales are psychologically distressed.

HADS scores were not normally distributed, thus non-parametric tests were used to analyse the between and within group differences. Median scores for anxiety and depression at baseline, six and 12 months follow-up in the experimental and control group are displayed in Figure 28. The median scores for anxiety and depression were within the normal range (i.e. 0 – 7) at baseline, six and 12 months in both groups.

Mann Whitney tests revealed that baseline scores for anxiety and depression were similar between the groups, (98%CI –3, 1) and (98%CI –1, 1), respectively. Wilcoxon signed rank tests found that anxiety and depression scores did not significantly change from baseline to six and 12 months follow-up in the experimental group and the control group (Table 31). From six to 12 months, anxiety scores remained unchanged in both groups, however there was a trend for a median increase in depression by 0.5 in both groups (Table 31). The difference between the groups for the change in anxiety was not significant from baseline to six months (98%CI –2, 1), baseline to 12 months (98%CI –1, 2) and six to 12 months (98%CI –1, 1). Similarly, the difference between the groups for the change in depression was not significant from baseline to six months (98%CI –1, 1). However, the increase in depression was greater in the control group compared to the experimental group from baseline to 12 months (98%CI 0, 1) and six to 12 months (98%CI 0, 1).

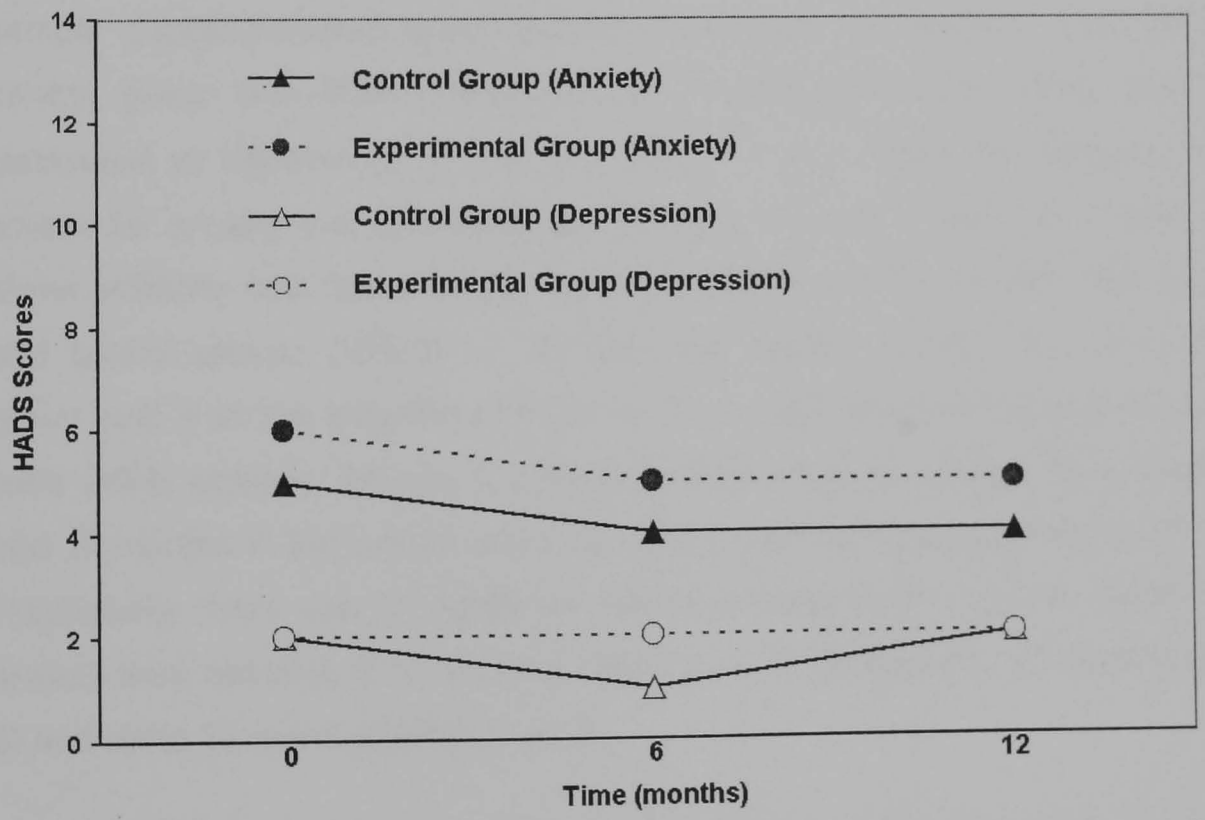


Figure 28 Median scores for anxiety and depression at baseline, six and 12 months in the experimental and control groups.

Experimental group: n=35 baseline, n=32 at six months, n=33 at 12 months. Control group: n=35 baseline, n=33 at six months, n=32 at 12 months.

Table 31 Median Change in Anxiety and Depression from Baseline to Six and 12 Months by Group

	0 – 6 months	0 – 12 months	6 - 12 months
Anxiety			
Experimental	0 (-1.5, 1)	-0.5 (-1, 0.5)	0 (-1, 0.5)
Control	-0.5 (-1.5, 1)	0 (-1.5, 1)	0 (-0.5, 1)
Depression			
Experimental	-0.5 (-1, 0.5)	0 (-0.5, 0.5)	0.5 (0, 1)
Control	0 (-1, 0.5)	0.5 (-0.5, 1.5)	0.5, (0, 1.5)

Note. Values represent median change and 98% confidence interval for the change

Psychologically Distressed Patients

Psychologically distressed patients were identified as those having a baseline HAD score ≥ 8 on the anxiety or depression subscales. At baseline, 33% (23/70) of the sample population were psychologically distressed. In addition, 25% (9/35) of the control group and 40% (14/35) of the experimental group were psychologically distressed at baseline ($\chi^2 = 1.6$, $df = 1$, $p = 0.23$). Figure 29 displays the median scores for anxiety and depression at baseline, six and 12 months in both groups. A Mann Whitney test found similar baseline anxiety scores between the experimental and control groups (98%CI –1, 5). Baseline anxiety scores were 10 in the control group and 9 in the experimental group. Thus, distressed individuals in both groups were mildly anxious. Anxiety scores did not significantly change from baseline to six and 12 months in the control group and the experimental group (Figure 29, Table 32). Additionally, there was no significant difference between the groups for the change in anxiety from baseline to six months (98%CI –5, 5), baseline to 12 months (98%CI –6, 5) and six to 12 months (98%CI –4, 3).

Figure 29 shows that median scores for depression were within the normal range (i.e. less than 8) at baseline, six and 12 months in both groups. There was no significant difference between the groups (98%CI –2, 5) for depression scores at baseline. Depression scores did not significantly change from baseline to six and 12 months in the experimental group and the control group (Table 32). From six to 12 months, there was no significant change in depression in the control group, however there was a borderline increase in depression from six to 12 months in the experimental group

(Table 32). There was no difference between the groups for the change in depression from baseline to six months (98%CI –2, 4), baseline to 12 months (98%CI –2, 5) and six to 12 months (98%CI –2, 6).

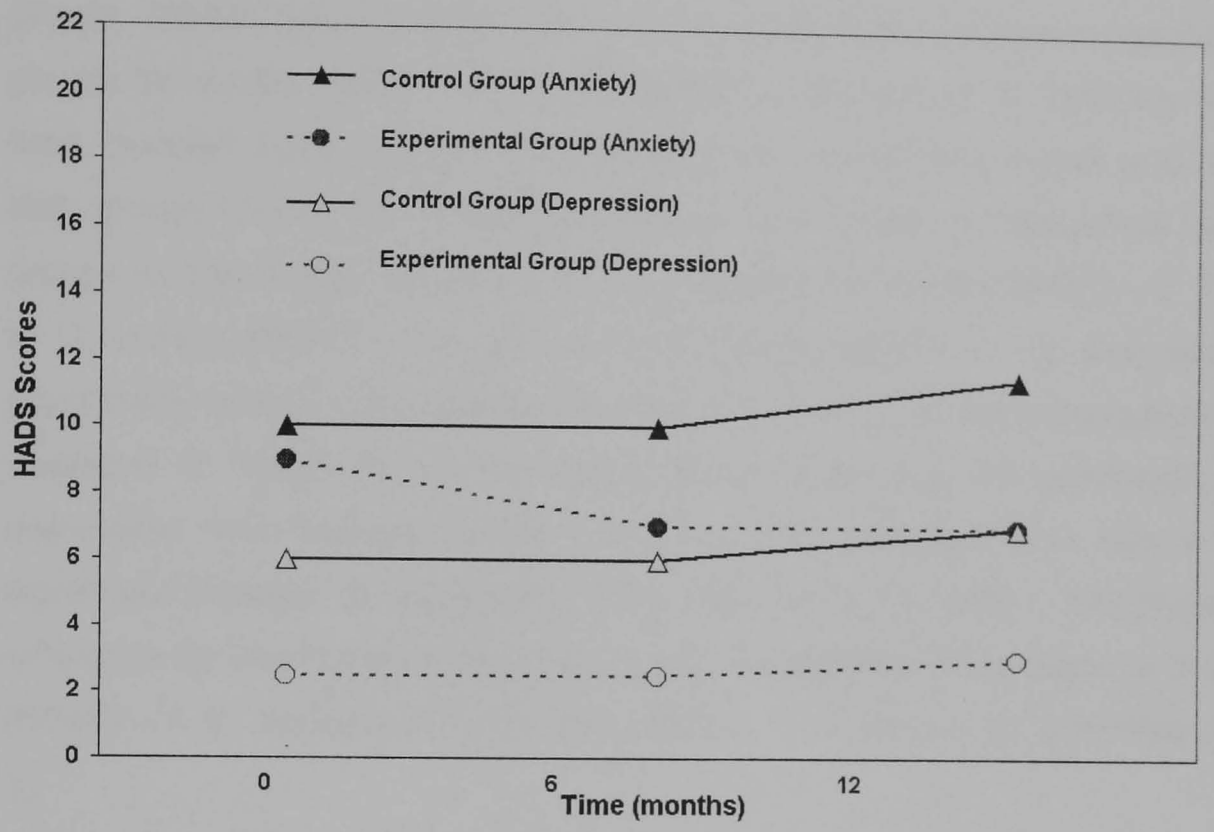


Figure 29 Median scores for anxiety and depression at baseline, six and 12 by group in patients who were psychologically distressed at baseline.

Table 32 Median Change in Anxiety and Depression by Group in Patients Who Were Psychologically Depressed at Baseline

	0 – 6 months	0 – 12 months	6 - 12 months
Anxiety			
Experimental	-1.5 (-4, 1.5)	-1 (-3, 1.5)	0.5 (-1, 2)
Control	-1 (-6, 6)	-1.5 (-6, 4)	0 (-4, 4)
Depression			
Experimental	0 (-1.5, 1.5)	0 (-1, 3)	0.5 (0, 1.5)
Control	0.2 (-2, 4)	2.5 (-2, 6)	3 (-2, 6)

Note. Values represent median change and 98% confidence interval for the change

Non-Distressed Patients

Patients with a baseline score less that 8 on the anxiety or depression subscales were classified as non-psychologically distressed. Figure 30 shows that anxiety and depression scores were within the normal range at baseline, six and 12 months in both groups. Similar baseline scores were observed between the experimental and control groups for anxiety (98%CI –2, 2) and depression (98%CI –2, 2). Wilcoxon signed rank tests revealed that anxiety scores remained unchanged from baseline to follow-up in both groups (Table 33). In addition, there was no significant difference between the groups for the change in anxiety from baseline to six months (98%CI –2, 1), baseline to 12 months (98%CI –1, 2) and six to 12 months (98%CI –1, 2). Depression did not significantly change from baseline to six and 12 months in the experimental group, as displayed in Table 33. In the control group, there was no significant change in depression from baseline to six and 12 months, however there was a borderline significant increase in depression by 0.5 from six to 12 months. The between group difference for the change in depression was not significant from baseline to six months (98%CI –1, 1), baseline to 12 months (98%CI –1, 1) and six to 12 months (98%CI –1, 1).

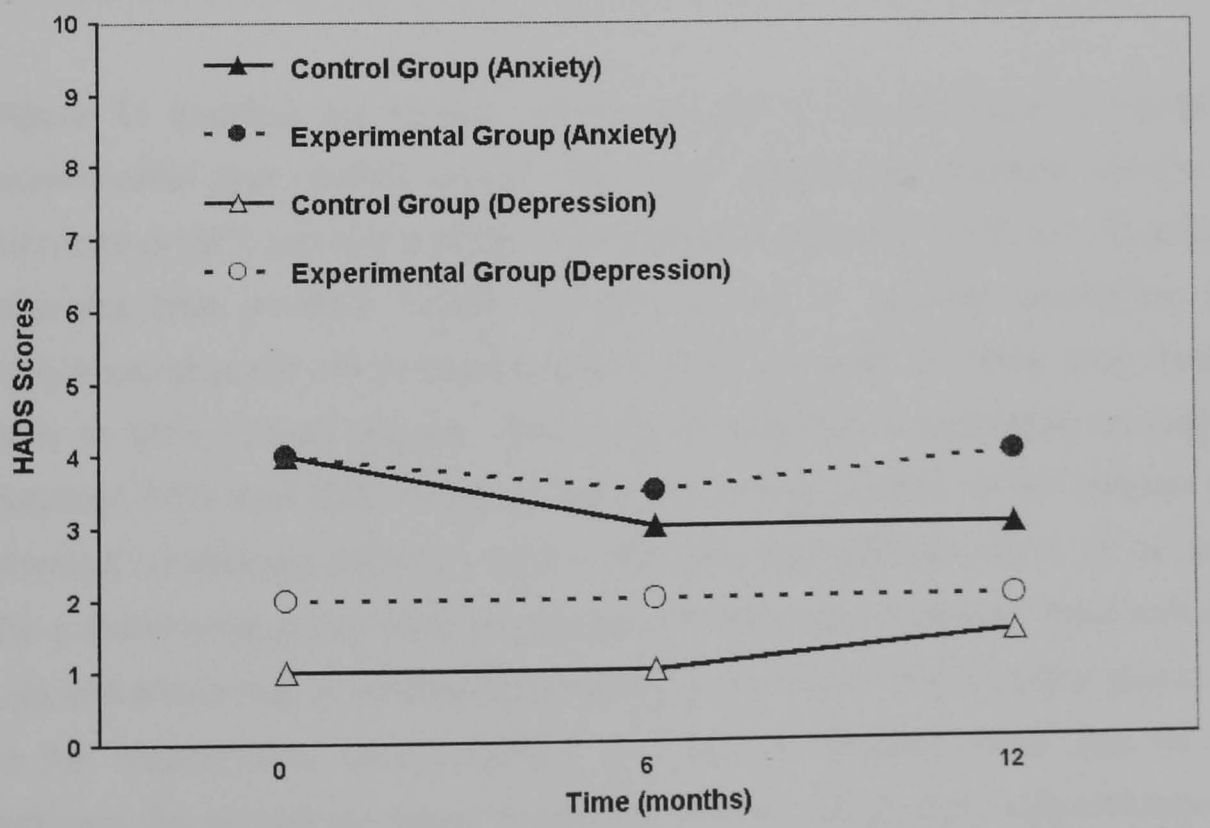


Figure 30 Median scores for anxiety and depression at baseline, six and 12 by group in non-distressed patients at baseline

Table 33 Median Change in Anxiety and Depression in Non-Distressed Patients

	0 – 6 months	0 – 12 months	6 - 12 months
Anxiety			
Experimental	0.5 (-0.5, 1)	0 (-1, 1)	-0.5 (-1.5, 0.5)
Control	-0.5 (-1.5, 1)	0.5 (-1, 1.5)	0 (-1, 1)
Depression			
Experimental	-0.5 (-1.5, 0.5)	0 (-1, 0.5)	0.5 (-0.5, 1)
Control	0 (-1, 0.5)	0 (-0.5, 0.5)	0.5 (0, 1)

Note. Values are median change and 98% confidence interval for the change

Quality of Life

Quality of life was assessed at baseline, six and 12 months by the UK SF-36 version 2. This questionnaire assess the following 8 dimensions: physical functioning (PF), social functioning (SF), role limitations due to physical problems (RLP), role limitations due to emotional problems (RLE), pain (P), energy/vitality (EV), mental health (MH), and health perception (HP). For each dimension, the scale ranges from 0 (worst possible health) to 100 (best possible health). The SF-36 data were not normally distributed, thus non-parametric tests were used to detect between and within group differences.

Figure 31 displays the median scores for the SF-36 subscales at baseline in the experimental and control group. The figure shows that baseline values for social functioning (SF) and role limitations emotional (RLE) were 100% in both groups, which indicates best possible health. Baseline values for physical functioning (PF), role limitations physical (RLP) mental health (MH) and pain (P) were high, ranging from 80% to 90% in both groups. Scores for energy/vitality and health perception were between 60% and 70% in both groups. There was a trend for the median scores for physical functioning (98%CI –10.0, -0.01) and pain (98%CI –11.1, 0) to be higher in the experimental group compared to the control group. Whereas, there was a trend for role limitations due to emotional problems to be higher in the control group compared to the experimental group (98%CI 0, 8.33). In contrast, there was no difference between the groups for social functioning (98%CI –0.01, 0.0), role limitations physical (98%CI –12.5, 6.2), mental health (98%CI –10.0, 5.0), energy/vitality (98%CI –6.25, 12.5) and health perception (98%CI –12.0, 10.0).

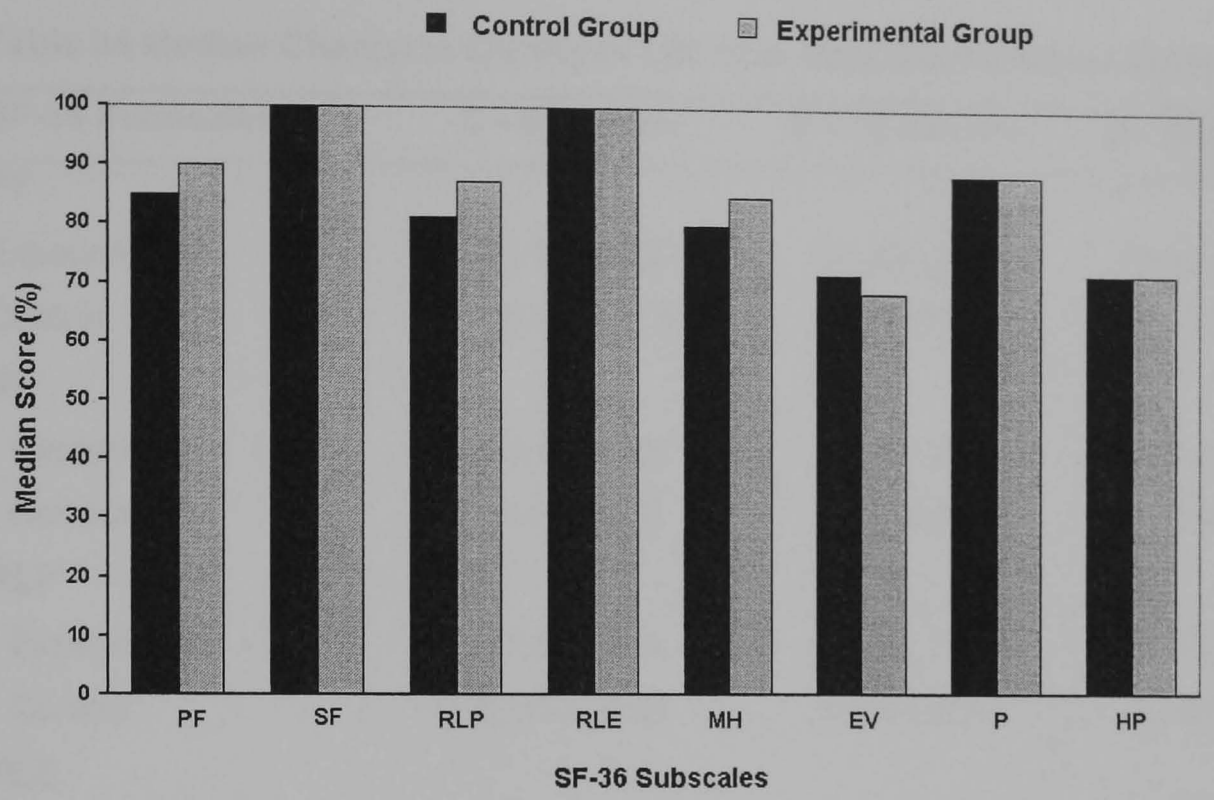


Figure 31 Median scores for SF-36 subscales at baseline by group.

A score of 100 indicates best possible health. Physical functioning (PF), social functioning (SF), role limitations physical (RLP), role limitations emotional (RLE), pain (P), energy/vitality (EV), mental health (MH) and health perception (HP).

The median change in the SF-36 subscales from baseline to follow-up in the experimental and control group is shown in Table 34. Scores for four SF-36 subscales were maintained from baseline to six and 12 months in the experimental group; these were physical function, mental health, energy/vitality and health perception. Similarly, there was no significant change in social function, role limitations due to physical, mental health and health perception from baseline to six and 12 months in the control group. From baseline to six months, there was a trend for an improvement in social function and a deterioration in pain in the experimental group. In the control group, there was a trend for a decline in role limitations due to emotional problems and energy/vitality in the control group. From baseline to 12 months, there was a trend for an improvement in three SF-36 subscales in the experimental group; these were social function, role limitations due to physical problems and role limitations due to emotional problems. In contrast, there was a trend for a decline in physical function and role limitations emotional in the control group. From six to 12 months, there was a trend for an improvement in role limitations emotional and energy/vitality in the experimental

group, and a trend for an improvement in pain in both groups.

Table 34 Median Change in Quality of Life from Baseline to Follow-Up by Group

SF-36 Subscales	0 – 6 months	0 – 12 months	6 - 12 months
PF			
Experimental	0 (-5.0, 2.5)	0 (-5.0, 2.5)	0 (-2.5, 2.5)
Control	-2.5 (-7.5, 2.5)	-5.0 (-10.0, 0)	-2.5 (-7.5, 2.5)
SF			
Experimental	6.3 (0, 12.5)	0 (0, 12.5)	0 (0, 0)
Control	0 (-6.3, 6.3)	0 (-6.3, 6.3)	0 (0, 0)
RLP			
Experimental	0 (-6.3, 9.4)	6.3 (0, 12.5)	6.3 (-3.1, 15.6)
Control	0 (-9.4, 9.4)	0 (-9.4, 9.4)	0 (-9.4, 6.3)
RLE			
Experimental	0 (-4.2, 8.3)	0 (0, 8.34)	0 (0, 4.16)
Control	-4.2 (-16.7, 0)	-4.2 (-8.3, 0)	0 (-8.3, 4.2)
MH			
Experimental	0 (-2.5, 5.0)	1.5 (-2.5, 5.0)	0 (-2.5, 5.0)
Control	0 (-5.0, 7.5)	0 (-2.5, 5.0)	0 (-2.5, 5.0)
EV			
Experimental	0 (-6.3, 6.3)	3.1 (-3.1, 9.4)	3.13 (0, 6.2)
Control	-3.1 (-9.4, 0)	-3.1 (-9.4, 3.1)	0 (-6.3, 6.3)
P			
Experimental	0 (-11.1, 0)	0 (-5.6, 5.6)	0 (0, 5.6)
Control	0 (-11.1, 5.6)	0 (-5.6, 5.6)	0 (0, 5.6)
HP			
Experimental	-1.0 (-7.5, 5.0)	0 (-5.0, 7.5)	1.5 (-5.0, 7.5)
Control	-2.5 (-8.0, 5.0)	-2.5 (-11.0, 6.0)	-2.0 (-10.0, 6.5)

Notes. Values are median change and 98% confidence interval for the change.

A score of 100 indicates best possible health. PF = Physical function, SF = Social function, RLP = Role limitations physical, RLE = Role limitations emotional, P = Pain, EV = Energy/vitality, MH = Mental health, HP = Health perception.

In addition, the difference between the groups for the change in SF-36 dimensions from baseline to six and 12 months was examined. These findings are shown in Figures 32, 33 and 34. There was a trend for a greater improvement in social function, role limitations due to emotional problems and energy/vitality from baseline to six and 12 months in the experimental group compared to the control group, as shown in Figures 32 and 33. From six to 12 months, there was a borderline significant difference between the groups for the change pain favouring the experimental group (Figure 34).

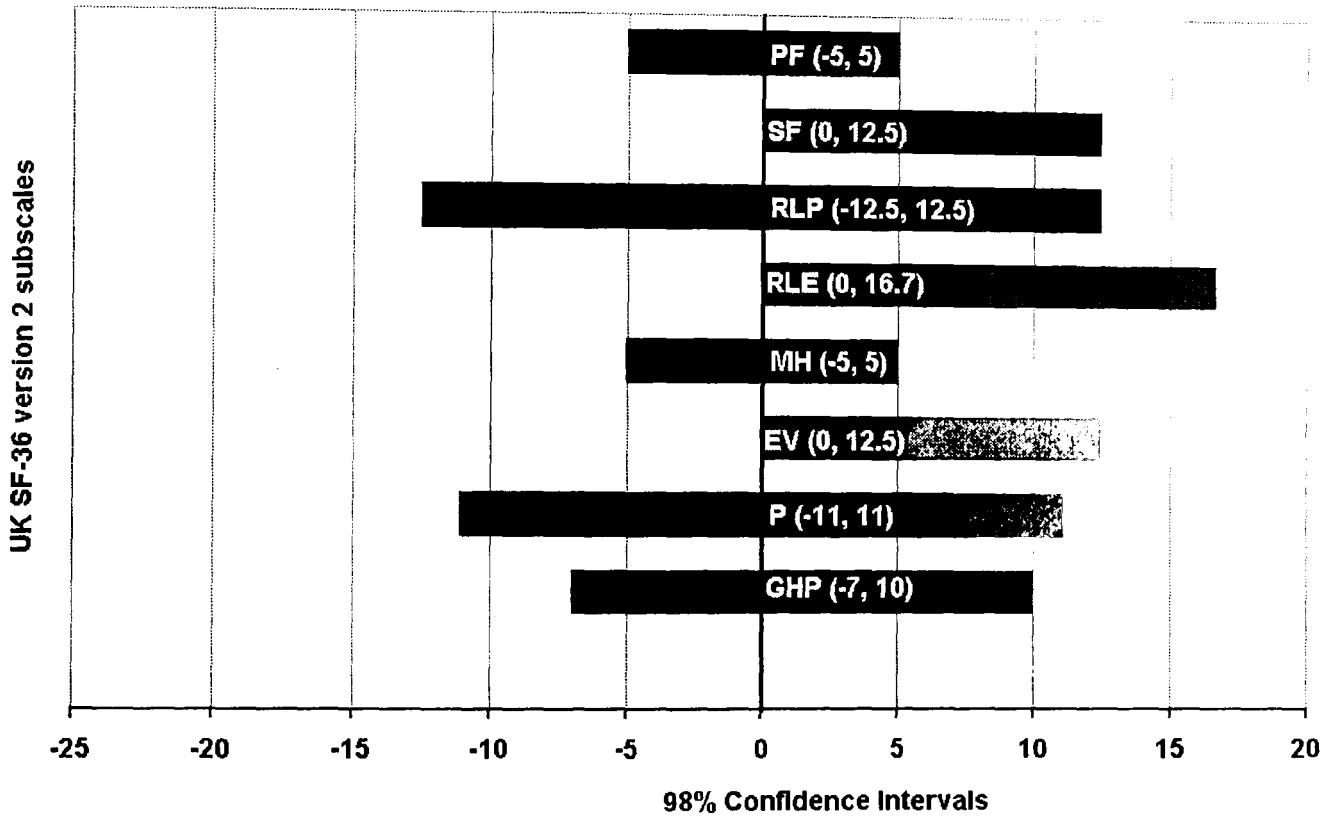


Figure 32 98% confidence intervals for the difference between the groups for the change in quality of life from baseline to six months.

Red bars represent borderline significant differences between the groups for changes in SF-36 subscales. Red bars skewed to the right indicate an advantage to the experimental group.

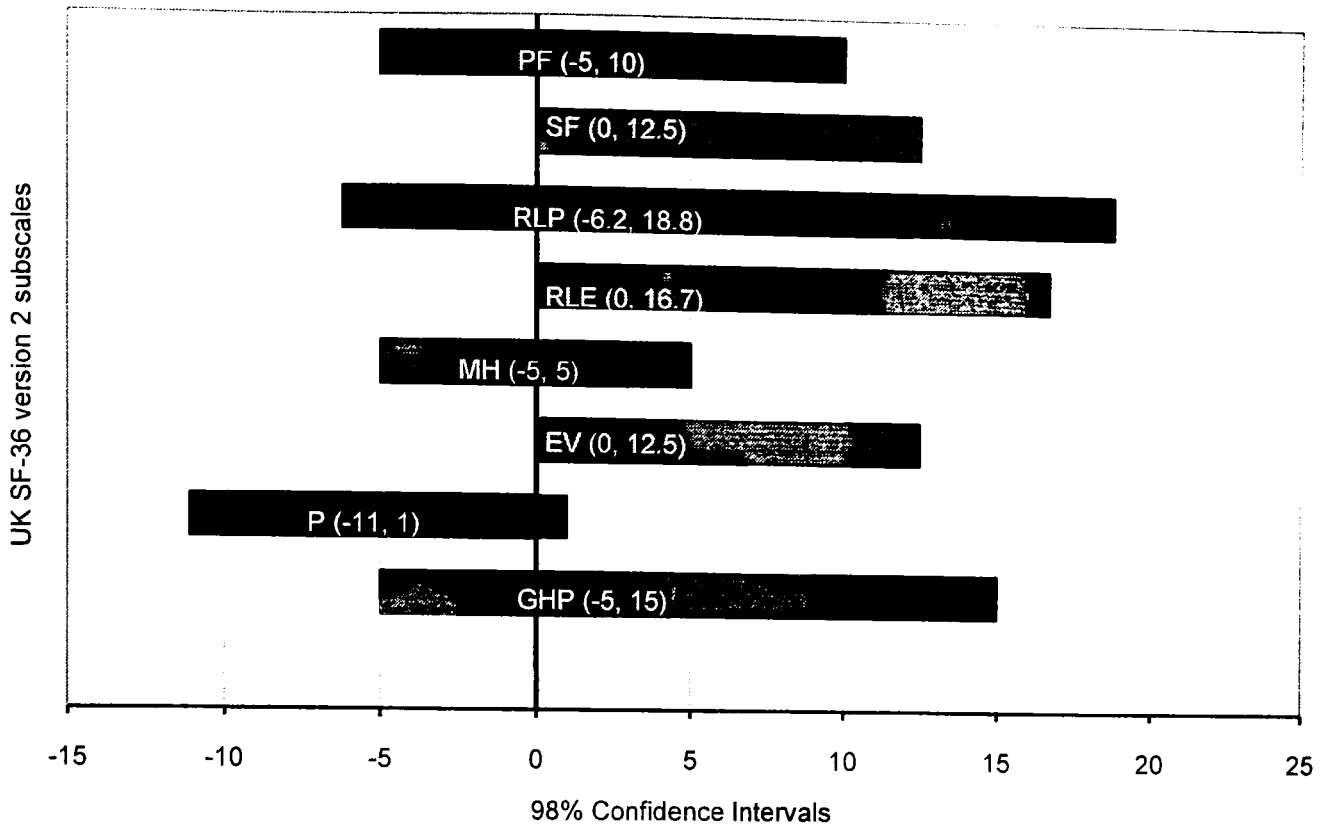


Figure 33 98% confidence interval for the difference between the groups for the change in quality of life from baseline to 12 months.

Red bars represent borderline significant differences between the groups for changes in SF-36 subscales. Red bars skewed to the right indicate an advantage to the experimental group.

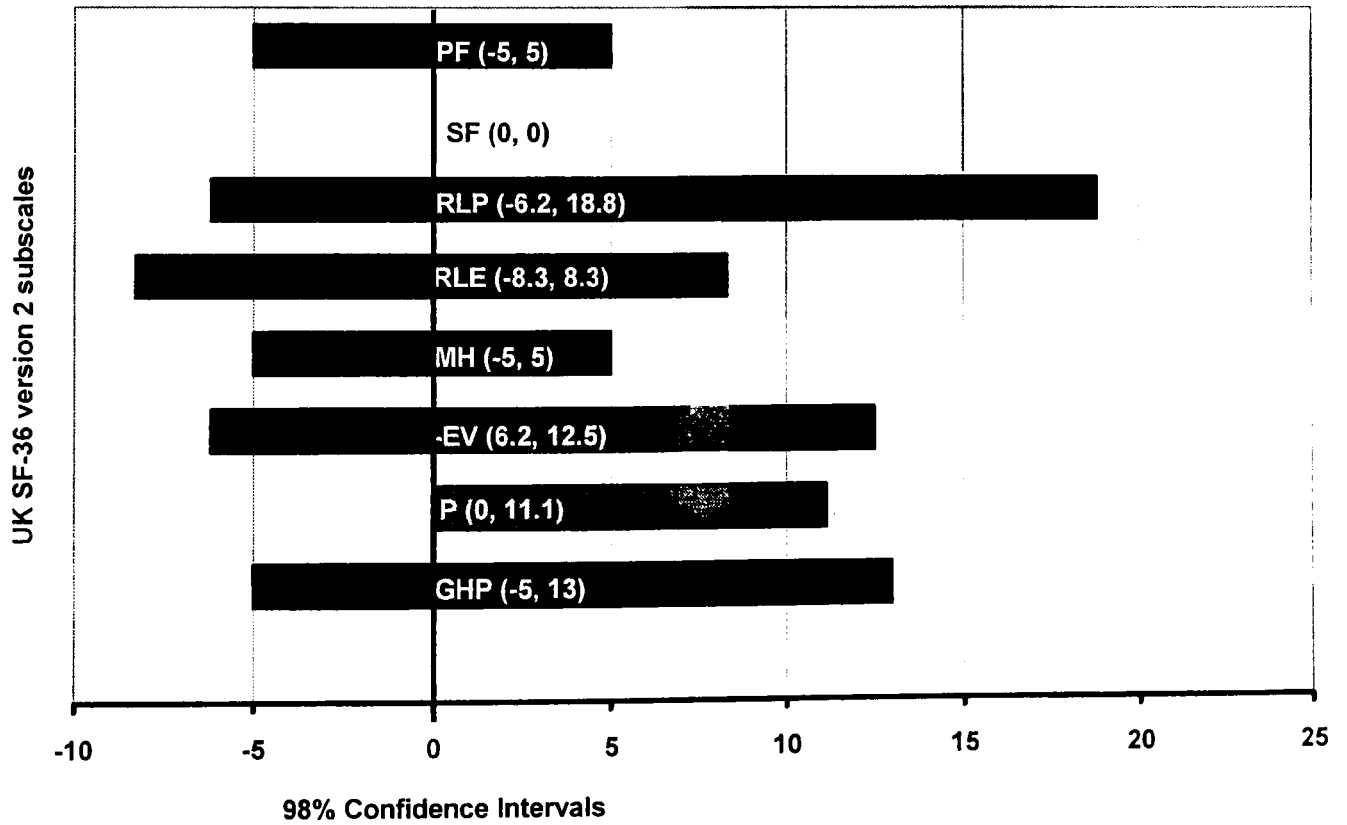


Figure 34 98% confidence intervals for the difference between the groups for the change in quality of life from six to 12 months. Red bars represent borderline significant differences between the groups for changes in SF-36 subscales. Red bars skewed to the right indicate an advantage to the experimental group.

Clinical Outcomes from Baseline to Six and 12 Months

In the experimental group, one patient received coronary artery bypass surgery, three patients were admitted to hospital with unstable angina and subsequently received angioplasty, and one patient was diagnosed with Type 2 diabetes over the 12 month study period. In the control group, two patients died which were classed as sudden cardiac deaths, one control received non-cardiac surgery and one patient was diagnosed with Type 2 diabetes during the study period. Overall, cardiac mortality was 5.7% (2/35) in the control group and 0% in the experimental group. However, the revascularisation rate (i.e. CABG and PTCA) was 11.4% (4/35) in the experimental group and 0% in the control group.

CHAPTER FIVE

DISCUSSION

In total, 85 patients were invited to participate in the study, 15 of these patients declined to take part.

Baseline characteristics of the study group were reported. The majority of patients were men (80%) and had suffered a myocardial infarction (65.8%). Information from the BACR national database on client groups taking part in phase III cardiac rehabilitation programmes are 68% MI and 25% female.[5] Thus, the study sample appears to be representative of people who attend phase III cardiac rehabilitation programmes.

Baseline Physical Activity of Study Sample

This study investigated the effect of an exercise consultation on maintenance of physical activity following completion of a phase III exercise programme. Baseline physical activity levels were assessed within one month of completing phase III. Thus, it was expected that subjects would be physically active at baseline. We found that 46% of the study sample were in the action stage of change, and 37% were in maintenance at baseline. Thus, 83% of patients were regularly physically active following completion of phase III. Similarly, Bock[96] found that after completion of a 12-week phase II supervised exercise programme, 96% of the study group were in the action and maintenance stages, thus were considered regularly active.

At baseline, patients participated in a median 248 minutes/week of moderate intensity physical activity, 26 minutes/week of vigorous activity and 288 minutes/week of total physical activity (i.e. moderate and vigorous activity combined) measured by the 7DPAR. Thus, patients were exceeding the 1995 ACSM/CDC physical activity guidelines of 150 minutes/week of moderate intensity activity.[8] Bock[96] reported that patients participated in 10.98 hours/week of moderate to vigorous physical activity measured by the 7DPAR after completion of a 12-week phase II exercise programme. Thus, there was a large difference between physical activity participation in the present study and the results reported by Bock. However, the 7DPAR was self-administered in

Bock's study[96] and was interview-administered in the present study. Administering the questionnaire in an interview format may generate more accurate and reliable information than self-administering the 7DPAR, as interviewing subjects can minimise guessing and over-reporting physical activity participation.[221] In addition, subjects may mis-classify the intensity level of various activities. For example, in the present study some individuals classed ironing in the moderate intensity category because it made them hot and tired, when ironing is clearly a light intensity activity. However, the researcher could clarify if the patient thought that ironing was a similar effort to a brisk walk (i.e. definition of moderate intensity), whereas determining this in a self-administered format is not possible. In a pilot study, we found that patients participated in a median of 555 minutes/week (9.15 hours/week) of moderate to vigorous physical activity shortly after completion of phase III.[178] The Scottish Physical Activity Questionnaire[279] (SPAQ), a self-administered questionnaire, was used to measure physical activity in the pilot study. Results of this pilot study revealed that subjects reported participating in 5 to 6 hours of gardening or decorating at a moderate to vigorous intensity, however it is unlikely that these activities were exclusively moderate to vigorous intensity. Thus, it is possible that individuals over-estimate physical activity participation in self-administered questionnaires more than interview-administered instruments. An alternative explanation for lower self-reported physical activity levels after phase III found in this study compared to other trials is that physical activity was monitored objectively using accelerometers over the same sampling period as the 7DPAR. Thus, individuals may be more likely to report their activities accurately and state the activities they actually performed instead of activities that they may normally do. In addition, subjects completed an activity recording sheet, which was used to assist patients with their recall and prevent subjects from over-estimating the intensity or duration of activities.

The CSA accelerometers recorded a median of 4025432 activity counts/week at baseline in the total group. As previously discussed, CSA recordings can be used to estimate energy expenditure or the time spent in various intensities of physical activity, which would allow investigators to interpret how active their subjects were. However, studies have shown that accelerometers substantially underestimate the energy cost of household, gardening and leisure activities and energy expenditure under free-living conditions.[191;196-198] Thus, researchers have recommended using the raw output from accelerometers (i.e. activity counts) to quantify physical activity rather than converting

the data to estimate energy expenditure. A limitation of using the raw data from accelerometers is that it is not possible to describe how active the study sample was at baseline. In addition, no other studies have measured physical activity using accelerometers after completing exercise-based cardiac rehabilitation. Thus, it is not possible to compare the accelerometer results from this study with findings from other cardiac rehabilitation studies. A study conducted by Kirk et al measured physical activity using the 7DPAR and CSA accelerometers in a group of sedentary patients with type II diabetes.[177] Patients participated in 45 minutes/week of moderate intensity activity measured by the questionnaire, thus these patients were not meeting physical activity guidelines. In addition, CSA accelerometer readings were 2908974 counts/week in this inactive group of patients, which is much lower than the 4025432 counts/week recorded in the current study.

Effect of an Exercise Consultation on Physical Activity

Physical activity was measured using subjective and objective methods. There are advantages and limitations to using each method, thus researchers have recommended using multiple methods to measure physical activity as no single technique is able to quantify all aspects of physical activity.[217] The 7DPAR measures the duration of moderate and vigorous intensity physical activity, thus can determine whether individuals are meeting current physical activity recommendations. Accelerometers provide an objective measure of physical activity, however they do not give meaningful information on patterns of physical activity. Although accelerometers are able to detect if any changes occurred in physical activity over the study period.

At baseline, total physical activity was 300 minutes/week in the experimental group and 275 minutes/week in the control group. Thus, both of these groups were regularly physically active at baseline. No significant change was observed in moderate, vigorous and total physical activity measured by the 7DPAR from baseline to six and 12 months in the experimental group. Similarly, total activity counts/week recorded by the CSA accelerometer did not significantly change from baseline to follow-up in the experimental group. These findings suggest that physical activity was maintained over the study period in the experimental group. At 12 months, the experimental group participated in 360 minutes/week of moderate to vigorous intensity physical activity, thus were clearly exceeding the physical activity guidelines of 150 minutes/week of moderate intensity activity.[8] In the control group, there was a non-significant decline in

total and moderate intensity physical activity measured by the 7DPAR from baseline to six months by 55 minutes/week and 47 minutes/week, respectively. In addition, total physical activity significantly decreased by 115 minutes/week from baseline to 12 months and 63 minutes/week from six to 12 months in the control group. Similarly, there was a significant decrease in moderate intensity activity by 83 minutes/week from baseline to 12 months and a trend for a decrease by 48 minutes/week from six to 12 months in the controls. These results show a significant decline in self-reported physical activity over the study period in the control group. Despite a decrease in self-reported physical activity, at 12 months control patients were participating in 205 minutes/week of moderate to vigorous activity, thus were regularly physically active. Total activity counts/week measured by accelerometry decreased by 5.2% from baseline to six months, 8% from baseline to 12 months and 3% from six to 12 months, although this decline was not significant. Thus, the results suggest that physical activity measured by self-report and CSA accelerometers declined from baseline to six months in the control group. In contrast, a further decrease in self-reported physical activity was observed from six to 12 months, however there was no further decline in CSA accelerometer counts/week. Thus, findings from the CSA accelerometers did not parallel the decrease in self-reported physical activity from six to 12 months in the control group.

Results from the stage of change questionnaire support the results of the 7DPAR. At baseline, the majority of patients in the experimental and control groups were in the action and maintenance stages of change, thus were regularly physically active (88% experimental group vs 77% control group), with no significant difference between the groups. At six months, a similar number of patients in each group were in action or maintenance (84% experimental group vs 69% control group). However at 12 months, a greater proportion of the experimental group were regularly physically active compared to the control group, this difference was near significant (85% experimental group vs 67% control group). Furthermore, the results show that a greater proportion of experimental patients compared to controls remained in action and maintenance or progressed to action or maintenance from baseline to 12 months and six to 12 months. Overall, the results show that physical activity measured by self-report was maintained in the experimental group compared to the control group over the study period. These findings suggest that the exercise consultation was more effective than standard exercise information in maintaining self-reported physical activity for 12

months following completion of a phase III exercise programme. Results from the CSA accelerometers agree with the findings from the self-report method in the experimental group, suggesting that physical activity was maintained in this group for 12 months after phase III. In contrast, although a decline in physical activity measured by CSA accelerometers was observed over the study period in the control group, this decline was not significant and did not parallel the marked decrease in self-reported physical activity.

A significant decrease in self-reported physical activity from baseline to 12 months was observed in the control group. Similarly, a number of studies have reported a decline in physical activity following completion of exercise-based cardiac rehabilitation.[20;56;67;79;91;94-96;98] Bock measured physical activity using the 7DPAR in 62 cardiac patients before and after a 12-week supervised exercise programme and at three months follow-up.[96] On programme completion, the time spent in moderate to vigorous intensity activity had increased significantly. In contrast, three months after rehabilitation, nearly 50% of participants had reduced their physical activity participation compared to the end of the programme. Furthermore, 16% of patients had regressed from the action/maintenance stage to an earlier stage of change. The present study found that 33% of control patients regressed from action or maintenance to preparation or remained in the preparation stage from baseline to 12 months.

The change in total activity counts/week measured by the CSA accelerometer from baseline to 12 months did not parallel the decrease in self-reported physical activity in the control group. Instead, the CSA accelerometer results suggested that physical activity was maintained over the study period in both groups. However, CSA accelerometers have limitations, which could explain these conflicting findings. Accelerometers cannot record all types of activity such as water activities, activities that increase energy expenditure without a proportional increase in bodily acceleration (e.g. walking uphill) and activities that require upper body movement (e.g. washing windows) unless the accelerometer was attached to the wrist. Modest to poor correlations have been observed between activity counts and measured VO_2 during daily lifestyle activities (e.g. housework, gardening and leisure activities).[191;196] Most of these activities required modest leg movement and a large amount of upper body work, which would not be detected accurately using accelerometers attached the ankle. In contrast, the 7DPAR would be able to record these types of daily lifestyle

activities. This reasoning is supported by poor to modest correlations ranging from 0.2 to 0.46 between CSA activity counts/week and self-reported physical activity observed in this study. Thus, the decrease in self-reported physical activity in the control group could reflect a decline in lifestyle physical activity, which would not be recorded by the accelerometers. In addition, it is possible that repeated use of the CSA accelerometers throughout the study may have made them less sensitive to measuring physical activity at 12 months compared to baseline and six-month measurement periods. Results show that the change in CSA accelerometer counts/week and self-reported physical activity from baseline to six months appear to track each other in both groups. However, despite further changes in self-reported physical activity from six to 12 months in both groups, there was no further change in CSA accelerometer readings. There are no studies investigating the effect of repeatedly using CSA accelerometers on the reliability of these instruments for measuring physical activity.

An alternative explanation for the discrepancy between the 7DPAR and CSA results is that the experimental group may have over-estimated the duration and/or intensity of their activities at six and 12 months. The aim of the exercise consultations was to encourage patients to remain regularly active following completion of the phase III exercise programme. Thus, the experimental group may have overestimated their physical activity to demonstrate to the researcher that they had remained active over the study period. Conversely, as the researcher was not blinded to group allocation, she could have unfairly influenced the information collected by the 7DPAR. However, a standardised interview process[222] was followed when administering the questionnaire to limit the interviewer's bias.

This study demonstrated that exercise consultation was more effective than standard exercise information in maintaining self-reported physical activity for 12 months after completion of a phase III exercise programme. Despite the decline in physical activity and other lifestyle changes following completion of phase III documented in several studies, there is limited research on effective interventions to encourage maintenance of physical activity in phase IV. Brubaker[181] randomized 31 patients, who had completed a 12-week phase III supervised exercise programme to either a phase IV home-based maintenance programme or standard care. In addition, a comparison group (n = 17) who attended a phase IV supervised maintenance programme after completion of phase III were randomly selected. The

phase IV home-based programme lasted 9 months and included an initial physical activity counselling session on self-monitoring exercise intensity and daily activity levels, problem solving exercise barriers, and regular telephone contact to discuss exercise compliance, feedback on exercise levels and to provide support and encouragement. This intervention seems to be similar to the exercise consultation used in the present study. Standard care involved advice on continuing with exercise and other lifestyle modifications. The comparison group attended a phase IV supervised maintenance programme for 9 months, involving supervised exercise training three times a week and regular counselling on exercise compliance and modification of other risk factors. Exercise capacity, blood lipids, and body composition were assessed before and after phase III and at 9 months follow-up. However, physical activity levels were not assessed. Results revealed comparable, significant improvements in these outcomes in all 3 groups on completion phase III. In addition, these improvements in blood lipids, exercise capacity and body composition were maintained in all groups at 9 months follow-up. These findings suggest that a home-based exercise programme is just as effective for patients who cannot attend a phase IV supervised maintenance exercise programme. Although the standard care patients received no further intervention in phase IV, they also maintained these changes, suggesting that follow-up testing alone may be sufficient to maintain lifestyle changes after phase III. A number of patients stated that knowing they were being reassessed at 12 months motivated them maintain lifestyle changes. Thus, it is possible that assessing physical activity and cardiorespiratory fitness at regular intervals in the present study could have encouraged patients to remain active over the study period. Without this level of monitoring, a greater decline in physical activity may have occurred in the control group over the study period. In fact, several control patients stated that they were grateful for being invited to take part in the study as it "encouraged them to keep active after they finished phase III".

Effect of an Exercise Consultation on Cardiorespiratory Fitness

Measuring exercise capacity and submaximal endurance capacity was used as indicators of cardiorespiratory fitness.

Exercise Capacity

Cardiorespiratory variables measured at peak exercise were used to determine

exercise capacity. Exercise capacity parameters included peak oxygen uptake (VO_2), total exercise duration, peak ventilation (V_E), peak heart rate (HR), peak respiratory exchange ratio (RER) and peak oxygen pulse (VO_2/HR).

Baseline peak VO_2 corrected for total body weight (ml/kg/min) was similar between the groups. At baseline (i.e. on completion of phase III), peak VO_2 was 26.9 ml/kg/min (SD 6.6) in the experimental group and 24.4 ml/kg/min (SD 6.5) in the control group. It is difficult to compare baseline results in the present study with other studies reporting exercise capacity values on completion of exercise-based cardiac rehabilitation. This is because many studies have estimated peak VO_2 from treadmill test duration,[20;46;127;85;35;41;58;88;47;66] which can overestimate exercise capacity by up to 40%.[57] In addition, several studies determined exercise capacity by measuring watts achieved during cycle testing.[36;37;50;51;53;55;56;67] Furthermore, findings from the present study cannot be compared with trials that directly measured peak VO_2 using cycle testing, as exercise capacity is 10 to 15% lower in cycle versus treadmill testing. In contrast, a few studies have assessed exercise capacity by directly measuring peak VO_2 during treadmill testing.[48;49;57;59;69;82;99;121;280;281] However, various treadmill protocols were used in these trials, which influences the measurement of peak oxygen uptake.[237] Although, several of these studies reported similar values for peak VO_2 after three to five months of exercise training as the baseline results found in the present study.[48;49;99;121;281]

There was a trend for a decrease in peak VO_2 (ml/kg/min) in experimental group by 3.7% and a statistically significant decrease by 6.7% in the control group from baseline to six months. From baseline to 12 months, peak VO_2 significantly decreased by 6.7% (1.8ml/kg/min) in the experimental group and 9.4% (2.3 ml/kg/min) in the control group. These findings suggest that exercise capacity was not maintained over the 12-month study period in either group. There was no significant change in body weight from baseline to follow-up in either group and peak VO_2 (ml/min), not corrected for total body weight, also decreased significantly from baseline to 12 months in the experimental and control group. In contrast to the decline in peak VO_2, total exercise duration did not significantly change from baseline to six and 12 months in either group.

Possible explanations for the decrease in peak VO_2 observed from baseline to 12 months in both groups are discussed. Firstly, improvements in peak aerobic capacity with exercise training may be partly due to a reduction in body fat, which overestimates the true improvement in exercise capacity with training.[280] Thus, it has been suggested that changes in peak VO_2 corrected for lean body mass should be used to assess the individual effect of exercise conditioning alone.[280] Body composition was not assessed in the present study, but it is possible that changes in body composition could have partially caused the decline in exercise capacity after completion of phase III exercise training. Secondly, the decline in peak VO_2 could not be caused by a decrease in motivation during follow-up exercise tests compared with baseline, as there was no significant change in total exercise duration over the study period. In addition, peak respiratory exchange ratio (RER), an indicator of exercise effort, did not decrease in either group over the study period. Finally, it is likely that the decrease in peak VO_2 reflects a decline in the capacity of the cardiovascular system to supply oxygen to exercising skeletal muscles and the potential for oxygen extraction by the exercising muscle. As previously discussed, improvements in exercise capacity after training in CHD patients is primarily due to peripheral adaptations rather than central (cardiac) adaptations.[70-73] Peripheral changes reflect an improvement in the supply of oxygen to active skeletal muscles, and enhanced oxygen extraction and utilisation by the exercising muscles. Thus, it is likely that a decrease in peak VO_2 is a result of a deterioration in these peripheral adaptations. Peak oxygen pulse (VO_2/HR ml/beat) was calculated in the present study, which measures the amount of oxygen carried in each stroke volume and gives an index of cardiac function. There was no significant change in peak oxygen pulse in either group over the study period. This finding further supports the concept that a decline in peak VO_2 was not due deterioration in cardiac function, but rather due to negative changes in the skeletal muscles.

This study found a decline in peak VO_2 by 6.7% (1.8ml/kg/min) in the experimental group and 9.4% (2.3 ml/kg/min) in the control group from baseline to 12 months, suggesting that aerobic fitness decreased in both groups after completion of phase III. Other studies have found that improvements in exercise capacity with cardiac rehabilitation exercise training decline after programme completion.[37;50;55;67] As these studies did not directly measure peak VO_2 during treadmill testing, it is difficult to compare the decline in exercise capacity observed in the present study with findings

from other trials. Thus, it is not clear whether the magnitude of the decrease in peak VO_2 is clinically important. However, several studies have reported a significant relation between exercise capacity and mortality in CHD patients.[91;282-285] Researchers have found that a low fitness level (< 4 METs) measured before entry to exercise-based cardiac rehabilitation is a significant predictor of total and cardiac death.[91;282;283;285] Recently, Kavanagh[283] examined the relation between mortality and peak VO_2 measured on entry to a cardiac rehabilitation exercise programme in 12169 men after MI, CABG and CHD. The study found that estimated 15 year survival rates for patients with peak VO_2 values of <15, 15 to 22 and >22 ml/kg/min were 65%, 81% and 88%, respectively. Thus, estimated 15 year prognosis was substantially lower for patients in the least fit group (<15 ml/mg/min; 35% mortality) compared to the middle group (15 to 22 ml/kg/min, 19% mortality) and patients with the highest peak VO_2 (>22 ml/kg/min; 12% mortality). Results from these studies suggest that a fitness level of 15 ml/kg/min, which is equivalent to 4 METs, is an important cutoff for mortality risk. Thus, it may be that a decline in exercise capacity is not clinically important as long as patients maintain their exercise capacity above 15 ml/kg/min (4 METs) in the long-term. Despite a decrease in peak VO_2 over 12-months in the present study, the average peak VO_2 values at 12 months were 25.1 (SD 6.2) ml/kg/min in the experimental group and 22.0 (SD 6.1) ml/kg/min in the control group.

These studies also examined the relation between the change in peak VO_2 and mortality.[282-284] Kavanagh found that a 1ml/kg/min advantage in peak VO_2 was associated with a 9% improvement in prognosis. Vanhees[282] investigated the relation between mortality and the change in peak VO_2 with exercise-based cardiac rehabilitation in a group of MI and CABG patients. Peak VO_2 increased by 33% after the three-month exercise programme. Thirty seven (9%) patients died during the 6-year follow-up period, 21 (5%) died from cardiovascular causes. Total and cardiovascular mortality was significantly related to both the change in peak VO_2 with training and the peak VO_2 after training, which was independent of age and other significant covariates. In addition, the study found that a 1% increase in exercise capacity with training was associated with a 2% reduction in cardiovascular mortality, which was independent from the exercise capacity before training. Dorn also showed that an increase in exercise capacity reduces total and cardiovascular mortality.[284] This study found that each 1 MET increase in exercise capacity from baseline to the end of a three year trial of exercise-based cardiac rehabilitation was

associated with a significant reduction in all-cause and cardiac mortality throughout the 19-year follow-up period. These results included all patients who increased exercise capacity by at least 1 MET, regardless of initial exercise capacity level and group assignment (i.e. control group or rehabilitated group). Thus, findings from these studies suggest that small increases in exercise capacity have a marked effect on survival. Thus, it is possible that the decline in peak VO_2 by 6.7% (1.8 ml/kg/min or 0.5 METs) the experimental group and 9.4% (2.3 ml/kg/min or 0.7 METs) in the control group over the 12 month follow-up period in the present study may be clinically relevant.

Results revealed that although there was a decline in peak aerobic capacity, total exercise duration was maintained over the study period in both groups. The following reason is a possible explanation for the discrepancy in results between peak VO_2 and total exercise duration. Peak VO_2 measures aerobic capacity, which represents the capacity of the cardiovascular system to supply oxygen to exercising skeletal muscles and the potential for oxygen extraction by the exercising muscle. In contrast, total exercise duration measures total work performed without differentiating between aerobic and anaerobic metabolism. Thus, a decrease in peak VO_2 from baseline to 12 months indicates a decline in aerobic capacity. It is possible that total exercise duration was maintained over the study period because patients performed a greater amount of work anaerobically at 12 months compared with baseline. This concept is supported by a significant increase in RER at follow-up compared with baseline in both groups, which may indicate greater lactate production at peak exercise.

Results from the present study suggest that physical activity was maintained from baseline to 12 months in the experimental group, whereas exercise capacity significantly declined over the 12 month follow-up. The following reason may be an explanation for the discrepancy in results between physical activity and fitness. Studies in healthy adults suggest that to maintain initial improvements in aerobic fitness, exercise must be continued on a regular basis, as a significant reduction in cardiorespiratory fitness occurs after 2 weeks of stopping training. Although the minimal level of exercise necessary to maintain fitness is not known, evidence suggests that missing an exercise session periodically or reducing the frequency and duration of training will not adversely affect cardiorespiratory fitness as long as training

intensity is maintained.[7] A study found that if training intensity remained unchanged, cardiorespiratory fitness was maintained for up to 15 weeks when the duration and frequency of training was reduced. On the other hand, when the duration and frequency of training was held constant and the intensity was reduced there was a significant reduction in cardiorespiratory fitness. Thus in the present study, it is possible that the intensity of patients' physical activity was lower than the intensity of exercise training in phase III, which resulted in a decline in peak VO_2, whilst the level of physical activity was maintained. At the time of the study, there were few supervised exercise programmes available in the community for patients to attend. Thus, most of the patients stayed active by participating in moderate intensity activities, such as walking. Alternatively, experimental patients may not have met physical activity guidelines every week throughout the 12-month follow-up period, which may have caused the decline in aerobic fitness. Frequent lapses from activity would not have been detected in the study, as physical activity was only measured three times over 12 months.

Submaximal Endurance Capacity

Submaximal endurance capacity was determined by estimating the oxygen uptake (ml/min) at the lactate threshold. Oxygen uptake at the lactate threshold was 1251 ml/min in the experimental group and 1177 ml/min in the control group at baseline. These results are comparable to other studies reporting VO_2 values at the lactate threshold following participation in exercise-based cardiac rehabilitation.[81;82] Hambrecht found that VO_2 at LT was 1170 ml/min after 12 months of supervised and unsupervised exercise.[84] Although, few studies have evaluated the effect of the exercise-based cardiac rehabilitation on submaximal endurance capacity by measuring the onset of the lactate threshold.

Results revealed a significant increase in the VO_2 at the lactate threshold from baseline to six and 12 months in the experimental group relative to the control group. From baseline to six months, there was a trend for an increase in VO_2 at the LT by 47 ml/min (3.8%) in the experimental group and a decline by 52 ml/min (4.4%) in the control group. Furthermore, the lactate threshold significantly increased by 81 ml/min (6.5%) in the experimental group and decreased by 45 ml/min (3.8%) in the control group from baseline to 12 months. Hambrecht reported a small but significant increase in VO_2 at the LT by 7% (80 ml/min) after 12 months of supervised and unsupervised

exercise training. The magnitude of the increase in the LT is very similar to the results observed in the experimental group in the present trial. Hambrecht also found that VO_2 at LT decreased by 90 ml/min (8%) in the control group from baseline to 12 months, which is greater than the decline observed in the control group in the present study. Other studies have found significant improvements in the lactate threshold after three to six months of exercise training,[61;75;81;82] however they did not examine whether this beneficial effect was maintained after programme completion.

These results indicate that the onset of the LT was delayed in the experimental group compared to the control group from baseline to follow-up. Hambrecht demonstrated that the change in the LT significantly correlated with both changes in physical activity related energy expenditure measured by questionnaire and peak VO_2.[84] In the present study, changes in LT may be a result of continued participation in moderate to vigorous physical activity in the experimental group and a decline in physical activity in the control group. However, peak aerobic capacity declined over the study period in both groups. Studies that reported increases in the LT after exercise-based cardiac rehabilitation also found significant improvements in peak aerobic capacity.[61;81;82;84] This is in contrast to the present study, which found that peak VO_2 declined in both groups over the study period.

The delayed onset of the LT is beneficial to the daily lives of cardiac patients. The lactate threshold is the exercise VO_2 at which the concentration of lactate in arterial blood increases above the resting value due to the supplementation of aerobic energy production with anaerobic metabolism.[83] Below the lactate threshold, exercise at a given work rate can be sustained for a prolonged period of time without fatigue and with relatively little stress to the subject. In contrast, work rates above the lactate threshold lead to metabolic acidosis, an increased ventilatory response and muscle fatigue, thus exercise above this threshold cannot be sustained as long. Therefore, the delayed onset of the lactate threshold enhances an individual's ability to perform physical activity at a given work rate. Proposed mechanisms for a delayed onset of the lactate threshold include improved oxygen supply to active skeletal muscles, increased oxidative capacity of mitochondria and delayed activation of type II muscles fibres that function anaerobically.[83] However, positive changes in these peripheral mechanisms as an explanation for the increased LT in the experimental group relative to the control group seems unusual since peak aerobic capacity declined in both groups over the

study period, suggesting that negative changes occurred in peripheral mechanisms.

In the present study, the lactate threshold was indirectly estimated using the V-slope and ventilatory equivalent techniques. These methods are valid and reliable techniques for estimating the lactate threshold.[238;83] However, directly measuring the concentration of blood lactate during the exercise test would have confirmed that the changes in the LT in the experimental and control groups were due to changes lactate concentration.

Effect of an Exercise Consultation on the Processes of Exercise Behaviour Change

The processes of change are strategies that individuals use when changing their exercise behaviour. The frequency of using the five experiential processes (consciousness raising, social liberation, dramatic relief, environmental re-evaluation and self re-evaluation) did not significantly change from baseline to six months and six to 12 months in either group. From baseline to 12 months, there was a significant decrease in the use of dramatic relief in the control group and environmental re-evaluation in the experimental group. The use of the five behavioural processes (helping relationships, stimulus control, counter conditioning, self-liberation, reinforcement management) did not significantly change from baseline to follow-up in either group. Thus, nine out of ten processes of change remained stable over the study period in both groups. The following reasons may explain the lack of change in process use observed in this study. A meta-analysis[159] by Marshall and Biddle found that the use of experiential and behavioural processes increased with advancing stage of change, with the largest increase occurring from precontemplation to contemplation and preparation to action. Similarly, Marcus found that the transition from an inactive stage (i.e. precontemplation or contemplation) to an active stage (i.e. preparation, action or maintenance) was associated with a significant increase in the use of experiential and behavioural processes. In the present study, there were no patients in precontemplation or contemplation (i.e. inactive stages) at baseline or follow-up. Secondly, few patients in either group progressed from preparation to the action or maintenance stages.

The meta-analysis also found minimal change in the use of experiential and behavioural processes as individuals progressed from action to maintenance.

Likewise, Marcus found that process use did not significantly change for subjects who remained in an active stage (i.e. preparation, action or maintenance). In the present study, the majority of patients in both groups remained in the action and maintenance stages (i.e. regularly active) throughout the study period. Marcus also reported that subjects who relapse over the study period (regression from preparation, action or maintenance to either contemplation or precontemplation) reported a significant decline in the use of all behavioural processes and 1 experiential process (dramatic relief). Similarly, Bock found that individuals who had regressed three months after completion of a phase II programme had significantly lower scores for the frequency of using behavioural processes compared to participants who remained active at three months. In the present study, few patients in either group regressed from action or maintenance to the preparation stage of change. In addition, patients that did regress, moved to a somewhat active stage (i.e. preparation), whereas in Marcus's study, patients relapsed from an active to inactive stage.

It would have been interesting to conduct subgroup analysis to determine whether changes in process use occurred in patients that regressed and progressed through the stages of change during the study period. However, this could not be carried out due to the small number of patients in these categories. Overall, the exercise consultation compared to standard care had little effect on the frequency of using the experiential and behavioural processes of change. This lack of change in process use may be because the majority of patients in both groups remained in the action and maintenance stages of change throughout the study period. Although, a near significant difference was observed in the proportion of the experimental group in the action and maintenance stages of change at 12 months compared to the control group (85% experimental group vs 67% control group). In addition, a significantly greater proportion of patients in the experimental group compared to the control group remained regularly active or became regularly active from baseline to 12 months and six to 12 months. However, the magnitude of these differences were probably not sufficient to produce significant changes in process use.

Results from the present study and previous trials suggest that maintenance of physical activity does not require further changes in the use these processes. Findings from previous trials suggest that continued use of behavioural processes may be important to prevent relapse. Therefore, interventions to maintain physical activity after

completion of exercise-based cardiac rehabilitation should encourage people to increase the frequency of using the behavioural processes of change. For example, encouraging individuals to use reminders, rewards or seek support to help them remain active in the long-term. These strategies are incorporated into the exercise consultation.

Effect of an Exercise Consultation on Weight and BMI

There were no significant between or within group changes recorded in weight or BMI over the study period in either group. At baseline, 28.5% of the experimental group and 17.1% of the control group were obese (BMI>30kg/m^2). In addition, the proportion of overweight patients (BMI>25kg/m^2) in the experimental and control group was 82.9% and 77.1%, respectively. The number of overweight and obese patients did not significantly change in either group from baseline to follow-up. Thus the exercise consultation had no effect on body weight and body mass index compared to standard exercise information. The following reasons are possible explanations for the lack of significant changes in these variables observed in the present study. A systematic review found that the majority of studies reporting improvements in body weight and BMI with exercise-based cardiac rehabilitation included dietary advice in addition to exercise training.[1] This review stated that exercise only rehabilitation had an inconsistent effect on weight management. Thus, combining dietary modification and exercise training is recommended to optimise weight management. In the present study, the intervention did not include advice or counselling on diet in addition to physical activity, which may explain the lack of effect on weight and BMI. However, all patients participating in the present trial would have received information on healthy eating in phases I to III.[3] In addition, overweight patients would have received dietary advice for weight management in accordance with national guidelines.[3] Thus, the majority of participants in both groups may have been on a weight reducing diet. However, this study did not attempt to encourage patients to adhere to dietary changes after completion of phase III. In addition, it was not possible to assess whether patients continued to make dietary changes after completing phase III as eating habits were not measured. It is also possible that changes in body composition, specifically fat mass and fat free mass could have occurred, which would not be detected by measurement of body weight and BMI.

Effect of an Exercise Consultation on Lipids

No significant changes in total cholesterol, triglycerides and LDL cholesterol were observed from baseline to follow-up in either group. The literature suggests that exercise programmes have little impact on blood lipids, unless patients have abnormal lipid levels before exercise training.[28] In this study, baseline levels for total cholesterol, triglycerides and LDL cholesterol in both groups were in the normal range recommended by SIGN,[2] which may explain the lack of change in these variables over the study period.

Evidence suggests that a combination of diet, exercise training and lipid-lowering medication produces favourable lipid profiles and reaches lipid targets.[9] Guidelines state that lipid lowering medication should be prescribed to individuals with a total cholesterol $\geq$ 5mmol/l or LDL cholesterol $\geq$ 3 mmol/l.[2;115] Although, results from the Heart Protection Study indicate that all CHD patients should be prescribed lipid lowering medication irrespective of their initial cholesterol level.[119] Patients participating in the present study would have had their lipid levels assessed in phase I and received lipid-lowering medication if required. In addition, blood lipids would have been re-assessed during phase III and lipid lowering medication would have been altered if values were above national targets. In the present study, 88.6% of the control group and 100% of the experimental group were taking lipid-lowering medication on completion of phase III, which did not significantly change at six and 12 months follow-up. In addition, lipid levels were within normal ranges throughout the study period. Thus, the lack of change in lipid levels observed in this study may be because patients' blood lipids were very well controlled before entry to the trial.

This study did find an increase in HDL from baseline to follow-up in both groups. However, the only significant improvement in HDL occurred from baseline to 12 months in the experimental group. In addition, there was a significant improvement in the ratio of total cholesterol to HDL from baseline to six and 12 months in the experimental group, whereas no significant change was recorded in the control group. A meta-analysis[28] reported that exercise-based cardiac rehabilitation did not improve HDL cholesterol compared with usual care, although this finding was based on only three studies. Similarly, a systematic review observed that studies using exercise only rehabilitation or comprehensive rehabilitation documented inconsistent effects on HDL choletserol.[1] Conversely, other studies[61,121] have reported significant improvements in

HDL cholesterol with three to six months of moderate intensity exercise training. Brochu[121] found a significant increase in HDL cholesterol by 8% after a three month exercise programme, with no change in the other lipid levels. In the present study, HDL cholesterol increased significantly by 10% (0.11 mmol/l) in the experimental group and non-significantly by 5% (0.06 mmol/l) in the control group from baseline to 12 months. However, the difference between the groups for the change in HDL was not significant. The median values for HDL cholesterol were within the normal range (>0.91 mmol/l) in both groups at baseline and follow-up. Thus, the clinical importance of a 10% increase in HDL cholesterol observed in the experimental group is not clear. It is likely that patients with low levels of HDL cholesterol at baseline would benefit more from a 10% increase in HDL cholesterol at follow-up. However, subgroup analysis was not possible in this study due to the small number of patients.

There is evidence to suggest that changes in HDL cholesterol may be influenced more by changes in physical activity than fitness. Brochu[121] found no association between the change in peak VO_2 and changes in coronary risk factors, including HDL cholesterol. Furthermore, Hambrecht[84] reported that improvements in lipids were more closely correlated with increases in total energy expenditure than changes in peak VO_2.

Effect of an Exercise Consultation on Psychological Function

Anxiety and Depression

Anxiety and depression was measured using the Hospital Anxiety and Depression Scale. The scores for the anxiety and depression subscales can be used to classify individuals into normal (0-7), mild (8-10), moderate (11-14) and severe (≥15) categories.

At baseline, median scores for anxiety were 6 in the experimental group and 5 in the control group. Similarly, median depression scores at baseline were 2 in each group. Thus, low levels of anxiety and depression were reported following completion of the phase III exercise programme. Several studies have shown that participation in exercise-based cardiac rehabilitation significantly improves anxiety and depression and produces low levels of psychological distress on programme

completion.[37;38;66;109;110] Various questionnaires were used to measure anxiety and depression in these studies making it difficult to compare findings with the current trial. Lewin[110] found that mean HADS scores were 4.9 for anxiety and 3.1 for depression after six weeks of home-based cardiac rehabilitation. Similarly, Hevey[286] found that following completion of a 10-week programme, mean scores for anxiety and depression were 4.7 and 3, respectively. Thus, baseline scores for anxiety and depression observed in the present study were similar to results reported in previous trials that used the HADS scale to measure anxiety and depression after cardiac rehabilitation. Gradual improvements in anxiety and depression in control patients who received no formal rehabilitation have been demonstrated,[37;59;67] which may be due to the natural course of recovery after a cardiac event. Thus, the low baseline levels of anxiety and depression observed in this study may be because study participants were at least four months post cardiac event and had recently completed a cardiac rehabilitation programme.

Results revealed that anxiety and depression did not significantly change from baseline to six and 12 months in either group. However, a slight increase in depression occurred in the control group compared to the experimental group from baseline to 12 months and six to 12 months, but this difference did not reach significance. Despite this slight increase in depression, scores remained low in the control group at follow-up. Median scores for anxiety at 12 months follow-up were 5 in the experimental group and 4 in the control group. Similarly, depression scores were 2 in both groups at 12 months. Thus, the results show that low levels of anxiety and depression were maintained throughout the 12-month study period in both groups. Other studies have shown that improvements in anxiety and depression with exercise-based cardiac rehabilitation were maintained 6 to 12 months after programme completion.[37;67;110] Lewin reported a mean score of 5 for anxiety and 3.4 for depression 12 months after participation in a six week home-based rehabilitation programme.

Studies have demonstrated that the greatest psychological benefits of exercise-based cardiac rehabilitation occur in patients with high levels of anxiety or depression at study entry.[108;110;112;113] Thus, the lack of change in anxiety and depression observed in the present study may be because median scores for these variables were low at baseline. Thus, subgroup analysis was performed to determine if changes in anxiety and depression occurred in patients who had high baseline levels of psychological

distress, defined by a HADS score of 8 or more on anxiety or depression subscales. Results revealed that 33% of the study sample were psychologically distressed at baseline. In addition, 25% (9/35) of the control group and 40% (14/35) of the experimental group had anxiety or depression scores of 8 or more. In the distressed group, baseline scores for anxiety were 9 in the experimental group and 10 in the control group, which corresponded to mild anxiety according to the HADS classification. Anxiety scores did not significantly change from baseline to follow-up in either group, suggesting that mild levels of anxiety persisted in both groups over the study period. These findings suggest that the exercise consultation had no significant effect on anxiety compared to standard exercise information in patients who were psychologically distressed at study entry. However, the number of distressed patients may have been too small to detect between or within group differences in anxiety. The median score for depression was 6 in the control group and 2.5 in the experimental group, which is in the normal range. Results show that depression did not significantly change from baseline to six and 12 months in either group. However, there was a slight increase in depression in the experimental group from six to 12 months, although this did not reach significance. Despite this slight increase in depression in the experimental group, depression scores remained low at six and 12 months follow-up.

Patients with a baseline HADS score less than 8 for anxiety or depression were classed as non-psychologically distressed. As expected, median scores for anxiety and depression at baseline were low in both groups and did not significantly change from baseline to follow-up in either group. There was a borderline significant increase in depression from six to 12 months in the control group, although depression scores remained low at six and 12 months follow-up. Thus, low levels of anxiety and depression were maintained for 12 months in patients who were not psychologically distressed at study entry.

A randomised controlled trial[110] evaluated the effect of a 6-week home-based programme on anxiety and depression measured using the HADS scale at 6 weeks, 6 months and 1 year. The study found that 52% of the study group had scores of 8 or more on anxiety or depression subscales measured three days post MI. In the non-distressed group, mean anxiety and depression scores were low at baseline and did not significantly change at 12-months in the rehabilitation or control groups. These

results were similar to findings from the present study. In the distressed group, mean anxiety and depression scores significantly decreased in the rehabilitation group, with no change in the control group from baseline to 6 weeks, 6 months and 1 year. These findings conflict with the results of the present trial, which reported no significant change in anxiety or depression in patients who were distressed at baseline. Lewin found that mean anxiety scores declined from 10 at baseline to six at 12 months in the rehabilitation group and remained at 10 from baseline to 12 months follow-up in the control group. In the distressed group in the present study, median scores for anxiety declined from nine at baseline to seven at 12 months in the experimental group and increased from 10 to 11 in the control group. Thus similar to the results by Lewin, anxiety levels declined in the experimental group compared to the control group from baseline to 12 months, however this difference was not significant. In addition, anxiety decreased from the mild to normal category in the experimental group and remained in the mild category in the control group. However, the small number of patients that were psychologically distressed at baseline may have prevented any between or within group differences from becoming significant.

Quality of Life

Quality of life was assessed using the UK SF-36 version 2, which measures 8 dimensions of quality of life (physical functioning, social functioning, role limitations due to physical problems, role limitations due to emotional problems, pain, energy/vitality, mental health and general health perception. At baseline, median scores for social function and role limitations due to emotional problems were 100 in both groups, indicating best possible health. Scores for physical function, role limitations due to physical problems, mental health and pain ranged from 80 to 90 in each group. In addition, median values for energy/vitality and general health perception were between 60 to 70 in each group. These results indicate that quality of life was high at baseline in both groups. Jenkinson[259] published normative data for a UK general population for the questionnaire that was used in the present study. This study found that mean scores for four out of eight subscales ranged from 80 to 90 in the general population. Furthermore, values for three subscales ranged from 70 to 80, and the score for the energy/vitality subscale was 58 in this group. Thus, median scores on several SF-36 subscales observed in the present study were similar to normative data for the general population. Furthermore, values were higher in the present study compared to the general population for social function, role limitations due to emotional problems,

energy/vitality and pain.

Several studies have used the SF-36 to measure quality of life before and after participation in exercise-based cardiac rehabilitation.[61;80;89;108;286] However, these studies did not use version 2 of this questionnaire. Recently, Hevey[286] found that on completion of a 10 week rehabilitation programme, median scores ranged from 80 to 90% for most SF-36 subscales except role limitations due to physical problems (45%) and energy/vitality (68%). Thus, comparable quality of life scores were reported between Hevey's study and the present trial, except that general health perception was lower and role limitations physical was higher in the present study. Similarly, Morrin[89] and Ades[80] reported high quality of life scores ranging from 70 to 90% following participation in three months of supervised or home-based cardiac rehabilitation. In addition, Morrin[89] demonstrated that most quality of life scores were comparable for cardiac rehabilitation participants and normative data from the US.

The present study found no significant changes in the eight quality of life dimensions from baseline to follow-up in either group. These results suggest that high levels of quality of life were maintained in both groups throughout the study period. However, in the experimental group there was a trend for an improvement in four subscales including social function, role limitations due to physical problems, role limitations due to emotional problems and energy/vitality, and a deterioration in pain, although these changes did not reach significance. In the control group, there was a trend for a decline in role limitations due to emotional problems, energy/vitality, and physical function from baseline to follow-up, again these changes were not significant. Conversely, there was a borderline improvement in the pain subscale from six to 12 months in both groups. Additionally, there was a trend for a greater improvement in three dimensions in the experimental compared to the control group from baseline to follow-up; social function, role limitations due to emotional problems and energy/vitality.

Several trials have found that improvements in quality of life with exercise-based cardiac rehabilitation are maintained six to 12 months after programme completion.[37;59;79] In addition, two of these studies[59;79] found that exercise capacity and physical activity was also maintained 12 months after programme completion. Whereas, Oldridge[37] reported a decline in exercise capacity at 12 months follow-up,

despite quality of life scores remaining unchanged. Stahle[67] found significant improvements in several quality of life dimensions measured using a disease-specific questionnaire after a three-month rehabilitation programme. In contrast to other trials, Stahle found that some of these benefits had deteriorated at 12 months. In addition, physical activity levels and fitness had also declined from programme completion to 12 months follow-up. Similarly, Stern[66] found that improvements in several quality of life measures after a three-month rehabilitation programme had declined at 12 months. However, the improvement in exercise capacity was maintained at 12 months. Overall, conflicting results have been reported for changes in quality of life after completion of exercise-based cardiac rehabilitation. In addition, the change in quality of life did not parallel the change in fitness or physical activity in many of these studies. Similarly, the change in quality of life did not correspond with the change in exercise capacity or physical activity in the present trial. Newton reported that improvements in psychological functioning with exercise-based cardiac rehabilitation were independent of changes in exercise capacity.[38] Thus, it is possible that quality of life may not be influenced by fitness or physical activity, which may explain the differing results found for quality of life, fitness and physical activity observed in the present trial. Alternatively, a variety of disease-specific and generic instruments were used to measure quality of life in the previous trials, which may explain the contradictory results.

The present study did find a trend for an improvement in several quality of life dimensions in the experimental group and deterioration in a few SF36 subscales in the control group, although these changes did not reach significance. These lack of significant changes in quality of life may be because a generic instrument was used to measure quality of life. This instrument may not have been sensitive to changes that an individual with CHD experiences as his or her condition improves or deteriorates. However, studies have shown that disease-specific questionnaires are not responsive to change, whereas the SF-36 and the HADS were sensitive to change in cardiac rehabilitation settings.[247;249] In addition, many disease-specific instruments focus on a specific cardiac condition, thus would not have been appropriate for the heterogeneous population used in this study. However, it is possible that a disease-specific questionnaire may have been more able to detect changes in quality of life.

Effect of an Exercise Consultation on Mortality and Morbidity

Meta-analyses[31] have consistently found that exercise-based cardiac rehabilitation improves survival and has no significant effect on rates of non-fatal MI, or revascularisation (i.e. CABG or PTCA) compared with usual care, although few trials reported CABG or PTCA as an outcome. In the present trial, mortality was 5.7% (2/35) in the control group and 0% in the experimental group over the 12 month study period. The revascularisation rate (i.e. CABG and PTCA) was 11.4% (4/35) in the experimental group and 0% in the control group. However, the present trial[28;30] was not powered to detect any differences in mortality and morbidity between the experimental and control groups.

Summary and Conclusions

Participation in exercise-based cardiac rehabilitation produces many physiological and psychological benefits. In addition, sustaining these benefits requires maintenance of physical activity and other lifestyle changes in the long-term. However, evidence suggests that improvements in exercise capacity, physical activity and quality of life are not maintained following completion of rehabilitation programmes. However, the research is limited on effective interventions to encourage maintenance of physical activity in the long-term during phase IV. Physical activity counselling and exercise consultation, based on theoretical models of behaviour change, use strategies to increase and maintain physical activity. Research has shown that these interventions are effective for promoting and maintaining physical activity in the general population and clinical populations.[168;171;175-177;181] In addition, a pilot study found that the exercise consultation improved short-term (4 weeks) adherence to physical activity after completion of a phase III exercise programme.[178]

The research in this thesis evaluated the effect of an exercise consultation on maintenance of physical activity measured subjectively by self-report and objectively by accelerometry for 12 months after completion of phase III. This study demonstrated that exercise consultation was more effective than standard exercise information in maintaining self-reported physical activity for 12 months after completion of a phase III exercise programme. However, the objective measure of physical activity did not support these findings. This study found that the change in CSA accelerometer readings over the 12-month study period did not parallel the decrease in self-reported physical activity observed in the control group. Instead, the CSA accelerometer results

suggested that physical activity was maintained over the study period in both groups. Reasons for the lack of agreement between these outcomes have been discussed previously.

This study also determined the effect of the exercise consultation on measures of cardiorespiratory fitness. Small significant decreases in peak VO_2 from baseline to 12 months were observed in both groups. Thus, the exercise consultation was not effective in maintaining aerobic fitness for 12 months after completion of phase III. Reasons for the decline in peak VO_2 and the discrepancy between the results for physical activity and fitness in the experimental group are discussed. In contrast, the study found an improvement in the lactate threshold, which is an index of submaximal endurance capacity, in the experimental group compared to the control group from baseline to follow-up.

The effect of this intervention on processes of exercise behaviour change, blood lipids and psychological function was also assessed. Results revealed that the exercise consultation compared to standard exercise information had no significant effect on processes of exercise behaviour change, lipid profile and psychological function. However, these variables were normal at baseline, as patients had recently completed a phase III cardiac rehabilitation programme.

Finally, we expected to find greater differences in the study outcomes between the groups. However physical activity and other variables did not decline in the control group as much as was anticipated. It is possible that assessing physical activity, cardiorespiratory fitness and other variables at regular intervals in experimental and control patients could have encouraged them to remain active over the study period. Without this level of monitoring, a greater decline in physical activity may have occurred in the control group over the study period. In fact, several patients in the control group stated that they were grateful for being invited to take part in the study as it "encouraged them to keep active after they finished phase III".

Study Limitations

The limitations of the instruments used to measure the study outcomes have already been discussed. The main limitation of this research is related to time constraints of doctorate study. The exercise consultation was evaluated over a 12-month period.

However, the effect of the exercise consultation on maintenance of physical activity and other outcomes should be studied over a longer follow-up period. In addition, several variables were also maintained over the 12-month study period in the control group, which explains the lack of effect of the exercise consultation on some outcomes. However, a more substantial deterioration in these variables may have been observed in the control group over a longer time period.

Seventy patients were entered into the trial and only a small number of participants dropped out over the follow-up period. The primary aim of the study was to determine the effect of the exercise consultation on maintenance of physical activity. Several physical activity counselling trials in the general population based their sample size calculation on physical activity measured by the Stanford Seven-Day Recall.[171-173] These trials reported that more than 100 individuals were required to detect differences between groups. However, it would not have been possible to recruit this number of patients in the present study. Thus, the power calculation was based on physical activity measured by accelerometry. No studies have evaluated the effect of exercise-based cardiac rehabilitation on physical activity measured using accelerometers. However, a pilot study compared the effect of an exercise consultation with standard care on physical activity measured using accelerometry in a group of people with type II diabetes.[263] Thus, the difference between the experimental and control group for the mean change accelerometer readings was used to calculate the sample size. This calculation found than 50 patients in total were required to detect a 22% difference in accelerometer counts between the experimental and control groups. However, the actual difference between the groups for the change in activity counts from baseline to 12 months was 10% (392946/4025432). Therefore, this trial was not powered to detect small differences between groups. We expected to find greater differences between the groups in physical activity outcomes, however physical activity did not decline in the control group as much as was anticipated, thus the trial was underpowered to detect a difference. In addition, the study did find several trends for differences in quality of life and processes of behaviour change favouring the experimental group. However, the sample size may have been too small to determine if these differences were significant and clinically meaningful. However, if small changes in these outcomes are clinically important then future studies should be powered to detect these changes.

The small number of women in each group means that it was not possible to compare maintenance of physical activity in men and women after completion of phase III. No studies have investigated maintenance of physical activity in men and women following cardiac rehabilitation to gain information on gender differences in this health behaviour.

The same researcher delivered the exercise consultation and measured the outcomes of this study, thus the researcher was not blinded to group allocation when conducting the outcome measures. Therefore, the measurements could have been influenced in favour of the experimental group. However, standardised procedures for data collection were followed in an attempt to control for this limitation. For example, during the exercise tests the Cardiac Clinical Scientific Officer, who was blinded to group allocation, was responsible for encouraging the participants. In addition, questionnaires measuring psychological function and the processes of behaviour change were self-administered. Although the researcher administered the Seven-Day Physical Activity Recall, a standardised interview process[222] was followed to limit the interviewer's bias.

Implementing the Exercise Consultation

The results of this study demonstrate that the exercise consultation may be an effective intervention for maintaining physical activity after completion of phase III hospital-based exercise programmes. Presently, patients completing phase III can attend phase IV maintenance exercise programmes in the community. However, these exercise opportunities are not available in all areas. Furthermore, it is likely that a minority of patients will attend these phase IV programmes, due to barriers associated with supervised exercise training including transportation problems, high costs, limited access, work and domestic conflicts. Thus, the exercise consultation may provide an alternative to supervised exercise in phase IV. Furthermore, the intervention could be used to facilitate patients' progression from phase III hospital-based exercise programmes to community-based programmes or independent exercise. Thus, the exercise consultation could be routinely provided to cardiac patients on completion of phase III to encourage maintenance of physical activity in phase IV.

Is it feasible to incorporate the exercise consultation into current cardiac rehabilitation

services? Firstly, the consultations are relatively inexpensive in terms of time, resources and personnel. Exercise consultations last approximately 20 to 30 minutes, and the support phone calls are five to 10 minutes in duration. In addition, it is possible that patients could record their physical activity habits, and the pros and cons for physical activity before attending the intervention to reduce time during the consultation. Resources required to conduct the exercise consultation include the materials (e.g. goals sheet, and guidelines for physical activity) and a quiet room. The exercise consultation could be delivered by a number of professionals. In the United Kingdom, physiotherapists play a central role in the exercise component of cardiac rehabilitation. Physiotherapists have an ideal opportunity to deliver the exercise consultation to patients entering phase IV as they have good insight into the patient's progress from phases I to III cardiac rehabilitation with regards to physical activity and could incorporate the intervention into existing programmes. The approximate cost of a Senior 1 physiotherapist delivering a 30 minute exercise consultation is £6 to £7.17 per patient. In addition, BACR trained phase IV exercise staff could use the exercise consultation to provide support to patients who are having difficulty remaining active. The approximate cost of a BACR trained phase IV instructor delivering a 30 minute exercise consultation is £4.40 to £4.94 per patient. In order to train in the exercise consultation, individuals need to understand the behaviour change theories on which the consultation is based, and the counselling skills and strategies required to deliver the intervention. This could be achieved by appropriate in-service training. In addition, individuals would need to practice the consultation process.

Currently, the exercise consultation has been included in two courses for individuals involved in cardiac rehabilitation services. The MSc Module in Rehabilitation in Cardiology at Glasgow Caledonian University is aimed at specialist nurses and physiotherapists delivering phase III cardiac rehabilitation programmes. Training for these individuals involves a three-hour lecture on theories of exercise behaviour change, and counselling skills and strategies required to deliver the intervention. There is also a four-hour practical session, where the individuals have the opportunity to practice the exercise consultation process. The exercise consultation has also been incorporated into the British Association of Cardiac Rehabilitation (BACR) Phase IV Training. This course trains exercise instructors to deliver phase IV maintenance exercise programmes in the community for cardiac rehabilitation patients. Due to time constraints, this course involves a two-hour lecture on theories of behaviour change for

exercise and the exercise consultation process.

Future Research

Reviewing the literature on exercise-based cardiac rehabilitation for this thesis has identified several important areas for further research. Numerous studies have assessed physical activity participation for up to 11 years after completion of formal rehabilitation programmes. However, few trials have used a validated questionnaire or an objective method to measure physical activity. In addition, many trials reported the proportion of patients that "exercised regularly", however the definition of regular exercise varied greatly and was not based on current physical activity recommendations. Only one study used the current physical activity guidelines to determine the proportion of patients who remained regularly active after exercise rehabilitation.[96] Thus, more studies are required to accurately assess physical activity levels after completion of exercise rehabilitation and determine the proportion of patients that continue to meet current physical activity guidelines in phase IV.

Cardiac rehabilitation guidelines[2;5] recommend that all centres should audit their cardiac rehabilitation programmes to ensure that standards for cardiac rehabilitation set out by the Clinical Standards Board for Scotland and the National Service Framework for Coronary Heart Disease for England and Wales are being met. These guidelines have recommended a minimum common data set that should be collected by all centres, which could be accessed nationally for audit and evaluation of cardiac rehabilitation. Physical activity outcomes should be included in the data set, which could then be used to determine the proportion of patients that meet current physical activity guidelines in each phase of cardiac rehabilitation.

Many studies have examined the factors influencing uptake and adherence to supervised cardiac rehabilitation exercise programmes. Whereas, the factors that contribute to maintenance of physical activity following programme completion have not been explored. Understanding these factors is an important step in the development of interventions to improve maintenance of physical activity and exercise. Further research in this area is warranted. In addition, few studies have examined the effect of interventions to encourage long-term maintenance of physical activity following completion of phase III cardiac rehabilitation exercise programmes.

Thus, research is needed to test different forms of intervention aimed at improving long-term compliance to physical activity.

There are numerous phase IV maintenance exercise programmes available in the UK. However, the efficacy of these phase IV programmes to maintain the benefits gained during phase III exercise training, including exercise capacity and increased physical activity, has not been assessed. In addition, there is no data available on the proportion of patients who participate in these phase IV maintenance programmes after completing phase III.

The modest success of the exercise consultation in the present study provides the basis for further research with this intervention. Firstly, can the exercise consultation maintain physical activity for more than 12 months? Are repeat exercise consultations required? Could the exercise consultation be delivered successfully in a group or by post, telephone or World Wide Web? The possibility of delivering this intervention to patients in a group setting at the end of phase III is a good area of further study. First of all, delivering this intervention to groups of patients as an alternative to one to one consultations would be more feasible for cardiac rehabilitation services in terms of time and staff resources. In addition, conducting an exercise consultation in a group setting would provide patients with the opportunity to discuss certain issues with each other, such as potential barriers to maintaining activity after phase III, problem solving these barriers and identifying high risk situations for relapse. In addition, group discussion on exercise opportunities in the community, such as phase IV classes, may encourage patients to attend these programmes together. In general, patients routinely receive a discharge interview at the end of phase III, which provides cardiac rehabilitation staff with an ideal opportunity to review the patients' goals for remaining active that were devised during the group consultations. Physical activity counselling trials in the general population and other clinical groups have successfully delivered this type of intervention in a group setting.[171;287]

The exercise consultation may also be useful at the beginning of phase III to improve adherence to the supervised exercise programme and encourage patients to participate in physical activity outwith the exercise classes. A pilot study found that web-based and one to one exercise consultations were equally effective in increasing physical activity measured by the 7DPAR in a group of patients participating in a

phase III supervised exercise programme. Are there any other strategies that could be included in the exercise consultation to increase its efficacy? Recently, physical activity intervention studies and weight management programmes have found pedometers to be effective in promoting physical activity.[209] Thus, pedometers may be a promising strategy to encourage maintenance of physical activity in phase IV. Results from this trial showed that although physical activity was maintained in the experimental group, exercise capacity declined. Thus, should the aim of the exercise consultation in phase IV be maintenance of physical activity or cardiorespiratory fitness?

References

1. US Department for Health and Human Services and Agency for Health Care Policy and Research. Cardiac Rehabilitation. Clinical Practice Guideline Number 17. 1995. Rockville, The Agency. AHCPR Publication Number 96-0672.

2. Scottish Intercollegiate Guidelines Network (SIGN). Cardiac Rehabilitation: A national clinical guideline. (SIGN publication no. 57). 2002. Edinburgh, SIGN.

3. Coats A, McGee H, Stokes H, Thompson D. British Association of Cardiac Rehabilitation (BACR): Guidelines for Cardiac Rehabilitation. Oxford: Blackwell Science Ltd, 1995.

4. Campbell NC, Grimshaw JM, Ritchie LD, Rawles JM. Outpatient cardiac rehabilitation: are the potential benefits being realised? *Journal of the Royal College of Physicians* 1996;**30**:544-9.

5. Scottish Needs Assessment Programme. Provision of Cardiac Rehabilitation Services in Scotland - Needs assessment and guidelines for decision-makers. 2001. Glasgow, Public Health Institute of Scotland.

6. American College of Sports Medicine. The recommended quality and quantity of exercise for developing and maintaining cardiorespiratory and muscular fitness in healthy adults. *Med Sci Sport Exer* 1990;**22**:265-74.

7. American College of Sports Medicine. The recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness, and flexibility in healthy adults. *Med Sci Sport Exer* 1998;**30**:975-91.

8. Pate RR, Pratt M, Blair SN, Haskell WL, Macera CA, Bouchard C *et al.* Physical activity snd public health. A recommendation from the Centres for Disease Control and Prevention and the American College of Sports Medicine. *JAMA* 1995;**273**:402-7.

9. Balady GJ, Ades PA, Comoss P, Limacher M, Pina IL, Southard D *et al.* Core components of cardiac rehabilitation/secondary prevention programs: A

statement for healthcare professionals from the American Heart Association and the American Association of Cardiovascular and Pulmonary Rehabilitation. *Circulation* 2000;**102**:1069-73.

10. Campbell NC, Grimshaw JM, Rawles JM, Ritchie LD. Cardiac Rehabilitation in Scotland: is current provision satisfactory? *Journal of Public Health Medicine* 1996;**18**:478-80.

11. NHS Scotland. Coronary Heart Disease/Stroke Task Force Report. 2001. Edinburgh, Scottish Executive.

12. Bethell, H. J. N., Turner, S. C., Evans, J. A., and Rose, L. Cardiac rehabilitation in the United Kingdom: How complete is the provision? *Journal of Cardiopulmonary Rehabilitation* 2001;**21**:111-115.

13. Thompson DR, Bowman GS, Kitson AL, DeBono DP, Hopkins A. Cardiac rehabilitation services in England and Wales: a national survey. *International Journal of Cardiology* 1997;**59**:299-304.

14. Melville, M. R., Packham, C., Brown, N., Weston, C., and Gray, D. cardiac rehabilitation: socially deprived patients are less likely to attend but patients ineligible for thrombolysis are less likely to be invited. *Heart* 1999;**82**(3):373-377.

15. Bittner, V., Sanderson, B., Breland, J, and Green, D. Referral patterns to a university-based cardaic rehabilitation program. *American Journal of Cardiology* 1999;**83**(2):252-255.

16. Glaser, T. C., Blum, M., Schmeizl, R., Prigerson, H. G., Zaret, B., and Mazure, C. M. Gender differences in referral to cardiac rehabilitation programs after revascularization. *Journal of Cardiopulmonary Rehabilitation* 2001;**21**:24-30.

17. Harlan WR, Sandler SA, Lee KL, Lam LC, Mark DB. Importance of baseline functional and socioeconomic factors for participation in cardiac rehabilitation. *American Journal of Cardiology* 1995;**76**:36-9.

18. Pell JP,.Morrison CE. Factors associated with low attendance at cardiac

rehabilitation. *British Journal of Cardiology* 1998;**5**:152-5.

19. Oldridge NB,.Spencer J. Exercise habits and perceptions before and after graduation or dropout from supervised cardaic exercise rehabilitation. *Journal of Cardiopulmonary Rehabilitation* 1985;**5**:313-9.

20. Fontana AF, Kerns RD, Rosenberg RL, Marcus JL, Colonese KL. Exercise training for cardiac patients: Adherence, fitness and benefits. *Journal of Cardiopulmonary Rehabilitation* 1986;**6**:4-15.

21. McGee HM,.Horgan JH. Cardiac rehabilitation programmes: are women less likely to attend? *British Medical Journal* 1992;**305**:283-4.

22. Cooper A, Lloyd G, Weinman J, Jackson G. Why patients do not attend cardiac rehabilitation: role of intentions and illness beliefs. *Heart* 1999;**82**:234-6.

23. Ades PA, Waldmann ML, Polk DM, Coflesky JT. Referral patterns and exercise responses in the rehabilitation of female coronary patients aged >61 years. *American Journal of Cardiology* 1992;**69**:1422-5.

24. Pell JP, Morrison CE, Blatchford O, Dargie H. Retrospective study of influence of deprivation on uptake of cardiac rehabilitation. *British Medical Journal* 1996;**313**:267-8.

25. Lieberman L, Meana M, Stewart D. Cardiac Rehabilitation: Gender differences in factors influencing participation. *Journal of Women's Health* 1998;**7**:717-23.

26. Evenson, K. R. and Fleury, J. Barriers to outpatient cardaic rehabilitation participation and adherence. *Journal of Cardiopulmonary Rehabilitation* 2000;**20**:241-246.

27. Blackburn, G. G., Foody, J. M., Sprecher, D. L., Park, E., Hansen, C., and Pashkow, F. J. Cardiac rehabilitation participation patterns in a large, tertiary care centre: Evidence for selection bias. *Journal of Cardiopulmonary Rehabilitation* 2000;**20**:189-195.

28. Jolliffe JA, Rees K, Taylor RS, Thomson D, Oldridge N, Ebrahim S. Exercise-based rehabilitation for coronary heart disease (Cochrane Review). *Cochrane Library* 2000; **4**:1-59.

29. Bland M. An introduction to medical statistics. Oxford: Oxford University Press, 1995.

30. Oldridge NB, Guyatt GH, Fischer ME, Rimm AA. Cardiac rehabilitation after myocardial infarction: Combined experience of randomised clinical trials. *JAMA* 1988;**260**:945-50.

31. O'Conner GT, Buring JE, Yusuf S, Goldhaber SZ, Olmstead EM, Paffenberger RS. An overview of randomised controlled trials of rehabilitation with exercise after myocardial infarction. *Circulation* 1989;**80**:234-44.

32. Denollet J,.Brutsaert DL. Reducing emotional distress improves prognosis in coronary heart disease: 9-year mortality in a clinical trial of rehabilitation. *Circulation* 2001;**104**:2018-23.

33. NHS Centre for Reviews and Dissemination.University of York. Cardiac Rehabilitation. Effective Health Care 4(4). 1998.

34. Pollock ML, Schmidt DH. Heart Disease and Rehabilitation. Champaign, IL: Human Kinetics, 1995.

35. DeBusk RF, Houston N, Haskell WL, Fry M, Parker M. Exercise training soon after myocardial infarction. *American Journal of Cardiology* 1979;**44**:1226-9.

36. Bethell HJN,.Mullee MA. A controlled trial of community based coronary rehabilitation. *British Heart Journal* 1990;**64**:370-5.

37. Oldridge NB, Guyatt G, Jones N, Crowe J, Singer J, Feeny D. Effects on quality of life with comprehensive rehabilitation after acute myocardial infarction. *American Journal of Cardiology* 1991;**67**:1084-9.

38. Newton M, Mutrie N, McArthur JD. The effects of exercise in a coronary rehabilitation programme. *Scottish Medical Journal* 1991;**36**:38-41.

39. Bertie J, King A, Reed N, Marshall AJ, Ricketts C. Benefits and weaknesses of a cardiac rehabilitation programme. *Journal of the Royal College of Physicians* 1992;**26**:147-51.

40. Haskell, W. L., Savin, W., Oldridge, N., and DeBusk, R. Factors influencing estimated oxygen uptake during exercise testing soom after MI. *American Journal of Cardiology* 1982;**50**:299-304.

41. Miller N, Haskell WL, Berra K, DeBusk RF . Home versus group exercise training for increasing functional capacity after myocardial infarction. *Circulation* 1984;**70**:645-9.

42. Lavie CJ,.Milani RV. Effects of cardiac rehabilitation and exercise training on exercise capacity, coronary risk factors, behavioral characteristics and quality of life in women. *American Journal of Cardiology* 1995;**75**:340-3.

43. Cannistra LB, Balady GJ, O'Malley CJ, Weiner DA, Ryan TJ. Comparison of the clinical profile and outcome of women and men in cardiac rehabilitation. *American Journal of Cardiology* 1992;**69**:1274-9.

44. O'Callaghan WG, Teo KK, O'Riordan J, Webb H, Dolphin T, Horgan J. Comparative response of male and female patients with coronary artery disease to exercise rehabilitation. *European Heart Journal* 1984;**5**:649-51.

45. Ades PA, Waldmann ML, McCann WJ, Weaver SO. Predictors of cardiac rehabilitation participation in older coronary patients. *Archives of Internal Medicine* 1992;**152**:1033-5.

46. Lavie CJ, Milani RV, Littman AB. Benefits of cardiac rehabilitation and exercise training in secondary coronary prevention in the elderly. *Journal of the American College of Cardiology* 1993;**22**:678-83.

47. DeBusk RF, Miller NH, Superko R, Dennis CA, Thomas RJ, Henry L. A case-management system for coronary risk factor modification after acute myocardial infarction. *Annals of Internal Medicine* 1994;**120**:721-9.

48. Dugmore LD, Tipson RJ, Phillips MH, Flint EJ, Stentiford NH, Bone MF. Changes in cardiorespiratory fitness, psychological wellbeing, quality of life,

and vocational status following a 12 month cardiac exercise rehabilitation programme. *Heart* 1999; **81**:359-66.

49. Dressendorfer RH, Franklin BA, Cameron JL, Trahan KJ, Gordon S, Timmis GC. Exercise training frequency in early post-infarction cardiac rehabilitation: Influence on aerobic conditioning. *Journal of Cardiopulmonary Rehabilitation* 1995;**15**:269-76.

50. Heldal M, Sire S, Dale J. Randomised training after myocardial infarction: Short and long-term effects of exercise training after myocardial infarction in patients on beta-blocker treatment. A randomised, controlled study. *Scandinavian Cardiovascular Journal* 2000;**34**:59-64.

51. Holmback AM, Sawe U, Fagher B. Training after myocardial infarction: Lack of long-term effects on physical capacity and psychological variables. *Archives of Physical and Medical Rehabilitation* 1994;**75**:551-4.

52. Kallio V,.Hamalainen H. Reduction in sudden deaths by a multifactorial intervention programme after acute myocardial infarction. *Lancet* 1979;1091-1094.

53. Hamalainen H, Luurila OJ, Kallio V, Knuts LR, Arstila M, Hakkila J. Long-term reduction in sudden deaths after a multifactorial intervention programme in patients with myocardial infarction: 10 year results of a controlled investigation. *European Heart Journal* 1989;**10**:55-62.

54. Roman O, Gutierrez M, Luksic I, Chavez E , Camuzzi AL, Villalon E. Cardiac rehabilitation after acute myocardial infarction: 9-year controlled follow-up study. *Cardiology* 1983;**70**:223-31.

55. Carson P, Phillips R, Lloyd M, Tucker H, Neophtou M, Buch NJ. Exercise after myocardial infarction: a controlled trial. *Journal of the Royal College of Physicians* 1982;**16**:147-51.

56. Lidell E,.Fridlund B. Long-term effects of a comprehensive rehabilitation programme after myocardial infarction. *Scand J Caring Sci* 1996;**10**:67-74.

57. Froelicher, V., Jensen, D., and Sullivan, M. A randomised controlled trial of

the effects of exercise training after coronary artery bypass surgery. *Archives of Internal Medicine* 1985;**145**:689-692.

58. Wosornu D, Bedford D, Ballantyne D. A comparison of the effects of strength and aerobic exercise training on exercise capacity and lipids after coronary artery bupass surgery. *European Heart Journal* 1996;**17**:854-63.

59. Oldenburg B, Martin A, Greenwood J, Bernstein L, Allan R. A controlled trial of behavioral and educational intervention following coronary artery bypass surgery. *Journal of Cardiopulmonary Rehabilitation* 2001;**15**:39-46.

60. Willoughby, D. S., Roozen, M., and Barnes, R. Effects of aerobic exercise on the functional capacity and cardiovascular efficiency of elderly post CABG patients. *Journal of Aging and Physical Activity* 1997;**5**:87-97.

61. Belardinelli R, Paolini I, Cianci G, Piva R, Georgiou D, Purcaro A. Exercise training intervention after coronary angioplasty: The ETICA trial. *Journal of the American College of Cardiology* 2001;**37**:1891-900.

62. Lisspers, J., Sundin, O., Hofman-Bang, C., Nordlander, R., Nygren, A., Ryden, L., and Ohman, A. Behavioural effects of a comprehensive, multifactorial program for lifestyle change after percutaneous transluminal coronary angioplasty: A prospective, randomized, controlled study. *Journal of Psychosomatic Research* 1999;**46**(2):143-154.

63. Hofman-Bang C, Lisspers J, Norlander R, Nygren A, Sundin O, Ohman A. Two-year results of a controlled study of residential rehabilitation for patients treated with percutaneous transluminal coronary angioplasty. *European Heart Journal* 1999;**1999**:1465-74.

64. Engblom E, Ronnemaa T, Hamalainen H, Kallio V, Vanttinen E, Knuts LR. Coronary heart disease risk factors before and after bypass surgery: results of a controlled trial on mulitfactorial rehabilitation. *European Heart Journal* 1992;**13**:232-7.

65. Naughton J. Role of physical activity as a secondary intervention for healed myocardial infarction. *American Journal of Cardiology* 1985;**55**:21D-6D.

66. Stern, M. J., Gorman, P. A., and Kaslow, L. The group counselling v exercise therapy study: A controlled intervention with subjects following myocardial infarction. *Archives of Internal Medicine* 1983;**143**:1719-1725.

67. Stahle A, Mattsson E, Ryden L, Unden AL, Nordlander R. Improved physical fitness and quality of life following training of elderly patients after acute coronary events. *European Heart Journal* 1999;**20**:1475-84.

68. Stahle, A., Nordlander, R., Ryden, L., and Mattsson, E. Effects of organized aerobic group training in elderly patients discharged after an acute coronary syndrome: A randomized controlled study. *Scandinavian Journal of Rehabilitation Medicine* 1999;**31**:101-107.

69. Ades PA, Maloney A, Savage P, Carhart RL . Determinants of physical functioning in coronary patients: Response to cardiac rehabilitation. *Archives of Internal Medicine* 1999;**159**:2357-60.

70. Bethell HJN. Exercise in Rehabilitation. *British Journal of Sports Medicine* 1999;**33**:79-86.

71. Blumenthal JA, Califf R, Williams S, Hindman M. Continuing medical education cardiac rehabilitation: A new frontier for behavioral medicine. *Journal of Cardiopulmonary Rehabilitation* 1983;**3**:637-56.

72. Finlayson J. The role of exercise in rehabilitation after uncomplicated myocardial infarction. *Physiotherapy* 1997;**83** :517-24.

73. Ades, P. A. and Coello, C. E. Effects of exercise and cardiac rehabilitation on cardiovascular outcomes. *Medical Clinics of North America* 2000;**84**(1): 251-265..

74. Ades, P. A., Waldmann, M. L., Meyer, W. L., Brown, K., Poehlman, E. T., and Pendlebury, W. W. Skeletal muscle and cardiovascular adaptations to exercise conditioning in older coronary patients. *Circulation* 1996;**94**:323-330.

75. Goodman JM, Pallandi DV, Reading JR, Plyley MJ, Liu PP, Kavanagh T. Central and peripheral adaptations after 12 weeks of exercise training in post-coronary artery bypass surgery patients. *Journal of Cardiopulmonary*

Rehabilitation 1999;**19**:144-50.

76. Cottin, Y., Walker, P., Rouhier-Marcer, I., Cohen, M., Louis, P., and Didier, J. P. Relationship between increased peak oxygen uptake and modifications in skeletal muscle metabolism following rehabilitation after myocardial infarction. Journal of Cardiopulmonary Rehabilitation 1996;**16**:169-174.

77. Wright DJ, Williams SG, Riley R, Marshall P, Tan LB. Is early, low level, short term exercise cardiac rehabilitation following coronary artery bypass surgery beneficial? A randomised controlled trial. *Heart* 2002;**88**:83-4.

78. Horgan J, Bethell HJN, Carson P, Davidson C, Julian D, Mayou RA. Working party report on cardiac rehabilitation. *British Heart Journal* 1992;**67**:412-8.

79. Higgins, H., Hayes, R. L., and McKenna, K. T. Rehabilitation outcomes following percutaneous coronary interventions (PCI). Patient Education and Counseling 2001:**43**:219-230.

80. Ades, P. A., Pashkow, F. J., Fletcher, G. F., Pina, I. L., Zohman, L., and Nestor, J. R. A controlled trial of cardiac rehabilitation in the home setting using electrocardiographic and voice transtelephonic monitoring. *American Heart Journal* 2000;**139**(3):543-548.

81. Lavie, C. J. and Milani, R. V. Effects of cardiac rehabilitation and exercise training on peak aerobic capacity and work efficiency in obese patients with coronary artery disease. *American Journal of Cardiology* 1999;**83**(10):1477-1480.

82. Joughin HM, Digenio AG, Daly L, Kgare E. Physiological benefits of a prolonged moderate intensity endurance training programme in patients with coronary artery disease. *South African Medical Journal* 1999;**89**:545-50.

83. Wasserman K, Hansen JE, Sue DY, Whipp BJ , Casaburi R. Principles of exercise testing and interpretation. Williams and Wilkins, 1994.

84. Hambrecht R, Niebauer J, Marburger C, Grunze M, Kalberer B, Hauer K. Various intensities of leisure-time physical activity in patients with coronary artery disease: Effects on cardiorespiratory fitness and progression of

coronary atherosclerotic lesions. *Journal of the American College of Cardiology* 1993;**22**:468-77.

85. Hedback EL, Perk J, Engall J, Areskog N. Cardiac rehabilitation after coronary artery bypass grafting: Effects on exercise performance and risk factors. *Archives of Physical and Medical Rehabilitation* 1990;**71**:1069-73.

86. Schuler G, Hambrecht R, Schilierf G, Grunze M, Methfessel S, Hauer K. Myocardial perfusion and regression of coronary artery disease in patients on a regimen of intensive physical exercise and low fat diet. *Journal of the American College of Cardiology* 1992;**19**:34-42.

87. Kinsey MG, Fletcher BJ, Rice CR, Watson PH, Fletcher GF. Coronary risk factor modification followed by home-monitored exercise in coronary bypass surgery patients: A four year follow-up study. *Journal of Cardiopulmonary Rehabilitation* 1989;**9**:207-12.

88. Heath GW, Maloney PM, Fure CW. Group exercise versus home exercise in coronary artery bypass graft patients: Effect on physical activity habits. *Journal of Cardiopulmonary Rehabilitation* 1987;**7**:190-5.

89. Morrin, L., Black, S., and Reid, R. Impact of duration in a cardiac rehabilitation program on coronary risk profile and health related quality of life outcomes. *Journal of Cardiopulmonary Rehabilitation* 2000;**20**:115-121.

90. Engblom E, Hietanen EK, Hamalainen H, Kallio V, Inberg M, Knuts LR. Exercise habits and physical performance during comprehensive rehabilitation after coronary artery bypass surgery. *European Heart Journal* 1992;**13**:1053-9.

91. Bethell HJN, Turner SC, Mullee MA. Cardiac rehabilitation in the community: 11 year follow-up after a randomized controlled trial. *Heart* 1999;**3**:188.

92. Carlsson R, Lindberg G, Westin L, Israelsson B. Influence of coronary nursing management follow up on lifestyle after acute myocardial infarction. *Heart* 1997;**77**:256-9.

93. Haskell WL, Alderman EL, Fair JM, Maron DJ, Mackey SF, Superko R.

Effects of intensive multiple risk factor reduction on coronary athersclerosis and clinical cardiac events in men and women with coronary artery disease: The Stanford Coronary Risk Intervention Project (SCRIP). *Circulation* 1994;**89**:975-90.

94. Prosser G, Carson P, Phillips R. Exercise after myocardial infarction: Long-term rehabilitation effects. *Journal of Psychosomatic Research* 1985;**29**:535-40.

95. Vidmar PM,.Rubinson L. The relationship between self-efficacy and exercise compliance in a cardiac population. *Journal of Cardiopulmonary Rehabilitation* 1994;**14**:246-54.

96. Bock, B. C., Albrecht, A., Traficante, R. M., Clark, M. M., Pinto, B. M., and Tilkemeier, P. Predictors of exercise adherence following participation in a cardiac rehabilitation program. *International Journal of Behavioral Medicine* 1997;**4**(1):60-75.

97. Perk J, Hedback B, Engvall J. Effects of cardiac rehabilitation after coronary artery bypass grafting on readmissions, return to work, and physical fitness. *Scand J Soc Med* 1990;**18**:45-51.

98. BenAri E, Rothbaun DA, Linnemeier TA, Landin R, Tavel R, Steinmetz EF . Return to work after successful coronary angioplasty: Comparison between a comprehensive rehabilitation program and patients receiving usual care. *Journal of Cardiopulmonary Rehabilitation* 1992;**12**:20-4.

99. Carlson J, Johnson J, Franklin BA, Vanderlaan RL. Program participation, exercise adherence, cardiovascular outcomes, and program cost of traditional versus modified cardiac rehabilitation. *American Journal of Cardiology* 2000;**86**:17-23.

100. Frasure-Smith N, Lesperance F, Talajic M. Depression following myocardial infarction: Impact on 6-month survival. *JAMA* 1993;**270**:1819-25.

101. Frasure-Smith N, Lesperance F, Talajic M. Depression and 18-month prognosis after myocardial infarction. *Circulation* 1995;**91**:999-1005.

102. Mayou RA, Gill D, Thompson DR, Day A, Hicks N, Volmink J. Depression and anxiety as predictors of outcome after myocardial infarction. *Psychosomatic Medicine* 2000;**62**:212-9.

103. Glazer, K. M., Emery, C. F., Frid, D. J., and Banyasz, R. E. Psychological predictors of adherence and outcomes among patients in cardiac rehabilitation. Journal of Cardiopulmonary Rehabilitation 2002;**22**:40-46.

104. Turner, S. C., Bethell, H. J. N., Evans, J. A., Goddard, J. R., and Mullee, M. A. Patient characteristics and outcomes of cardiac rehabilitation. *Journal of Cardiopulmonary Rehabilitation* 2002;**22**:253-260.

105. Creed F. The importance of depression following myocardial infarction. *Heart* 1999;**82**:406-7.

106. Lesperance F, Frasure-Smith N, Talajic M. Major depression before and after myocardial infarction: Its nature and consequences. *Psychosomatic Medicine* 1996;**58**:99-110.

107. Engblom E, Korpilahti K, Hamalainen H, Ronnemaa T, Puukka P. Quality of life and return to work 5 years after coronary artery bypass surgery: Long-term results of cardiaac rehabilitation. *Journal of Cardiopulmonary Rehabilitation* 1997;**17**:29-36.

108. Milani RV, Lavie CJ, Cassidy MM. Effects of cardiac rehabilitation and exercise training programmes on depression in patients after major coronary events. *American Heart Journal* 1996;**132**:726-32.

109. Sandler DA, Sandler G, Benison D, Wheatcroft S, Leakey P, Blair A. Psychological and physical benefits of an exercise-based cardiac rehabilitation programme for patients recovering from a first MI. *The British Journal of Cardiology* 1999;**6**:102-11.

110. Lewin B, Robertson IH, Cay EL, Irving JB, Campbell M. Effects of self-help post-myocardial-infarction rehabilitation on psychological adjustment and use of health services. *Lancet* 1992;**339**:1036-40.

111. Dracup K, Moser D, Marsden C, Taylor SE , Guzy PM. Effects of a

multidimensional cardiopulmonary rehabilitation program on psychosocial function. *American Journal of Cardiology* 1991;**68**:31-4.

112. Oldridge NB, Gottlieb M, Guyatt G, Jones N, Streiner D, Feeny D. Predictors of health-related quality of life with cardiac rehabilitation after acute myocardial infarction. *Journal of Cardiopulmonary Rehabilitation* 1998;**18**:95-103.

113. Engebretson TO, Clark MM, Niaura RS, Phillips T, Albrecht A, Tilkemeier P. Quality of life and anxiety in a phase II cardiac rehabilitation program. *Med Sci Sport Exer* 1999;**31**:216-23.

114. Scottish Intercollegiate Guidelines Network (SIGN). Secondary prevention of coronary heart disease following myocardial infarction. SIGN publication no. 41. 2000. Edinburgh, SIGN.

115. Wood, D., Durrington, P., McInnes, G., Poulter, N., Rees, A., and Wray, R. Joint British recommendations on prevention of coronary heart disease in clinical practice. *Heart* 1998;**80**(2S):1S-29S.

116. Scandinavian Simvastatin Survival Study Group. Randomised trial of cholesterol lowering in 4444 patients with coronary heart disease. *Lancet* 1994;344:1383-1389.

117. The Long-Term Intervention with Pravastatin in Ischaemic Disease (LIPID). Prevention of Cardiovascular events and death with pravastatin in patients with coronary heart disease and a broad range of initial cholesterol levels. *New England Journal of Medicine* 1998;**339**:1349-1357.

118. Sacks, F. M., Pfeffer, M. A., and Moye, L. A. The effect of pravastatin on coronary events after myocardial infarction in patients with average cholesterols. *New England Journal of Medicine* 1996;**335**: 1001-1009.

119. Heart Protection Study Collaborative Group. MRC/BHF Heart Protection Study of cholesterol lowering with simvastatin in 20536 high risk individuals: a randomised placebo-controlled trial. *Lancet* 2002;**360**: 7-22.

120. Smith, S. C., Blair, S. N., Bonow, R. O., Brass, L. M., Cerqueira, M. D., and

Dracup, K. AHA/ACC guidelines for preventing heart attack and death in patients with atherosclerotic cardiovascular disease: 2001 update. *Journal of the American College of Cardiology* 2001;**38**:1581-1583.

121. Brochu, M., Poehlman, E. T., Savage, P., Munn, K. F., Ross, S., and Ades, P. A. Modest effects of exercise training alone on coronary risk factors and body composition in coronary patients. *Journal of Cardiopulmonary Rehabilitation* 2000;**20**:180-188.

122. Warner, J. G., Brubaker, P. H., Zhu, Y., Morgan, T. M., Ribisl, P. M., and Miller, H. S. Long-term (5-year) changes in HDL cholesterol in cardiac rehabilitation patients: Do sex differences exist? *Circulation* 1995;**92**:773-777.

123. Fox, K. F., Nuttall, M., Wood, D. A., Wright, M., Arora, B., and Dawson, E. A cardiac prevention and rehabilitation programme for all patients at first presentation with coronary heart disease. *Heart* 2001;**85**:533-538.

124. Johnston M, Foulkes J, Johnston D, Pollard B, Gudmundsdottir H. Impact on patients and partners of inpatient and extended cardiac counseling and rehabilitation: A controlled trial. *Psychosomatic Medicine* 1999;**61**:225-33.

125. Oldridge NB, Wicks JR, Hanley C, Sutton JR, Jones NL. Non-compliance in an exercise rehabilitation program for men who have suffered a myocardial infarction. *CMA Journal* 1978;**118**:361-4.

126. Carmody TP, Senner JW, Malinow MR, Matarazzo JD. Physical exercise rehabilitation: Long-term dropout rate in cardiac patients. *Journal of Behavioral Medicine* 1980;**3**:163-8.

127. Dorn, J., Naughton, J, Imamura, D, and Trevisan, M. Correlates of compliance in a randomised exercise trial in myocardial infarction patients. *Medicine and Science in Sport and Exercise* 2001;**33**(7):1081-1089.

128. Marcus B, Dubbert P, Forsyth LH, McKenzie TL, Stone EJ, Dunn AL. Physical activity behavior change: issues in adoption and maintenance. *Health Psychology* 2000;**19**:32-41.

129. Oldridge NB,.Streiner DL. The Health Belief Model: predicting compliance and dropout in cardiac rehabilitation. *Med Sci Sport Exer* 1990;**22**:678-83.

130. Oldridge NB, Jones N, Buck CW, Jones NL , Andrew GM, Parker JO. Predictors of dropout from cardiac exercise rehabilitation: Ontario Exercise-Heart Collaborative Study. *American Journal of Cardiology* 1983;**51**:70-4.

131. Blumenthal JA, Williams S, Wallace AG, Williams RB, Needles TL. Physiological and psychological variables predict compliance to prescribed exercise therapy in patients recovering from myocardial infarction. *Psychosomatic Medicine* 1982;**44**:519-27.

132. Oldridge NB, Ragowski B, Gottlieb M. Use of outpatient cardiac rehabilitation services: Factors associated with attendance. *Journal of Cardiopulmonary Rehabilitation* 1992;**12**:25-31.

133. Halm M, Penque S, Doll N, Beahrs M. Women and cardiac rehabilitation: referral and compliance patterns. *Journal of Cardiovascular Nursing* 1999;**13**:145-55.

134. Biddle SJH,.Nigg CR. Theories of exercise behavior. *International Journal of Sports Psychology* 2000;**31**:290-304.

135. Becker MH,.Maiman LA. Sociobehavioural determinants of compliance with health care and medical care recommendations. *Medical Care* 1975;**13**:10-24.

136. Biddle SJH, Mutrie N. The psychology of physical activity for health. London: Routledge, 2001.

137. Fishbein M, Ajzen I. Belief, Attitude, intention and behavior. New York: **Addison-Wesley**, 1975.

138. Ajzen I, Fishbein M. Understanding attitudes and predicting social behaviour. NJ: Prentice Hall , 1980.

139. Hausenblas HA, Carron AV, Mack DE. Application of the theories of reasoned action and planned behaviour to exercise behaviour: A meta-

analysis. *Journal of Sport and Exercise Psychology* 1997;**19**:36-51.

140. Blanchard CM, Courneya KS, Rodgers WM, Fraser SN, Murray TC, Daub B. Is the theory of planned behaviour a useful framework for understanding exercise adherence during phase II cardiac rehabilitation. *Journal of Cardiopulmonary Rehabilitation* 2003; **23**:29-39.

141. Andrew GM,.Oldridge NB. Reasons for dropout from exercise programs in post-coronary patients. *Med Sci Sport Exer* 1981;**13**:164-8.

142. Robertson D,.Keller C. Relationships among health beliefs, self-efficacy, and exercise adherence in patients with coronary artery disease. *Heart and Lung* 1992;**21**:56-63.

143. Tirrell BE,.Hart LK. The relationship of health beliefs and knowledge to exercise compliance in patients after coronary artery bypass. *Heart and Lung* 1980;**9**:487-93.

144. Hellman EA. use of the stages of change in exercise adherence model among older adults with a cardiac diagnosis. *Journal of Cardiopulmonary Rehabilitation* 1997;**17**:145-55.

145. Bandura A. Towards a unifying theory of behaviour change. *Psychol Rev* 1977;**84**:191-215.

146. Wankel LM. Decision making and social support strategies for increasing exercise involvement. *Journal of Cardiopulmonary Rehabilitation* 1984;**4**:124-35.

147. King AC,.Fredrickson DS. Low-cost strategies for increasing exercise behavior. *Behavior Modification* 1984;**8**:3-21.

148. Martin JE, Dubbert P, Katell AP. Behavioural control of exercise in sedentary adults. *Journal of Consulting and Clinical Psychology* 1984;**52**:795-811.

149. Nigg CR, Courneya KS, Estabrooks PA. Maintaining attendance at a fitness center: An application of the decisional balance sheet. *Behavioral Meidicine*

1997;**23**:130-7.

150. Belisle M, Roskies E, Levesqie JM. Improving adherence to physical activity. *Health Psychology* 1987;**52**:159-72.

151. Oldridge NB,.Jones NL. Improving patient compliance in cardiac exercise rehabilitation: Effects of written agreement and self monitoring. *Journal of Cardiopulmonary Rehabilitation* 1984;**4**:257-62.

152. Daltroy LH. Improving cardiac patient adherence to exercise regimens: A clinical trial of health education. *Journal of Cardiopulmonary Rehabilitation* 1985;**5**:40-9.

153. Marlatt GA, Gordon JR. Relapse prevention: maintenance strategies in the treatment of addictive behaviours. New York: Guilford Press, 1985.

154. Simkin LR,.Gross AM. Assessment of coping with high-risk situations for exercise relapse among healthy women. *Health Psychology* 1994;**13**:274-7.

155. King AC, Taylor CB, Haskell WL, DeBusk RF. Strategies for increasing early adherence to and long-term maintenance of home-based exercise training in healthy middle-aged men and women. *American Journal of Cardiology* 1988;**61**:628-32.

156. Marcus BH,.Simkin LR. The transtheoretical model: applications to exercise behaviour. *Med Sci Sport Exer* 1994;**26**:1400-4.

157. Marcus BH,.Simkin LR. The stages of exercise behaviour. *Journal of Sports Medicine and Physical Fitness* 1993;**33**:83-8.

158. Cardinal BJ. The stages of exercise scale and stages of exercise behaviour in female adults. *Journal of Sports Medicine and Physical Fitness* 1995;**35**:87-92.

159. Marshall, S. J. and Biddle, S. J. H. The transtheoretical model of behaviour change: A meta-analysis of applications to physical activity and exercise. *Annals of Behavioral Medicine* 2001;**23**(4): 229-246.

160. Marcus BH, Rossi JS, Selby VC, Niaura RS, Abrams DB. The stages and processes of exercise adoption and maintenance in a worksite sample. *Health Psychology* 1992;**11**:386-95.

161. Jue NH,.Cunningham SL. Stages of exercise behaviour change at two time periods following coronary artery bypass graft surgery. *Progress in Cardiovascular Nursing* 1998;**13**:23-33.

162. Marcus BH, Simkin LR, Rossi JS, Pinto BM. Longitudinal shifts in employees stages and processes of exercise behaviour change. *American Journal of Health Promotion* 1996;**10**:195-200.

163. Marcus BH, Rakowski W, Rossi JS. Assessing motivational readiness and decision making for exercise. *Health Psychology* 1992;**11**:257-61.

164. Marcus BH,.Owen N. Motivational readiness, self-efficacy and decision-making for exercise. *Journal of Applied Social Psychology* 1992;**22**:3-16.

165. Marcus BH, Banspach SW, Lefebvre RC, Rossi JS, Carleton RA, Abrams DB. Using the stages of change model to increase the adoption of physical activity among community participants. *American Journal of Health Promotion* 1992;**6**:424-9.

166. Marcus BH, Emmons KM, Simkin-Silverman LR, Linnan LA, Taylor ER, Bock BC *et al*. Evaluation of motivationally tailored vs standard self-help physical activity interventions at the workplace. *American Journal of Health Promotion* 1998;**12**:246-53.

167. Marcus BH, Bock BC, Pinto BM, Forsyth LH, Roberts MB, Traficante RM . Efficacy of an individualized, motivationally-tailored physical activity intervention. *Annals of Behavioral Medicine* 1998;**20**:174-80.

168. Bock BC, Marcus BH, Pinto BM, Forsyth LH. Maintenance of physical activity following an individualized motivationally tailored intervention. *Annals of Behavioral Medicine* 2001;**23**:79-87.

169. Goldstein MG, Pinto BM, Marcus B. Physician-based physical activity counseling for middle-aged and older adults: A randomised controlled trial.

Annals of Behavioral Medicine 1999;**21**:40-7.

170. Steptoe A, Doherty S, Rink E, Kerry S, Kendrick T, Hilton S. Behavioural counselling in general practice for the promotion of healthy behaviour among adults at increased risk of coronary heart disease: a randomised controlled trial. *British Medical Journal* 1999;**319**:943-8.

171. Dunn AL, Marcus B, Kampert J, Garcia ME , Kohl HW, Blair SN. Comparison of lifestyle and structured interventions to increase physical activity and cardiorespiratory fitness. *JAMA* 1999;**281**:327-34.

172. Calfas KJ, Long BJ, Sallis JF, Wooten WJ, Pratt M, Patrick K. A controlled trial of physician counseling to promote the adoption of physical activity. *Preventive Medicine* 1996;**25**:225-33.

173. The Writing Group for the Activity Counseling Trial research group. Effects of physical activity counseling in primary care.The Activity Counseling Trial: A randomized controlled trial. *JAMA* 2001;**286**:687.

174. Loughlan C,.Mutrie N. Conducting an exercise consultation: Guidelines for health professionals. *Journal Institute of Health Education* 1995;**33**:78-82.

175. Loughlan C,.Mutrie N. An evaluation of the effectiveness of three interventions in promoting physical activity in a sedentary population. *Health Education Journal* 1997;**56**:154-65.

176. Lowther M,.Mutrie N. The long-term effect of two physical activity interventions on the physical activity levels of members of the general public who are not regularly active. *Journal of Sports Science* 2000;**18**:17-8.

177. Kirk, A. F. Promoting and maintaining physical activity in people with Type 2 diabetes. Thesis 2003. University of Glasgow.

178. Hughes, A. R., Kirk, A. F., Mutrie, N., and MacIntyre, P. D. Exercise consultation improves exercise adherence in phase IV cardiac Rehabilitation. *Journal of Cardiopulmonary Rehabilitation* 2003;**22**(6):421-425.

179. DeBusk RF, Haskell WL, Miller N, Berra K, Taylor CB, Berger WE. Medically

directed at-home rehabilitation soon after clinically uncomplicated acute myocardial infarction: A new model for patient care. *American Journal of Cardiology* 1985;**55**:251-7.

180. Thompson DR. Cardiac Rehabilitation: How can it improved? *Journal of Psychosomatic Research* 1995;**39**:519-23.

181. Brubaker PH, Rejeski WJ, Smith MJ, Sevensky KH, Lamb KA, Sotile WM. A home-based maintenance exercise program after center-based cardiac rehabilitation: effects on blood lipids, body composition and functional capacity. *Journal of Cardiopulmonary Rehabilitation* 2000;**20**:50-6.

182. Caspersen C, Powell KE, Christenson GM. Physical activity, exercise, and physical fitness: Definitions and distinctions for health related research. *Public Health Reports* 1985;**100**:126-31.

183. Montoye HJ, Kemper HCG, Saris WHM, Washburn RA. Basic Principles and Laboratory Methods. *Measuring physical activity and energy expenditure*, pp 6-10. Human Kinetics, 1996.

184. Bland, J. M. and Altman, D. G. Statistical methods for assessing agreement between two methods of clinical measurement. *Lancet* 1986:**1**:307-310.

185. Patterson, P. Reliability, validity, and methodological response to the assessment of physical activity via self-report. *Research Quarterly for Exercise and Sport* 2000;**71**(2):15-20.

186. Gretebeck, R. J. and Montoye, H. J. Variability of some objective measures of physical activity. *Medicine & Science in Sports & Exercise* 1992;**24**(10): 1167-1172.

187. Chen, K. Y. and Sun, M. Improving energy expenditure estimation by using a triaxial accelerometer. Journal of Applied Physiology 1997;**83**(6): 2112-2122.

188. Montoye HJ, Kemper HCG, Saris WHM, Washburn RA. Doubly labeled water. *Measuirng physical activity and energy expenditure*, pp 17-21. Human

Kinetics, 1996.

189. Montoye HJ, Kemper HCG, Saris WHM, Washburn RA. Movement assessment devices. *Measuring physical activity and energy expenditure*, pp 72-9. Human Kinetics, 1996.

190. Computer Science and Applications Inc. Activity monitor operator's manual:Model 7164. 2002.

191. Hendelman, D., Miller, K., Baggett, C., Debold, E., and Freedson, P. Validity of accelerometry for the assessment of moderate intensity physical activity in the field. *Medicine & Science in Sports & Exercise* 2000;**32**(9):S442-S449.

192. Fairweather, S., Reilly, J. J., Grant, S., Whittaker, A., and Paton, J. Y. Using the Computer Science and Applications (CSA) activity monitor in preschool children. *Pediatric Exercise Science* 1999;**11**:413-420.

193. Melanson, E. and Freedson, P. Validity of the Computer Science and Applications, Inc. (CSA) activity monitor. *Medicine & Science in Sports & Exercise* 1995;**27**(6):934-940.

194. Trost, S. G., Ward, D. S., Moorehead, S. M., Watson, P. D., Riner, W., and Burke, J. R. Validity of the computer science and applications (CSA) activity monitor in children. Medicine & Science in Sports & Exercise 1998;**30**(4):629-633.

195. Freedson, P., Melanson, E., and Sirard, J. Calibration of the Computer Science and Applications, Inc. accelerometer. *Medicine & Science in Sports & Exercise* 1998;**30**(5):777-781.

196. Bassett, D. R., Ainsworth, B. E., Swartz, A. M., Strath, S. J., O'Brien, W. L., and King, G. A. Validity of four motion sensors in measuring moderate intensity physical activity. *Medicine & Science in Sports & Exercise* 2000;**32**(9):S471-S480.

197. Strath, S. J., Bassett, D. R., Swartz, A. M., and Thompson, D. L. Simultaneous heart rate-motion sensor technique to estimate energy expenditure. *Medicine & Science in Sports & Exercise* 2001;**33**(12):2118-

2123.

198. Leenders, N. Y. J. M, Sherman, W. M, Nagaraja, H. N., and Kien, C. L. Evaluation of methods to assess physical activity in free-living conditions. *Medicine & Science in Sports & Exercise* 2001;**33**(7):1233-1240.

199. Ekelund, U. and Sjostrom, M. Physical activity assessed by activity monitor and doubly labeled water in children. *Medicine & Science in Sports & Exercise* 1999;**31**(1):171-175.

200. Kochersberger, G., McConnell, E., Kuchibhatla, M. N., and Pieper, C. The reliability, validity, and stability of a measure of physical activity in the elderly. *Arch Phys Med Rehabil* 1996;**77**:793-795.

201. Nichols, J. F., Morgan, C. G., Sarkin, J. A., Sallis, J. F., and Calfas, K. J. Validity, reliability, and calibration of the Tritrac accelerometer as a measure of physical activity. *Medicine & Science in Sports & Exercise* 1999;**31**(6):908-912.

202. Jakicic, J. M., Winters, C., Lagally, K., Ho, J., Robertson, R. J., and Wing, R. R. The accuracy of the Tritrac-R3D accelerometer to estimate energy expenditure. *Medicine & Science in Sports & Exercise* 1999;**31**(5):747-754.

203. Welk, G. J., Blair, S. N., Wood, K., Jones, S., and Thompson, R. A comparative evaluation of three accelerometry based physical activity monitors. *Medicine & Science in Sports & Exercise* 2000;**32**(9):S489-S497.

204. Meijer, G. A., Westerterp, K. R., Koper, H., and Hoor, F. T. Assessment of energy expenditure by recording heart rate and body acceleration. *Medicine & Science in Sports & Exercise* 1989;**21**(3):343-347.

205. Sherman, W. M, Morris, D. M., Kirby, T. E., Petosa, R. A., Smith, B. A., Frid, D. J., and Leenders, N. Evaluation of a commercial accelerometer (Tritrac-R3 D) to measure energy expenditure during ambulation. *International Journal of Sports Medicine* 1998;**19**:43-47.

206. Fehling PC, Smith DL, Warner SE, Dalsky GP. Comparison of accelerometers with oxygen consumption in older adults during exercise.

Medicine & Science in Sports & Exercise 1999;**31**(1):171-175.

207. Johnson NA,.Heller RF. Prediction of patient non-adherence to with home-based exercise for cardiac rehabilitation: The role of perceived barriers and perceived benefits. *Preventive Medicine* 1998;**27**:56-64.

208. Eston, R. G., Rowlands, A. V., and Ingledew, D. K. Validity of heart rate, pedometry, and accelerometry for predicting the energy cost of children's activities. *Journal of Applied Physiology* 1998;**84**(1):362-371.

209. Bassett, D. R., Ainsworth, B. E., Leggett, S. R., Mathien, C. A., Main, J. A., Hunter, D. C., and Duncan, G. E. Accuracy of five electronic pedometers for measuring distance walked. *Medicine & Science in Sports & Exercise* 1996;**28**(8):1071-1077.

210. Montoye HJ, Kemper HCG, Saris WHM, Washburn RA. Estimating energy expenditure from physiologic response to activity. *Measuring physical activity and energy expenditure*, pp 97-109. Human Kinetics, 1996.

211. Freedson, P. S. and Miller, K. Objective monitoring of physical activity using motion sensors and heart rate. *Research Quarterly for Exercise and Sport* 2000;**71**(2):21-29.

212. Racette, S. B., Schoeller, D. A., and Kushner, R. F. Comparison of heart rate and physical activity recall with doubly labeled water in obese women. *Medicine & Science in Sports & Exercise* 1995;**27**(1):126-133.

213. Luke, A., Maki, K. C., Barkey, N., Cooper, R., and McGee, D. Simultaneous monitoring of heart rate and motion to assess energy expenditure. *Medicine & Science in Sports & Exercise* 1997;**29**(1):144-148.

214. Spurr, G. B., Prentice, A. M., Murgatroyd, P. R., Goldberg, G. R., Reina, J. C., and Christman, N. T. Energy expenditure from minute-by minute heart rate recording: comparison with indirect calorimetry. *American Journal of Clinical Nutrition* 1988;**48**:552-559.

215. Livingstone, M. B. E., Coward, W. A., Prentice, A. M., Davies, P. S. W., Strain, J. J., and McKenna, P. G. Daily energy expenditure in free-living

children: comparison of heart rate monitoring with the doubly labeled water method. *Am J Clin Nutr* 1992;**56**:343-352.

216. Rennie, K. L., Hennings, S. J., Mitchell, J., and Wareham, N. J. Estimating enrgy expenditure by heart-rate monitoring without individual calibration. *Medicine & Science in Sports & Exercise* 2001;**33**(6):939-945.

217. Schutz Y, Weinsier RL, Hunter GR. Assessment of free living physical activity in humans: An overview of currently available and proposed new measures. *Obesity Research* 2001;**9**:368-79.

218. Montoye HJ, Kemper HCG, Saris WHM, Washburn RA. Questionnaires and interviews. *Measuring Physical Activity and Energy Expenditure*, pp 42-56. Human Kinetics, 1996.

219. Sallis, J. F. and Saelens, B. E. Assessment of physical activity by self-report: Status, limitations and future directions. *Research Quarterly for Exercise and Sport* 2000;**71**(2):1-14.

220. Blair, S. N., Haskell, W. L., Ho, P., Paffenbarger, R. S., Vranizan, K. M., and Farquhar, J. W. Assessment of habitual physical activity by a seven day recall in a community survey and controlled experiments. *American Journal of Epidemiology* 1985;**122**:794-804.

221. Riki, R. E. Reliability, validity and methodological issues in assessing physical activity in older adults. *Research Quarterly for Exercise and Sport* 2000;**71**(2):89-96.

222. Pereira, M. A., Fitzgerald, S. J., Gregg, E. W., Joswiak, M. L., Ryan, W. J., and Suminski, R. R. Seven-Day physical activity recall. *Medicine & Science in Sports & Exercise* 1997;**29**(6):S89-S103.

223. Duncan, G. E., Sydeman, S. J., Perri, M. G., Limacher, M. C., and Martin, A. D. Can sedentary adults accurately recall the intensity of their physical activity. *Preventive Medicine* 2001;**33**:18-26.

224. Williams, E., Kleges, R, Hanson, C. L., and Eck, L. H. A prospective study of the reliability and convergent validity of three physical activity measures in a

field research trial. Journal of Clinical Epidemiology 1989;**42**(12):1161-1170.

225. Gross, L. D., Sallis, J. F., Buono, M. J., and Roby, J. J. Reliability of interviewers using the seven-day physical activity recall. *Research Quarterly for Exercise and Sport* 1990;**61**(4):321-325.

226. Sallis, J. F., Haskell, W. L., Wood, P. D., Fortmann, S. P., Rogers, T., and Blair, S. N. Physical activity assessment methodology in the five-city project. American Journal of Epidemiology 1985;**121**(1):91-106.

227. Jacobs, D. R., Ainsworth, B. E., Hartman, T. J., and Leon, A. S. A simultaneous evaluation of 10 commonly used physical activity questionnaires. *Medicine & Science in Sports & Exercise* 1993;**25**(1):81-91.

228. Richardson MT, Ainsworth BE, Jacobs DR, Leon AS. Validation of the Stanford 7-Day Recall to assess habitual physical activity. *Ann Epidemiol* 2001;**11**:145-53.

229. Baranowski T. Validity and reliability of self-report measures of physical activity: An information-processing perspective. *Research Quarterly for Exercise and Sport* 1988;**59**:314-27.

230. Bonnefoy M, Normand S, Pachiaudi C, Lacour JR, Laville M, Kostka T. Simultaneous validation of ten physical activity questionnaires in older men: A doubly labeled water study. *J Am Geriatr Soc* 2001;**49**:28-35.

231. Conway, J. M., Seale, J. L., Jacobs, D. R., Irwin, M. L., and Ainsworth, B. E. Comparison of energy expenditure estimates from doubly labeled water, a physical activity questionnaire and physical activity records. American Journal of Clinical Nutrition 2002;**75**(3):519-525.

232. Irwin, M. L., Ainsworth, B. E., and Conway, J. M. Estimation of energy expenditure from physical activity measures: Determinants of accuracy. *Obesity Research* 2001;**9**(9):517-525.

233. Leenders, N. Y. J. M, Sherman, W. M, and Nagaraja, H. N. Comparisons of four methods of estimating physical activity in adult women. *Medicine &*

Science in Sports & Exercise 2000;**32**(7):1320-1326.

234. Taylor, C. B, Coffey, T., Berra, K., Iaffaldano, R., Casey, K., and Haskell, W. L. Seven-day activity and self-report compared to a direct measure of physical activity. *American Journal of Epidemiology* 1984;**120**:818-824.

235. Fletcher GJ, Balady GJ, Froelicher V, Hartley LH, Haskell WL, Pollock ML. Exercise Standards: A statement for healthcare professionals from the American Heart Association. *Circulation* 1995;**91**:580-615.

236. American College of Sports Medicine. ACSM's guidelines for exercise testing and prescription. Williams and Wilkins, 1995.

237. ESC Working Group on Exercise Physiology PaE. Guidelines for cardiac exercise testing. *European Heart Journal* 1993;**14**:969-88.

238. Beaver WL, Wasserman K, Whipp BJ. A new method for detecting anaerobic threshold by gas exchange. *J Appl Physiol* 1986;**60**:2020-7.

239. Zigmond AS,.Snaith RP. The hospital anxiety and depression scale. *Acta Psychiatr Scand* 1983;**67**:361-70.

240. Snaith RP, Zigmond AS. The hospital anxiety and depression scale. Berkshire: NFER-NELSON, 1994.

241. Herrmann C. International experiences with the hospital anxiety and depression scale: a review of validation data and clinical results. *Journal of Psychosomatic Research* 1997;**42**:17-41.

242. Visser MC, Koudstaal PJ, Erdman RAM, Deckers JW, Passchier J, Gijn JV. Measuring quality of life in patients with myocardial infarction or stroke: a feasibility study of four questionnaires in The Netherlands. *Journal of Epidemiology and Community Health* 1995;**49**:513-7.

243. Johnston M, Pollard B, Hennessey P. Construct validation of the hospital anxiety and depression scale with clinical populations. *Journal of Psychosomatic Research* 2000;**48**:579-84.

244. Bjelland I, Dahl AA, Haug TT, Neckelmann D. The validity of the Hospital Anxiety and Depression Scale: An updated review. *Journal of Psychosomatic Research* 2002;**52**:69-77.

245. Crawford JR, Henry JD, Crombie C, Taylor EP. Normative data for the HADS from a large non-clinical sample. *British Journal of Clinical Psychology* 2001;**40**:429-34.

246. McGhee HM, Hevey D, Horgan JH. Psychosocial outcome assessments for use in cardiac rehabilitation service evaluation: a 10-year systematic review. *Social Science and Medicine* 1999;**48**:1373-93.

247. Hevey, D. and McGhee, H. M. Responsiveness of psychosocial instruments to multifactorial cardiac rehabilitation. Hughes, A. R. Personal Communication 20-6-2000.

248. AACVPR outcome committee. Outcome measurement in cardiac and pulmonary rehabilitation. *Journal of Cardiopulmonary Rehabilitation* 1995;**15**:394-405.

249. Smith HJ, Taylor R, Mitchell A. A comparison of four quality of life instruments in cardiac patients: SF-36, QLI, QLMI, and SEIQoL. *Heart* 2000;**84**:390-4.

250. Spertus J, Winder JA, Dewhurst T, Deyo RA, Prodzinski J, McDonell M. Development and evaluation of the Seattle Angina Questionnaire: A new functional status measure for coronary artery disease. *Journal of the American College of Cardiology* 1995;**25**:333-41.

251. Dempster M,.Donnelly M. Measuring the health related quality of life of people with ischaemic heart disease. *Heart* 2000;**83**:641-4.

252. Evidence regarding the validity, reliability, and responsiveness of health related quality of life instruments among people with ischaemic heart disease. *Heart* 2002.

253. Brown N, Melville M, Gray D, Young T, Skene AM, Hampton JR. Comparison of the SF-36 health survey questionnaire with the Nottingham Health Profile

in long-term survivors of a myocardial infarction. *Journal of Public Health Medicine* 2000;**22**:167-75.

254. Jette DU,.Downing J. Health status of individuals entering a cardiac rehabilitation program as measured by the medical outcomes study 36-item short-form survey (SF-36). *Physical Therapy* 1994;**74**:521-7.

255. Marquis P, Fayol C, Joire JE. Clinical validation of a quality of life questionnaire in angina pectoris patients. *European Heart Journal* 1995;**16**:1554-60.

256. Wolinsky FD, Wyrwich KW, Nienaber NA, Tierney WM. Generic versus disease-specific health status measures: An example using coronary artery disease and congestive heart failure patients. *Evaluation and the Health Professionals* 1998;**21**:216-43.

257. Dougherty CM, Dewhurst T, Nichol WP, Spertus J. Comparison of three quality of life instruments in stable angina pectoris: Seattle Angina Questionnaire, Short Form Health Survey (SF-36), and Quality of Life Index-Cardiac Version III. *Journal of Clinical Epidemiology* 1998;**51**:569-75.

258. Brown N, Melville M, Gray D, Young T, Munro J, Skene AM. Quality of life four years after acute myocardial infarction: short form 36 scores compared with a normal population. *Heart* 1999;**81**:352-8.

259. Jenkinson C, Stewart-Brown S, Petersen S, Paice C. Assessment of the SF-36 version 2 in the United Kingdom. *Journal of Epidemiology and Community Health* 1999;**53**:46-50.

260. Loughlan, C. and Mutrie, N. Recruitment of sedentary NHS staff for a workplace exercise programme using an adapted stages of change exercise questionnaire. *Journal of Sports Science* 1995;**13**:63-64.

261. Jenkinson, C., Layte, R., Wright, L., and Coulter, A. The UK SF-36: An analysis and interpretation manual. 1996. Oxford, Health Services Research Unit.

262. Florey, C. Sample size for beginners. *British Medical Journal* 1993;**306**:1181-

1184.

263. Kirk AF, Higgins LA, Hughes AR, Fisher BM, Mutrie N, Hillis S *et al.* A randomized, controlled trial to study the effect of exercise consultation on the promotion of physical activity in people with Type 2 diabetes: a pilot study. *Diabetic Medicine* 2001;**18**:877-82.

264. Rose, D. and O'Reilly, K. The ESRC Review of Government Social Classification. 1998. Essex, Office for National Statistics and Economic and Social Research Council.

265. Mutrie, N. Exercise Consultation. 1999. University of Glasgow, Media Services. Video Recording

266. British Heart Foundation. Physical activity and your heart. 1999

267. Marcus, B. H., Selby, V. C., Niaura, R. S., and Rossi, J. S. Self-efficacy and the stages of exercise behaviour change. *Research Quarterly for Exercise and Sport* 1992;**63**:60-66.

268. Lowther, M. The effects of a fitness assessment and exercise consultation on physical activity intention and behaviour in a socially and economically deprived community: an application of the transtheoretical model of behaviour change. 2000. University of Glasgow. Thesis

269. Kirk, A. F., Hughes, A. R., Tait, A., Thomson, T., and MacIntyre, P. D. Physical assessment in clinical populations in Scotland. *Medicine and Science in Sport and Exercise* 2001;**33**(5):S56.

270. McLaughlin, J. E., King, G. A., Howley, E. T., Bassett, D. R., and Ainsworth, B. E. Validation of the Cosmed K4b^2 portable metabolic system. International *Journal of Sports Medicine* 2001;**22:**280-284.

271. Parr, B. B., Strath, S. J., Bassett, D. R., and Howley, E. T. Validation of the Cosmed K4b^2 portable metabolic measurement system. *Medicine & Science in Sports & Exercise* 2002;S300.

272. Crowe, A. Validation of the Cosmed K4b^2 for measuring gas exchange in

cardiac rehabilitation participants. 2002 University of Glasgow. Thesis

273. MacFarlane, D. J. Automated metabolic gas analysis systems: A review. *Sports Medicine* 2001;**31**(12):841-861.

274. Cosmed. Cosmed $K4b^2$ user manual. Third Edition. 1998. Italy.

275. Neder, A. Exercise testing protocol for cardiac rehabilitation patients. Personal Communication 1999.

276. Ward, S. and Neder, A. Procedures for determining the lactate threshold using the V-slope and ventilatory equivalent methods. Personal Communication 1999.

277. Rodgers WM, Courneya KS, Bayduza AL. Examination of the transtheoretical model in exercise in 3 populations. *American Journal of Health Behaviour* 2001;**25**:33-41.

278. Friedewald, W. T., Levy, R. I., and Fredrickson, D. S. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparation ultracentrifuge. *Clinical Chemistry* 1972;**18**:499-502.

279. Lowther M, Mutrie N, Loughlan C, McFarlane C. Development of a Scottish physical activity questionnaire: A tool for use in physical activity interventions. *British Journal of Sports Medicine* 1999;**33**:244-9.

280. Milani RV,.Lavie CJ. The effects of body composition changes to observed improvements in cardiopulmonary parameters after exercise training with cardiac rehabilitation. *Chest* 1998;**113**:599-601.

281. Brubaker PH, Warner JG, Rejeski WJ, Edwards DG, Matrazzo BA, Ribisl PM. Comparison of standard and extended length participation in cardiac rehabilitation on body composition, functional capacity, and blood lipids. *American Journal of Cardiology* 1996;**78**:769-73.

282. Vanhees L, Fagard R, Lutgarde T, Amery A. Prognostic value of training-induced change in peak exercise capacity in patients with myocardial infarcts and patients with coronary artery bypass surgery. *American Journal*

of Cardiology 1995;**76**:1014-9.

283. Kavanagh T, Mertens DJ, Hamm L, Beyene J, Kennedy J, Corey P. Prediction of long-term prognosis in 12169 men referred for cardiac rehabilitation. *Circulation* 2002;**106**:666-71.

284. Dorn J, Naughton J, Imamura D, Trevisan M. Results of a multicenter randomised clinical trial of exercise and long-term survival in myocardial infarction patients: The National Exercise and Heart Disease Project. *Circulation* 1999;**100**:1764-9.

285. Cheuk-Man Y, Chu-Pak L, Cheung B, Yee-Man F, Ho Y, Kam-Bik L. Clinical predictors of morbidity and mortality in patients with myocardial infarction or revascularisation who underwent cardiac rehabilitation, and importance of diabetes mellitus and exercise capacity. *American Journal of Cardiology* 2000;**85**:344-9.

286. Hevey D, Brown A, Cahill A, Newton H, Kierns M, Horgan J. Four-week multidisciplinary cardiac rehabilitation produces similar improvements in exercise capacity and quality of life to a 10-week programme. *Journal of Cardiopulmonary Rehabilitation* 2003;**23**:17-21.

287. Underwood, S. J., Barlow, C. E., Chandler, J. L., Kampert, J., Dunn, A. L., and Blair, S. N. Project Prime: 6 month changes in physical activity. *Medicine and Science in Sport and Exercise* 2000;**32**;97.

APPENDIX A

0141 842 7266
Elizabeth Hutchenson
0141 842 7308

LREC 5/99

4th July 2000

Dr Paul MacIntyre
Consultant Physician and Cardiologist
Argyll and Clyde Acute Hospitals NHS Trust
Royal Alexandra Hospital
Corsebar Road
PAISLEY
PA2 9PN

Dear Dr MacIntyre

THE EFFECT OF AN EXERCISE CONSULTATION ON EXERCISE ADHERENCE IN PHASE IV CARDIAC REHABILITATION

Many thanks for submitting an amendment to the above protocol. The committee reviwed this on the 5th July, 2000 and are happy to grant approval.

A list of Committee members present at meeting held on 19th January 2000 is appended.

Yours sincerely

J.J. Morrice F.R.C.S.
Chairman

LOCAL RESEARCH ETHICS COMMITTEE

C:\ADRIENNE\Thesis\Appendices\PM0102A[2].doc

APPENDIX B

CARDIAC REHABILITATION RESEARCH

FORM OF INFORMATION AND CONSENT

Title of Research
Physical activity levels in a group of cardiac rehabilitation patients

Summary
We invite you to take part in a study looking at physical activity in a group of cardiac rehabilitation patients. This study will allow you to learn more about the benefits of physical activity and will provide regular medical check-ups over one year as part of your rehabilitation.

If you agree to take part, you will be asked to visit the *Royal Alexandra Hospital* between 6 and 8 times over the course of 1 year. These visits will last 30 to 60 minutes: the details of each visit are described in more detail below.

Visit 1: Total duration 1hour.
During visit one you will be asked to complete a personal health questionnaire and your weight and height will be measured. You will also be asked to perform an exercise test, which will last approximately 10 minutes. We will assess your exercise performance by asking you to exercise on a treadmill and measuring the amount of oxygen you use. During the test, we will monitor your heart rate using an ECG and measure your blood pressure by an inflation cuff. We will measure your breathing during the exercise test by asking you to breathe through a mouthpiece. You will be asked to keep exercising until you can no longer continue. The test will be stopped if you develop chest pain or if there are changes on the ECG. You will also be given a small monitor about the size of a 50p piece, which you will be asked to wear on your ankle. This will record the amount of physical activity you do. The researcher will show you how to attach the monitor and instruct you to wear it during all waking hours (except during bathing or other water activities) for 7 days immediately after the first visit.

You will be asked to return for visit 2 one week after your first visit.

Visit 2: Total duration 30 minutes.
During this visit, a blood sample will be taken from your arm. A fasting blood sample is required, therefore you will be asked to refrain from eating for 12 hours before your visit. Breakfast will be provided after you have given the sample. During this visit you will return the activity monitor and the researcher will ask you about your physical activity during the previous 7 monitored days. You will also be asked to complete a questionnaire about your present exercise habits.

You may be asked to return for a third visit within 7 days after visit 2.

Visit 3: Total duration 30 minutes.
This visit involves a discussion with the research assistant on exercise. You will also receive a telephone call phone call 3 months after this visit.

Six and 12 months after visit 3, you will be asked to return to the Royal Alexandra Hospital where the procedures carried out during visits 1, 2 and 3 will be repeated. Visit 3 will not be

repeated at 1 year.

Final Points to Note

If you do not wish to participate, it will not affect your current or future medical treatment in anyway. You are free to withdraw from the study at any point without providing any explanation and with no obligation. Should you decide to take part your GP will be informed. The only people who will have access to your results with be those involved in the testing and analysis of the data. It is possible that the results of this study will be published in a medical journal. Although your own results will be part of any publication your identity will not be disclosed at any point. If you are, or are likely to become pregnant, you should not participate in this study. If you are harmed by taking part in this research, there is no special compensation arrangements. If you are harmed due to someone's negligence, then you may have grounds for legal action but you may have to pay for it. Regardless of this, if you wish to complain about any aspect of the way you have been approached or treated during the course of this study, the normal National Health complaints mechanism is available to you.

If you have any questions about the study or need to contact us for any reason, please use any of the following means:

Adrienne Hughes, Research assistant, L5 North, RAH, Paisley. 0141 580 4628 or 0141 330 2830

Dr Paul MacIntyre, Consultant Cardiologist: L5 North, RAH, Paisley. 0141 580 4628

Consent:

I,.....

give my consent to the research procedures described above, the nature, purpose and possible consequences of which have been described.

Signed:.....

Date: .....

Witness.....

Date: .....

APPENDIX C

EXERCISE CONSULTATION

- Current physical activity status (summarise from 7-day recall and Stage of Change)

Decisional Balance Table

Benefits/Pros	Barriers/Cons

- Problem solving barriers/cons of physical activity

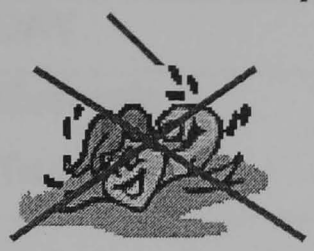
- Review current physical activity guidelines
- Past physical activity Status
- Possible activities
- High risk situations for lapse/relapse
- Plan to to cope with these situations.
- Sources of support for physical activity.

Goals

One Month	Three Month	Six Month

APPENDIX D

ACTIVITY MONITOR INFORMATION



- THE ACTIVITY MONITOR IS NOT WATERPROOF, HOWEVER, SMALL WATER SPLASHES WILL NOT CAUSE ANY DAMAGE. YOU WILL HAVE TO TAKE THE MONITOR OFF TO BATHE, SWIM OR SHOWER. PLEASE RECORD THE TIME YOUR REMOVE AND RE-ATTACH THE MONITOR ON THE BACK OF THIS SHEET.

- THE ACTIVITY MONITOR SHOULD BE WORN ON YOUR ANKLE ABOVE YOUR ANKLE BONE. USE THE STRAP PROVIDED, AND MAKE SURE THE ARROW IS POINTING UPWARD.

- WEAR THE MONITOR DURING ALL WAKING HOURS, EXCEPT WHEN BATHING. LEAVE THE MONITOR IN PLACE UNTIL YOU GO TO BED. WHEN YOU TAKE THE MONITOR OFF RECORD THE TIME ON THE BACK OF THIS SHEET. ON WAKING IN THE MORNING, REATTACH THE MONITOR TO YOUR ANKLE IMMEDIATELY. RECORD THE TIME OF REATTACHMENT ON THE BACK OF THIS SHEET. CONTINUE WEARING THE MONITOR IN THIS MANNER EVERY DAY UNTIL YOUR NEXT VISIT.

- AS THESE TOP-OF-THE-RANGE MONITORS ARE EXPENSIVE, YOU ARE ASKED TO TAKE VERY GOOD CARE OF THEM.

- PLEASE RECORD ANY DAY AND TIME THE ACTIVITY MONITOR IS NOT WORN ON THE BACK OF THIS SHEET.

RECORD FOR ACTIVITY MONITOR

Please be as accurate as possible with this record

SLEEPING

DAY	MONITOR ATTACHED	MONITOR REMOVED
Example Tuesday	9.05am	10.16pm

BATHING OR SWIMMING

DAY	MONITOR ATTACHED	MONITOR REMOVED

ANY PROBLEMS WITH ACTIVITY MONITOR

DAY	REASON	MONITOR ATTACHED	MONITOR REMOVED

APPENDIX E

PHYSICAL ACTIVITY RECORDING SHEET

Please keep a record of any activities you do over the following week. However, only record activities that are of a **MODERATE INTENSITY OR ABOVE**. Moderate intensity activities require about the same effort as a brisk walk.

DAY	TYPE OF ACTIVITY & DURATION
e.g. Monday	Attended an exercise class lasting 30 minutes.