Exercise-related cardiac arrest in cardiac rehabilitation

The Johannesburg experience

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Summary

Prescribed physical activity plays a major role in the rehabilitation of patients with coronary artery disease, and as with any other form of treatment its benefits must be weighed against its possible risks.

This study attempted to establish the safety of cardiac rehabilitation as a medical intervention at the Johannesburg Cardiac Rehabilitation Centre from its inception in September 1982 to July 1988, and analyses the medical status of patients who suffered a cardiac arrest (CA) in order to determine possible factors predictive of sudden death.

Between September 1982 and July 1988, 1574 patients were admitted to the unit; 480 000 man-hours of exercises were accumulated with 4 episodes of CA, giving an incidence of CA of 1/120 000 patient-hours. Three of the 4 episodes were fatal, giving an incidence of fatal CA of 1/160 000 patient-hours. This incidence is acceptably low and comparable with other cardiac rehabilitation programmes, making exercise as prescribed at the Johannesburg Cardiac Rehabilitation Centre a safe form of medical intervention.

Patients at risk of CA during exercise were essentially not identifiable, since they did not come from a group currently recognised as at particularly high risk. A combination of inferior infarction with occluded dominant right coronary artery, good collateralisation and asymptomatic ischaemia was present in all CA patients. The likelihood of these pathological features being predictors of exercise-related sudden death requires further investigation.

Prescribed physical activity plays a major role in the rehabilitation of patients with coronary artery disease.

As with any other form of treatment, the benefit of cardiac rehabilitation must be weighed against the possible risks, the most important being myocardial infarction and cardiac arrest (CA).1,2

‘Normal’ subjects participating in high-intensity activities such as jogging have been found to have an increased risk of CA,3,4 notwithstanding the fact that such high-intensity activities appear to protect against CA.5 Epidemiological studies reported that cardiovascular and coronary artery disease mortality are inversely related to the level of long-term physical activity.

Experience in North America has shown that the risk of myocardial infarction and CA in cardiac rehabilitation programmes is low.6 An important factor contributing to the present low risk rates is identification of patients at highest risk of CA during cardiac rehabilitation.7

Cardiac rehabilitation has been practised in South Africa since the mid-1970s at a number of centres, but to date no information on local experience of cardiovascular complications has been published.

The aims of this study were: (i) to establish the safety of cardiac rehabilitation as a medical intervention by investigating the incidence of CA at the Johannesburg Cardiac Rehabilitation Centre from its inception in September 1982 to July 1988; and (ii) to analyse the medical status of patients who suffered a CA in order to establish possible factors that could identify those at risk of sudden death.

Methods

All subjects were referred to the unit for cardiac rehabilitation with the approval of their attending physician. Medical conditions included known coronary artery disease, namely myocardial infarction, coronary artery bypass graft, percutaneous transluminal coronary angioplasty and angina; asymptomatic patients with coronary risk factors were also referred.

A complete medical history was recorded, followed by comprehensive cardiovascular examination and a resting ECG. Symptom-limited multistage exercise testing (initially using a cycle ergometer and later a motorised treadmill) was performed on all subjects, provided no contraindications existed. Oxygen uptake, minute volume and respiratory quotient were measured using a metabolic cart (Jaeger Sprint). Continuous ECG and blood pressure monitoring were done at each stage of the exercise test and in the recovery phase.

Exercise end-points were physical exhaustion, development of severe angina, ST depression of more than 3 mm, sustained ventricular tachyarrhythmias and a drop in systolic blood pressure of more than 10 mmHg.8 The test was considered positive if 1 mm horizontal or down-sloping ST depression persisting 80 milliseconds after the J point was observed.

Peak oxygen uptake and anaerobic threshold were used to prescribe the intensity of exercise, in terms of a training heart rate.

Patients were required to exercise at their prescribed intensity 3 times a week for 30 - 50 minutes of continuous dynamic effort. Aerobic exercise consisted of walking/jogging when possible, or stationary cycling in high-risk groups or when weight-bearing exercise was contraindicated.

The patient’s compliance with the programme was monitored on a daily basis in terms of attendance and intensity (% of training heart rate achieved). Participants were instructed to report any untoward symptoms that occurred between exercise sessions and any during the supervised exercise session. Patients were re-evaluated at 6 and 18 months after their admission when, their medical condition permitting, they embarked on a home exercise programme, reporting to the unit once a month.
All sociodemographic, clinical and physiological data were stored on a personal computer using a DBase III database. The unit was computerised from June 1986.

All patients were reviewed with regard to the incidence of the major cardiovascular complication, CA. Patients who suffered CA were evaluated with regard to age, sex, occupation, presence of risk factors, medication, previous history, coronary angiographic results, exercise testing variables, risk, exercise prescription and compliance with the programme.

Information regarding the number of patients, number of exercise sessions and incidence of CA was extracted manually between September 1982 and June 1986. Between June 1986 and July 1988 the data were extracted from the database.

In practice, a session, including warm-up, exercise and cooldown, lasted approximately 1 hour, and so an ‘exercise session’ was assumed to be 1 hour for calculation purposes. Attendance figures were kept accurately for every patient except for 1982 and early 1983, when they were extracted with some degree of estimation.

As the pattern of referrals to the unit has not changed over the period under review, we used the computerised data available since June 1986 as being representative of the demographic data and the distribution of previous myocardial infarction in the group.

Results

The 1,574 patients admitted to the unit between September 1982 and July 1988 accumulated a total of 480,000 exercise man-hours.

Of these patients, 387 were admitted between June 1986 and July 1988. The average age was 55 years for males and 58 years for females, with a male/female ratio of 8:1.

Sixty-three per cent of the 387 patients had had a previous myocardial infarction. The position of the myocardial infarction was inferior in 41% of cases and anterior in 32.5%, in the remaining 26.5% the position was posterior, lateral, sub-endocardial or unknown (Table 1).

### TABLE I. ADMISSIONS TO THE JOHANNESBURG CARDIAC REHABILITATION CENTRE, JUNE 1986 — JULY 1988*

<table>
<thead>
<tr>
<th>No. of admissions</th>
<th>387</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males (%)</td>
<td>89.2</td>
</tr>
<tr>
<td>Females (%)</td>
<td>10.8</td>
</tr>
<tr>
<td>Previous MI (%)</td>
<td>63.0</td>
</tr>
<tr>
<td>Age (yr) (mean ± SD)</td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>55 ± 8.9</td>
</tr>
<tr>
<td>Females</td>
<td>59 ± 9.7</td>
</tr>
<tr>
<td>Distribution of MI (%)</td>
<td></td>
</tr>
<tr>
<td>Ant.</td>
<td>32.5</td>
</tr>
<tr>
<td>Inf.</td>
<td>41.0</td>
</tr>
<tr>
<td>Other</td>
<td>26.5</td>
</tr>
</tbody>
</table>

*Between September 1982 and July 1988, 1,574 patients were admitted to the Centre. The data presented here are considered representative of this larger group. MI = myocardial infarction; Ant. = anterior; Inf. = inferior.

Between September 1982 and July 1988, 4 patients suffered CA. One survived and 3 died despite cardiopulmonary resuscitation being instituted immediately and DC defibrillation applied within 2 minutes of the event. Patients were walking or cycling during their normal exercise sessions when they collapsed without any prodromal symptoms. All were in CA as a consequence of ventricular fibrillation.

The incidence of CA was 1/120,000 patient-hours and the incidence of fatal CA 1/160,000 patient-hours.

All the patients who suffered CA were males, and their ages ranged from 55 to 64 years (mean 60.5 years) (Table II). They were all actively employed except for patient 2. All had a past history of smoking, patients 2 and 4 had hypercholesterolaemia, and patient 2 had a history of hypertension. Patient 2 had had a below-knee amputation as a consequence of a motor vehicle accident.

Three patients had suffered a previous inferior myocardial infarction 2–18 months before admission, and 1 was found to have a positive stress test on a routine check-up. Patient 1 had also had a previous anterior myocardial infarction a number of years before and had ventricular ectopy requiring treatment in subsequent symptom-limited stress tests.

Coronary angiography, done only in 3 of the patients, had shown double-vessel disease in 2 patients and single-vessel disease in the other. All had dominant right coronary arteries, which were totally occluded and collateral circulation was good. The ejection fraction was mildly reduced in patient 1.

The same 3 patients had had asymptomatic ischaemic exercise tests at the Rehabilitation Centre, with all but the amputee achieving good peak oxygen uptake on the test.

The patients who suffered CA were all regarded on admission as at low risk. Cycling at training heart rates of 130 and 135/min respectively was prescribed for patients 1 and 2, and walking/jogging at training heart rates of 95 and 80/min for patients 3 and 4.

The patients' compliance in terms of percentage attendance ranged from 21% to 92% (mean 57.5%). All patients showed excellent compliance with the intensity of the exercise (80–100%). Their time on the programme ranged from 4 to 20 months (mean 11,75 months).

Discussion

Incidence of cardiac arrest

Between September 1982 and July 1988, 480,000 man-hours of exercise were accumulated with 4 episodes of CA, giving an incidence of CA of 1/120,000 patient-hours. Three out of 4 episodes were fatal, giving an incidence of fatal CA of 1/160,000 patient-hours.

Haskell in a multicentre study provided information about cardiac rehabilitation complications in the USA between 1960 and 1977. He reported an incidence of CA of 1/32,593 patient-hours and an incidence of fatal CA of 1/116,402 patient-hours. Van Camp et al.6 through questionnaires mailed to 75% of all cardiac rehabilitation centres in the USA, determined the incidence of complications from January 1980 to December 1984. They reported an incidence of CA of 1/111,996 patient-hours and an incidence of fatal CA of 1/783,972 patient-hours.

The incidence of CA in our unit is low and similar to the experience of cardiac rehabilitation programmes in the USA between 1980 and 1984. The incidence of fatal CA in our unit is higher than that reported by Van Camp et al.6 in spite of early and vigorous cardiopulmonary resuscitation by experienced personnel.

The risk status of the patients in both studies was unknown, so strict comparisons are not appropriate.

In Van Camp et al.'s6 and our study the rate of CA is approximately one-fourth of that reported by Haskell and the fatality rate is also lower. These results could suggest improved safety in the present practice of cardiac rehabilitation.

Status of the patients

Three out of 4 patients included in this study (numbers 1, 2 and 4) had suffered a previous inferior myocardial infarction. This predominance of inferior infarct could be explained in
part by the fact that the distribution of inferior infarcts in our population is slightly higher than that of anterior infarcts.

Three patients (numbers 1, 3 and 4) had total occlusion of a dominant right coronary artery. Although these 3 patients had well-developed collaterals, thallium scanning studies have shown that angiographic appearance is not necessarily synonymous with flow and/or function. Myers,11 who analysed autopsies performed after 100 sudden deaths from coronary artery disease, found that sudden death was more often associated with recent occlusion of the right coronary artery and inferior infarction than with the left anterior descending artery and associated anterior infarction. These 3 patients had silent ischaemia during their exercise tests.

All 4 of the patients discussed collapsed while exercising. Repeated ischaemia during exercise at higher workloads in the absence of a warning system (angina) could lead to ventricular arrhythmias and sudden death. However, patients 3 and 4 were only walking when they collapsed, and collapse could easily have occurred in any other setting, such as in the street or at home in the course of normal activities, suggesting a more complicated mechanism than an increase in oxygen demands, possibly involving vasospasm.

Studies in sportsmen showed that coronary artery disease appears to be the most important cause of sudden death in the population over 40 years of age and that most of these sudden deaths occurred in asymptomatic patients, some of whom were even found to have healed myocardial infarcts at autopsy, which were apparently clinically silent.12-14

Risk stratification after myocardial infarction is mainly based on the extent of myocardial ischaemia and left ventricular dysfunction. According to the risk stratification algorithm of DeBusk et al.,17 by 3 weeks after a documented acute myocardial infarction, 50% of patients will be considered as at low risk with 1st-year mortality less than 2%, 30% will be at moderate to high risk with 10 - 25% 1st-year mortality, and 20% will fall into a high-risk group with more than 25% 1st-year mortality because of severe left ventricular dysfunction.

This algorithm was utilised to review our patients’ risk assessment. Patient 1, regarded initially as at low risk, with a decreased ejection fraction and complex ventricular arrhythmia late after acute myocardial infarction, could have been classified as at moderate risk. Surprisingly, this patient is the only one who is still alive. The other 3 patients are still regarded as having been at low risk and CA was an unexpected event.

The possible relationship between silent ischaemia and sudden death15-17 and the prevalence of silent ischaemia as estimated by Cohn,18 justify the need for identifying subjects with asymptomatic ischaemic episodes who could be at increased risk of sudden death, especially in a cardiac rehabilitation setting. This view is not necessarily universally accepted.19,20 Stricter precautions could be taken in those patients with silent ischaemia who have had a previous inferior infarction and disease of the right coronary artery. It seems that risk stratifications should be reviewed, as angiographic findings and the occurrence of asymptomatic ischaemic episodes are at present not considered and could become relevant in patients exercising in cardiac rehabilitation programmes.

### TABLE II. PROFILE OF PATIENTS WHO SUFFERED A CA WHILE EXERCISING AT THE JOHANNESBURG CARDIAC REHABILITATION CENTRE, SEPTEMBER 1982 – JULY 1988

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yrs)</th>
<th>Occupation</th>
<th>Risk factors</th>
<th>Angiography</th>
<th>Complications</th>
<th>MI</th>
<th>Angiography</th>
<th>RCA</th>
<th>Left main</th>
<th>LAD</th>
<th>CX</th>
<th>Collaterals</th>
<th>EF</th>
<th>Medication</th>
<th>Stress test</th>
<th>Protocol</th>
<th>FC (M)</th>
<th>BP response (mmHg)</th>
<th>ST' depression</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>60</td>
<td>Professor</td>
<td>Ex-smoker</td>
<td>Normal</td>
<td>None</td>
<td>None</td>
<td>Anterior</td>
<td>100%</td>
<td>Normal</td>
<td>100%</td>
<td>Normal</td>
<td>Good</td>
<td>44%</td>
<td>Sotalol</td>
<td>Dec 1982</td>
<td>WHO²¹</td>
<td>7.9</td>
<td>160/80</td>
<td>No</td>
</tr>
<tr>
<td>2</td>
<td>64</td>
<td>Retired</td>
<td>Ex-smoker;</td>
<td>Not done</td>
<td>None</td>
<td>None</td>
<td>Superior</td>
<td>100%</td>
<td>Normal</td>
<td>100%</td>
<td>Normal</td>
<td>Normal</td>
<td>60%</td>
<td>Nifedipine</td>
<td>Apr 1983</td>
<td>Chung²²</td>
<td>9.2</td>
<td>110/70</td>
<td>No</td>
</tr>
<tr>
<td>3</td>
<td>63</td>
<td>Director</td>
<td>Ex-smoker;</td>
<td>Normal</td>
<td>None</td>
<td>None</td>
<td>Inferior</td>
<td>None</td>
<td>Normal</td>
<td>None</td>
<td>Normal</td>
<td>Good</td>
<td>60%</td>
<td>Atenolol</td>
<td>Dec 1985</td>
<td>WHO</td>
<td>3.5</td>
<td>140/90</td>
<td>147/80</td>
</tr>
<tr>
<td>4</td>
<td>55</td>
<td>Presenter</td>
<td>Ex-smoker;</td>
<td>Not done</td>
<td>None</td>
<td>None</td>
<td>1st-year</td>
<td>None</td>
<td>1st-year</td>
<td>None</td>
<td>None</td>
<td>88%</td>
<td>60%</td>
<td>Diltiazem</td>
<td>Feb 1987</td>
<td>Chung</td>
<td>104</td>
<td>120/90</td>
<td>No</td>
</tr>
</tbody>
</table>

M = metabolic equivalents (METS) (1 MET = 3.5 mUkg⁻¹min⁻¹); MHR = maximal heart rate.

Conclusions

1. The incidence of CA at the Johannesburg Cardiac Rehabilitation Centre is acceptably low and comparable with other cardiac rehabilitation programmes. Exercise as prescribed at the Johannesburg unit can therefore be considered a safe form of medical intervention.

2. Patients at risk of CA during exercise were essentially not identifiable, since they did not come from a group currently recognised as at particularly high risk. A combination of inferior infarction with occluded dominant right coronary artery, good collateralisation, and asymptomatic ischaemia was the common factor present in all CA patients. The likelihood of these factors being predictors of sudden death requires further investigation.

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REFERENCES