



## Review

## 40 years of cardiac rehabilitation and secondary prevention in post-cardiac ischaemic patients. Are we still in the wilderness?



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## ABSTRACT

Cardiac rehabilitation (CR) is the sum of interventions required to ensure the best physical, psychological and social conditions so that patients with cardiac disease may assume their place in society and slow the progression of the disease. Exercise testing (ET) early after MI has been shown to result in earlier return to work than the non-performance of ET. Research quality CR has resulted in lower cardiovascular mortality and lower recurrent hospitalisation and has been shown to be cost-effective. However, the content of cardiac rehabilitation programmes varies considerably. The only randomised trial of CR as usually performed in the 'real world' showed that CR had no impact on cardiac death rates or any other outcome. Only 20–50% of eligible patients attend CR programmes and attendance at CR has not improved in the last 20 years despite major attempts to increase participation in CR. Alternative methods for provision of CR have been sought. These include home-based CR, case management approaches, and nurse coordinated prevention programmes. Telephone based programmes, such as The COACH Program, have been introduced to coach patients and improve behavioural and biomedical risk factors. These have been shown to improve risk factors better than usual patient care and to reduce recurrences of cardiac events after discharge from hospital due to MI. Expansion of novel approaches such as The COACH Program may help to counteract the non-attendance at CR.

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The Book of Exodus describes how Moses led the children of Israel out of Egypt towards the Promised Land. They spent 40 years wandering through the wilderness before reaching the Promised Land [1]. Unfortunately, this is the analogy of 40 years of cardiac rehabilitation and secondary prevention. This article – a narrative review – charts the wanderings of the children of cardiac rehabilitation and secondary prevention.

### 1. Definition of cardiac rehabilitation

This article is our sequel to the excellent overview of the subject by Goble and Worcester published in 1999 [2]. In their Best Practice Guidelines, Goble and Worcester gave four similar definitions of cardiac rehabilitation. Cardiac rehabilitation is “the sum of interventions required to ensure the best physical, psychological and social conditions so that patients with cardiac disease may preserve or assume their proper place in society and, through improved health behaviours, slow or reverse progression of the disease.” It should be noted that this definition

is not prescriptive in its methods for achieving its aims. As usually performed, cardiac rehabilitation usually involves components of exercise, education, and attention to psycho-social problems.

### 2. Origins of cardiac rehabilitation

Cardiac rehabilitation began in the English speaking world as a treatment of an iatrogenic disease. In 1939, Mallory et al. examined autopsy specimens of patients who had died after acute myocardial infarction (AMI) and concluded that it took 6 weeks for firm scar to develop after AMI [3]. Strict bed rest for 6 weeks was recommended for patients surviving AMI to prevent cardiac rupture. It was thought that premature activity after AMI could provoke cardiac rupture. This regimen of severe inactivity had certain consequences: severe weakness and extreme effort intolerance on mobilising after AMI; fear of activity after AMI; anxiety and depression; unemployment; and unusual syndromes such as the shoulder hand syndrome. In 1944, in a series of papers published in JAMA under the heading “Abuse of rest as a therapeutic measure for patients with cardiovascular disease”, [4,5] this regimen was deemed physiologically questionable and hazardous. In 1952, Levine and Lown introduced the “chair treatment after AMI” pleading that sitting in an armchair and using a commode chair, if necessary, required less effort

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than climbing on to a pan whilst lying in bed [6]. The 1950s saw the use of the term cardiac rehabilitation and the start of its development as a treatment [7–9]. Indeed, Hellerstein and Ford stated that “the process of rehabilitation begins at the moment the patient is first stricken with his disease” [9]. By the early 1970s, the period of bed rest had been reduced to a few days, partly as a result of the “chair treatment”, and partly as a result of underpowered randomised controlled trials [10–15]. At that time, a classic experiment on 3 normal young men and 2 athletes showed that 3 weeks of bed rest could reduce exercise tolerance by 50% and that exercise training (reconditioning) could reverse this feature [16]. This became the rationale for Wenger’s 14-step mobilisation programme which became the basis of outpatient cardiac rehabilitation [17].

### 3. Exercise testing

Jelinek et al. were interested in using exercise testing as a means of helping to advise patients on their physical activity and capacity to return to work, leisure, and sexual activity after uncomplicated myocardial infarction [18]. Thirty (16%) of 189 consecutive patients met the criteria for low risk and were submitted to a symptom limited exercise test eight days after myocardial infarction. Eighteen of these patients underwent a repeat exercise test 5 weeks after the infarction. Exercise duration increased by 18% from week 1 to week 5. This was accompanied by an increase in maximal heart rate from 135.6 to 153.9 beats per minute without changes in the resting or submaximal heart rate or systolic blood pressure. Jelinek et al. concluded that exercise tolerance was almost normal 8 days after an uncomplicated myocardial infarction and that subsequent improvement in exercise performance was related to improvements in the patients’ self-confidence. This allowed cardiologists to plan the individual patient’s rehabilitation and suggested that most patients were fit to resume work 3 or 4 weeks after their uncomplicated heart attack. Haskell and DeBusk confirmed these observations using symptom limited exercise tests at weeks 3, 7, and 11 after uncomplicated myocardial infarction [19]. Similar results were also seen by Wohl et al. at 3 and 6 weeks after myocardial infarction [20]. Jelinek et al.’s exercise testing regimen became the basis for their multi-disciplinary outpatient cardiac rehabilitation clinic [21]. They showed that these selected patients could return to work earlier than previously believed. Indeed, a sentinel case occurred when one patient who was not yet fit to return to work built an extra room on to his house whilst “recovering” from his AMI. Jelinek et al. showed that there were no complications from this programme over the next 5 years [22, 23]. They published their walking programmes in which patients were encouraged to walk 5 km in 1 h three weeks after an uncomplicated myocardial infarction (MI) [24]. This information did not resonate with the medical community and was only applied in a few hospitals. Dennis et al. also showed that the performance of an exercise test early after MI shortened the time patients took to return to work [25]. A subsequent study was performed on 121 consecutive patients to assess whether practising cardiologists could define a low risk clinical group suitable for early mobilisation three days after acute MI and whether actually mobilising them vigorously from day 4 to day 7 affected their left ventricular function when compared with a randomised group rested until day 8 post-admission. This study emphatically showed that clinicians could clearly stratify risk at day 3 after their heart attack and that early vigorous mobilisation did not adversely affect left ventricular function [26]. A similar randomised study showed that exercise training after anterior Q wave MI did not adversely affect left ventricular function [27]. After introducing near maximal exercise testing 3 days after MI [28], Topol et al. randomly assigned 40 patients with uncomplicated AMI and a negative exercise stress test 3 days after their infarction to discharge on day 3 versus the randomly assigned identical group who were discharged after day 7 following their infarction. There were no adverse effects of their early discharge and the patients discharged early returned to work at a mean time of 40.7 days

compared with 56.9 days in the group discharged later. A great reduction in the costs of treatment was demonstrated in this low risk group [29].

### 4. Subsequent advances in cardiac rehabilitation

Cardiac rehabilitation, including secondary prevention, has advanced in several ways since the 1980s: well-designed randomised controlled studies; the recognition of psychosocial factors including depression accompanying heart disease as a problem requiring therapy; evidence that structured cardiac rehabilitation, including secondary prevention, reduces mortality and morbidity; and evidence of the cost-effectiveness of cardiac rehabilitation. DeBusk’s group performed a series of elegant randomised trials which confirmed and expanded on some of Jelinek’s observations. They found that home training was equally safe and effective as medically supervised training and superior to no training in terms of aerobic capacity [30,31]. Goble and Worcester showed that moderate intensity exercise resulted in similar improvements to high intensity exercise in the health state of men after transmural AMI. In fact, high intensity exercise resulted in an improvement of about 1 met in maximal exercise capacity when compared to moderate intensity exercise programmes, and this improvement was lost over the 12 months after the acute event [32,33], most likely because of non-adherence to the high intensity programme. This has led to the promulgation of light exercise and education programmes as the basis of cardiac rehabilitation in Australia and the UK [34].

Programmes based on light exercise education without the need of a preliminary exercise test are appropriate and are being used in many developing countries around the world.

### 5. Depression in cardiac rehabilitation

Although anxiety and depression have long been recognised as common sequelae of AMI [35], the independent adverse prognostic significance of depression after MI was brought home to the medical profession by Frasure-Smith and Lesperance [36–38]. Since then many papers have reported their experiences in favour and against the relationship between depression and coronary heart disease. Systematic reviews have shown that depression is an independent risk factor for the development and prognosis of coronary heart disease [39,40] and a review of reviews showed that the more depressed the patient the worse the prognosis of the patient [41]. Depression has been found to adversely affect prognosis in patients with unstable angina, atrial fibrillation, heart failure, and with implanted defibrillators [42–46] and to be associated with a number of non-cardiac disorders including stroke, diabetes, asthma, cancer and arthritis [47]. The ENRICH study which looked at the impact of cognitive behavioural therapy in a randomised trial in 2481 patients with coronary heart disease found that the intervention improved depression and social isolation but did not increase event free survival [48]. A recent Cochrane review supported this conclusion and added that interventions which educated patients on cardiac risk factors or included patient led discussions and emotional support and included family members into the discussion were effective interventions on depression [49]. The use of sertraline or citalopram has been shown to be effective in reducing depression in patients warranting this form of treatment [50,51]. On the basis of this evidence the American Heart Association has recommended screening for depression with subsequent referral and treatment [52]. It is not clear whether there are sufficient numbers of qualified clinicians who can provide this treatment which is being recommended. However, Blumenthal and colleagues have shown that a third treatment, exercise training, was effective in reducing depression in patients with coronary heart disease and also in heart failure [53–55]. This supports the use of aerobic conditioning after acute coronary syndromes.

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