Annals of Physical and Rehabilitation Medicine xxx (2018) xxx–xxx

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Original article

Short report: Effect of exercise training on left ventricular mechanics after acute myocardial infarction–an exploratory study

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ARTICLE INFO

Article history:
Received 29 October 2017
Accepted 28 January 2018

Keywords:
Longitudinal strain
Left ventricular twist
Left ventricular remodelling

ABSTRACT

Background: Cardiac rehabilitation (CR) exercise training is beneficial after myocardial infarction (MI). Whilst the peripheral adaptations to training are well defined, little is known regarding the effect on left ventricular (LV) remodelling, particularly LV function. Efficient LV ejection and filling is achieved through deformation and rotation of the myocardium in systole and diastole – LV mechanics. The response of LV mechanics to CR exercise training in MI patients is unknown.
Methods: In this observational exploratory study, 36 (of 40 enrolled) male, MI patients completed either 10-weeks of twice-weekly gym based cardiovascular exercise at 60–80% VO_{2peak} ($n = 18$), or a non-exercise control period ($n = 18$). Cardiopulmonary exercise testing and speckle tracking echocardiography were performed at baseline and 10 weeks.
Results: Compared to the non-exercise group, VO_{2peak} improved with CR exercise training (Difference: +4.28 [95% CI: 1.34 to 7.23] $ml.kg^{-1}.min^{-1}$, $P = 0.01$). Neither conventional LV structural or functional indices, nor LV global longitudinal strain, significantly changed in either group. In contrast, LV twist and twist velocity decreased in the exercise group and increased in the non-exercise group (Difference: −3.95° [95% CI: −7.92 to 0.03°], $P = 0.05$ and $-19.2°.s^{-1}$ [95% CI: -35.9 to $-2.7°.s^{-1}$], $P = 0.02$, respectively).
Conclusion: In MI patients who completed CR exercise training, LV twist and twist velocity decreased, whereas these parameters increased in patients who did not exercise. These preliminary data may indicate reverse LV functional remodelling and improved functional reserve. The assessment of LV twist may serve as an indicator of the therapeutic benefit of CR exercise training and should be investigated in larger trials.

1. Introduction

Pathologic left ventricular (LV) remodelling is a significant problem after myocardial infarction (MI) [1]. In MI patients, cardiac rehabilitation (CR) exercise training improves functional capacity [2]. Whilst the contribution of peripheral adaptation to this improvement is relatively well understood [3], little is known about LV remodelling, particularly in relation to function. Recent data support exercise induced reverse LV remodelling, characterised by reduced LV volumes and increased ejection fraction [4]. However, LV systolic function is not well defined by ejection fraction alone. Rather, the motion of the LV during systole and diastole is a complex pattern of multi-directional deformation and rotation (LV mechanics) that underpins efficient systolic ejection and diastolic filling. Obliquely and opposingly wound endocardial and epicardial myofibres are electrically and mechanically activated, in sequence, from apex to base [5] resulting in longitudinal shortening (LV strain) [6] and LV twist (i.e. clockwise basal rotation and anticlockwise apical rotation) [7]. During diastolic untwist, rapid recoil sucks blood into the LV, utilising energy stored in the preceding twist.

Left ventricular mechanics are compromised by ischemic heart disease [8] and are known to predict pathologic remodelling after MI [9,10]. Specifically, LV longitudinal strain and twist are acutely impaired immediately following MI, and progressively decline with chronic pathologic remodelling [9,11]. In contrast, LV mechanics are likely to be preserved in the presence of reverse LV remodelling [9,11]. Given the association between LV mechanics and remodelling, the assessment of LV longitudinal strain and

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https://doi.org/10.1016/j.rehab.2018.01.003
1877-0657/

Please cite this article in press as: McGregor G, et al. Short report: Effect of exercise training on left ventricular mechanics after acute myocardial infarction–an exploratory study. Ann Phys Rehabil Med (2018), https://doi.org/10.1016/j.rehab.2018.01.003

twist may help determine the effect of CR exercise training on functional LV remodelling after MI.

By assessing patients with 2D speckle tracking echocardiography (STE), before and after a CR programme, the main objective of this exploratory study was to examine the effect of exercise training on LV longitudinal strain and twist in MI patients.

2. Materials and methods

2.1. Ethical considerations

This study was conducted in line with the principles of Good Clinical Practice, and complied fully with the Declaration of Helsinki. Research and Ethics Committee approval was gained (Coventry & Warwickshire (08/H1210/56) and informed consent obtained from all participants.

2.2. Study design

A prospective, non-randomised observational study was conducted. Forty male participants were allocated to an exercise ($n = 20$) or non-exercise group ($n = 20$) (Fig. 1). Ethical constraints and institutional policy prevented randomisation, however, groups did not differ demographically or clinically at baseline.

2.3. Participants and procedures

Conventional echocardiography, STE and cardiopulmonary exercise testing (CPET) were performed at baseline and after a 10-week exercise programme or non-exercise control period. Participants were optimally treated with percutaneous coronary intervention for acute MI. Complete revascularisation was undertaken and no clinically significant atherosclerotic lesions remained untreated. Clinical stability [12] was medically certified and further confirmed by the absence of exercise induced angina and/or ischemic ECG changes during cardiopulmonary exercise testing. Patients were excluded if comorbidities prevented full participation.

2.4. Exercise training

Gym based, continuous, moderate intensity cardiovascular exercise training (i.e. treadmill, cycle, rower, cross-trainer) was completed for 25–40 mins, twice weekly for 10 weeks. A 10-min progressive treadmill or cycle warm-up preceded the cardiovascular exercise. A standardised resistance machine training programme (1 set, 12 reps, 5 upper and 2 lower body exercises) [12] followed a five-minute cool down walk. Cardiovascular exercise workload was equivalent to 60–80% peak oxygen uptake ($VO_{2\ peak}$) from CPET, and after two sessions, the supervising Exercise Physiologist ensured that participants were exercising at a heart rate equivalent to 80% $VO_{2\ peak}$. Individualised exercise intensity was systematically re-prescribed every two weeks based on perceived exertion, and duration was incrementally increased to 40 minutes by week-five. Adherence of ≥ 17 of 20 sessions was required for inclusion in the analysis. The non-exercise group did not complete supervised CR exercise training but were advised on a cardio-protective lifestyle.

2.5. Cardiopulmonary exercise test (CPET)

In accordance with guidelines [13], a ramp CPET was performed on a cycle ergometer. Respired gas was analysed for oxygen uptake (VO_2), carbon dioxide production (VCO_2) and minute ventilation (V_E) (Oxycon Pro, Care Fusion Corp, San Diego, California, USA). ECG was continuously recorded and blood pressure measured at two-minute intervals. Tests were continued until symptom-limited volitional fatigue. A respiratory exchange ratio of > 1.15 was considered representative of maximal effort. Peak oxygen uptake ($VO_{2\ peak}$) was taken to be the mean O_2 uptake in the final 20 seconds of the test, whilst O_2 uptake at the anaerobic threshold

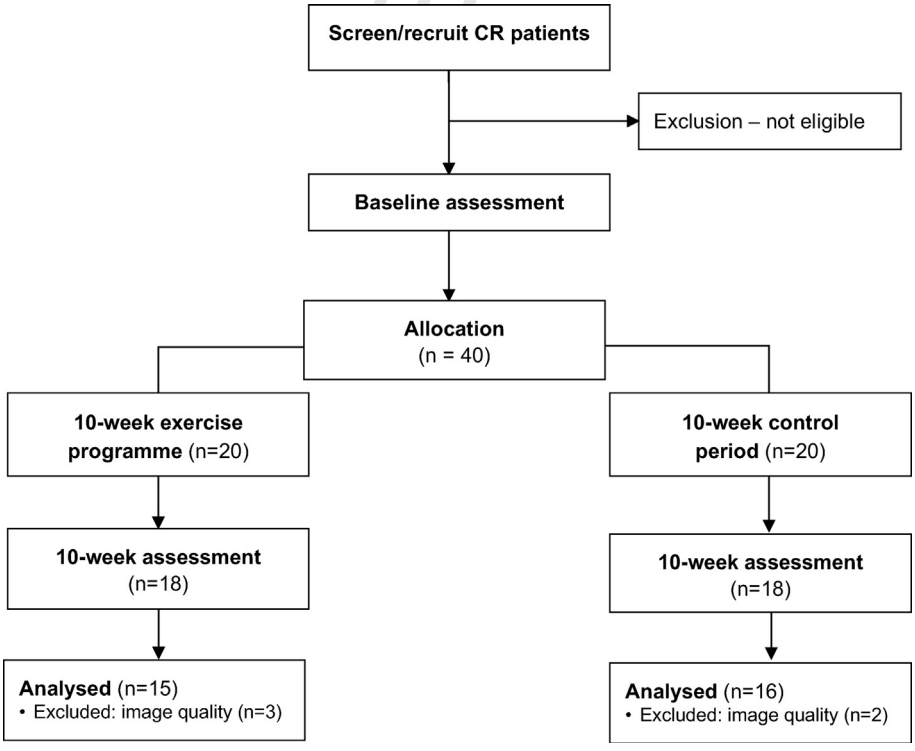

Fig. 1. Study flowchart.

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