Extended short report

Effects of resistive breathing on exercise capacity and diaphragm function in patients with ischaemic heart disease

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Abstract

Background: Muscle weakness has been suggested to result from the deconditioning that accompanies decreased activity levels in chronic cardiopulmonary diseases. The benefits of standard exercise programmes on exercise capacity and muscular strength in disease and health are well documented and exercise capacity is a significant predictor of survival in patients with chronic heart failure (CHF). Selective respiratory muscle training has been shown to improve exercise tolerance in CHF and such observations have been cited to support the suggestion that respiratory muscle weakness contributes to a reduced exercise capacity (despite biopsies showing the metabolic profile of a well trained muscle). *Aims:* This study aimed to determine the effects of selective inspiratory muscle training on patients with chronic coronary artery disease to establish if an improved exercise capacity can be obtained in patients that are not limited in their daily activities. *Methods:* Nine male patients performed three exercise tests (with respiratory and diaphragm function assessed before the third test) then undertook a 4-week programme of inspiratory muscle training. Exercise tolerance, respiratory and diaphragmatic function were re-assessed after training. *Results:* Exercise capacity improved from 812 ± 42 to 864 ± 49 s, $P < 0.05$, and velocity of diaphragm shortening increased (during quiet breathing from 12.8 ± 1.6 to 19.4 ± 1.1 mm s^{-1}, $P < 0.005$, and sniffing from 71.9 ± 9.4 to 110.0 ± 12.3 mm s^{-1}, $P < 0.005$). In addition, five from nine patients were stopped by breathlessness before training; whereas only one patient was stopped by breathlessness after training. *Conclusion:* The major findings in this study were that a non-intensive 4-week training programme of resistive breathing in patients with chronic coronary artery disease led to an increase in exercise capacity and a decrease in dyspnoea when assessed by symptom limited exercise testing. These changes were associated with significant increases in the velocity of diaphragmatic excursions during quiet breathing and sniffing. Patients that exhibited small diaphragmatic excursions during quiet breathing were most likely to improve their exercise capacity after the training programme. However, the inspiratory muscle-training programme was not associated with any significant changes in respiratory mechanics when peak flow rate, forced expiratory volume and forced vital capacity were measured. The resistive breathing programme used here resulted in a significant increase in the velocity of diaphragm movement during quiet breathing and sniffing. In other skeletal muscles, speed of contraction can be determined by the relative proportion of fibre types and muscle length (Jones, Round, Skeletal Muscle in Health and Disease. Manchester: University Press, 1990). The intensity of the training programme used here, however, is unlikely to significantly alter muscle

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morphology or biochemistry. Short-term training studies have shown that there can be increases in strength and velocity of shortening that do not relate to changes in muscle biochemistry or morphology. These changes are attributed to the neural adaptations that occur early in training (Northridge et al., Br. Heart J. 1990; 64: 313–316). Independent of the mechanisms involved, this small, uncontrolled study suggests that inspiratory muscle training may improve exercise capacity, diaphragm function and symptoms of breathlessness in patients with chronic coronary artery disease even in the absence of heart failure.

1. Methods

1.1. Study population

Nine male patients aged 50–67 (mean 62.0 ± 1.9) years with established coronary heart disease but no signs of heart failure, recruited from a cardiology outpatient clinic, were studied. Further details of these patients are given in Table 1. The study was approved by the locally appointed ethics committee and informed consent was obtained from all subjects. The investigation conforms with the principles outlined in the Declaration of Helsinki.

1.2. Study outline

The study had three components that comprised an initial baseline study phase, a training phase and then a post-training phase.

1.2.1. Baseline studies

Three symptom-limited treadmill exercise tests were performed within a 2-week period (see below for details). The patients were asked to attend 2–4 h after a light meal and treadmill tests were repeated, at least 2 days apart, on three separate occasions to allow familiarisation and minimise any training effect on treadmill walking. Standard respiratory tests were performed and then diaphragmatic function was as-

sessed by ultrasonography prior to the patient undertaking the last exercise test.

1.2.1.1. Exercise tolerance tests. The patients exercise capacity was assessed using a standard treadmill symptom-limited exercise tolerance test. The test followed a continuous graded protocol with regular increments in speed and elevation (the STEEP protocol [1]). The patients exercise capacity was assessed during the third baseline exercise test to minimise any training effect on treadmill walking during the inspiratory training period. Comparison of the measured heart rate response to exercise against age and gender predicted maximums was used to indicate the patients relative level of effort. Immediately upon cessation of exercise the patients were asked to give the reason for them having stop exercising.

1.2.1.2. Respiratory tests. Peak flow rate, forced expiratory volume (in the first second) and forced vital capacity were measured using a spirometer. After familiarisation, the mean of three trials was obtained to indicate basic respiratory function immediately prior to assessment of exercise capacity.

1.2.1.3. Diaphragm ultrasonography. Ultrasound records of diaphragmatic excursion were obtained using an Acuson 128XP ultrasound imaging system with a 4.0-MHz transducer (Acuson, CA, USA). An intercostal probe position was chosen between the mid-axillary and mid-clavicular line. The transducer was

Table 1
Subject characteristics

Subject	Age	Weight (kg)	Height (cm)	Status	EF[a] (%)	VO_{2max}[b]
1	62	79.83	170	CABG[c]	44	37.3
2	66	69.85	173	MI[d]	42	27.2
3	56	92.08	170	IHD[e]	48	38.8
4	67	79.83	168	MI	45	40.9
5	66	82.55	180	IHD	52	48.7
6	65	69.85	170	CABG	50	41.8
7	61	71.67	175	MI	40	26.7
8	64	73.83	173	IHD	50	40.9
9	50	76.20	175	CABG	49	46.6

[a] Ejection fraction calculated echocardiographically by the Simpson's bi-plane method.
[b] Extrapolated maximum oxygen consumption (ml kg^{-1} min^{-1}).
[c] Previous coronary artery by-pass graft.
[d] Previous myocardial infarction.
[e] Ischemic heart disease; ischemic heart disease was clinically defined as the presence of > 2 mm ST during an exercise test that was limited by angina or breathlessness.

held in a fixed skin position during all phases of respiration. Scans of the right hemi-diaphragm were performed in a longitudinal plane, which included the maximal renal bipolar length. Movement of the chest wall and abdomen was observed to exclude paradoxical movement of the diaphragm during respiration. The posterior–anterior (PA) excursion of the right hemi-diaphragm was measured by M-mode during quiet breathing. The cursor line was placed at the posterior identifiable extent of the crura on expiration. The M-mode cursor was placed at an angle of not less than 70° to the craniocaudal displacement line. From the M-mode trace, the maximum PA displacement of that part of the diaphragm for each respiratory cycle was determined and at least six consecutive respiratory cycles in quiet breathing were recorded. PA excursion was also measured during a voluntary sniff (defined as a rapid inspiration through the nose with similar PA displacement to that in quiet breathing, i.e. not a deep breath in). In addition to measuring the magnitude of diaphragmatic excursion, the velocity of diaphragmatic movements were also calculated during quiet breathing and sniffing.

1.2.2. Inspiratory muscle training

On completing preliminary baseline studies, the patients began a 4-week progressive inspiratory muscle training programme. The programme consisted of three supervised training sessions per week with each session lasting for 1 h. The training took the form of resistive breathing where the patients were required to inspire through a narrow tube attached to a Hans–Rudolph non-return valve while wearing a nose clip. The tube had an internal diameter of 5 mm. Each hour-long session consisted of 5 min resistive breathing followed by 10 min rest (with four repetitions per session). Progressive training was achieved by lengthening the inspiratory tube; for the first week only the diameter of the Hans–Rudolph inlet valve was restricted, but then the tubing length was progressively increased over weeks 2, 3 and 4 (by 33 cm per week).

1.2.3. Post-training studies

On completion of the inspiratory muscle training programme, the patients were asked to repeat the assessment of exercise capacity, respiratory tests and diaphragm function as described above. Only one exercise test was performed post-training.

1.3. Statistical analysis

At baseline the relationship between exercise tolerance, lung function and diaphragm ultrasonography measurements were examined by means of Pearson's correlation coefficients. The effects of training on
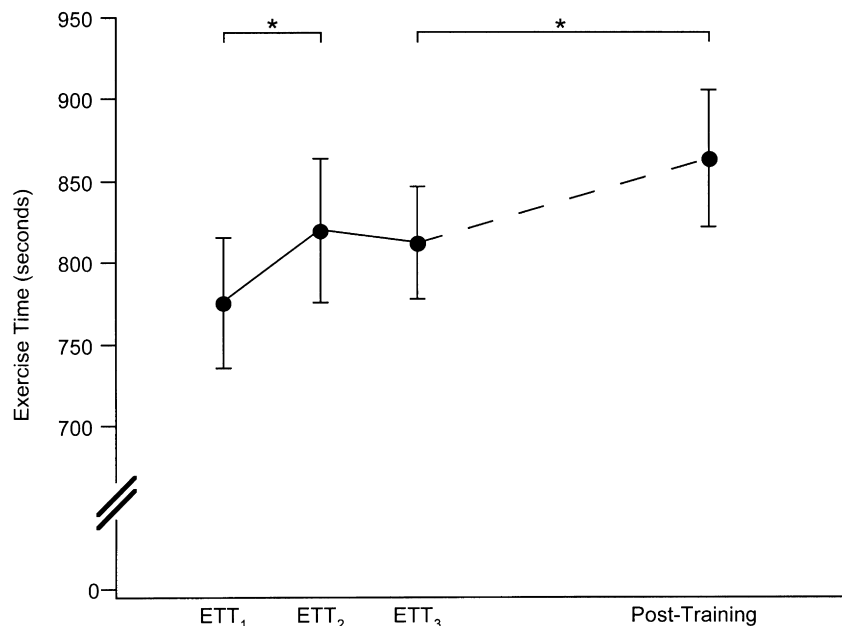

Fig. 1. Effects of inspiratory muscle training on exercise capacity. Mean ± S.E.M. exercise duration are plotted at each of the exercise tolerance tests performed during the baseline studies and after the inspiratory training programme. There is a significant increase in exercise duration between the first test (ETT_1) and the second test (ETT_2) (*$P < 0.05$) and between the third test (ETT_3) and the post-training test (*$P < 0.05$). However, there is no difference in exercise duration between ETT_2 and ETT_3 illustrating that there is no 'training effect'.

Table 2
Effects of selective inspiratory training on the magnitude and velocity of diaphragmatic excursion

	Baseline	Post-training
Q-ex[a] (mm)	17.4 ± 2.6	20.4 ± 1.8
Q-v[b] (mm s^{-1})	12.8 ± 1.6	19.4 ± 1.1
S-ex[c] (mm)	19.3 ± 2.3	24.5 ± 3.2
S-v[d] (mm s^{-1})	71.9 ± 9.4	110.0 ± 12.3

[a] PA excursion of the right hemi-diaphragm during quiet breathing.
[b] Velocity of the right hemi-diaphragm movement during quiet breathing.
[c] PA excursion of the right hemi-diaphragm during a sniff.
[d] Velocity of the right hemi-diaphragm movement during a sniff.

these measures were evaluated using paired Student's t-test. For all statistical relationships significance was taken at the 5% level. Data are expressed as mean $\pm$ S.E.M.

2. Results

2.1. Heart rate response to exercise

Measurements of heart rate response during the third exercise tolerance test before training showed that all patients made a good effort attaining heart rates of $92 \pm 4\%$ of their age-predicted maximum. After training, almost all patients achieved higher maximum heart rates (the groups mean maximum heart rate response increasing from 141 ± 5.7 to 151 ± 6.0 b.p.m.) representing $96 \pm 5\%$ of their age-predicted maximum. There was no significant change in resting heart rate levels following resistive training (70.8 ± 9.9 b.p.m. at the third baseline exercise test vs. 75.3 ± 7.7 b.p.m. post-training, $P = $ NS).

2.2. Exercise capacity

Fig. 1 shows the inspiratory muscle training resulted in a significant increase in exercise capacity within this group of patients (total exercise time was 812 ± 42 s at the third baseline exercise test vs. 864 ± 49 s post-training, $P < 0.05$). Moreover, there was no difference in exercise duration between the second and third exercise tests performed confirming that the patients had adequate experience of treadmill walking at the end of the baseline studies.

2.3. Respiratory tests

Inspiratory muscle training made no significant difference to peak flow rate, forced expiratory volume (in the first second) and forced vital capacity. How-

ever, there was a strong trend for peak flow rate to increase following the training regime (peak flow rate was 504.2 ± 12.5 l min^{-1} at baseline vs. 529.8 ± 13.8 l min^{-1} after training, $P = 0.052$).

2.4. Diaphragm ultrasonography

The effects of selective inspiratory training on the magnitude and velocity of diaphragmatic excursion are shown in Table 2. In essence, there was no significant difference in the magnitude of diaphragm excursion during quiet breathing (Q-ex) or sniffing (S-ex). However, inspiratory muscle training did result in significant changes in the velocities of diaphragm movement (the velocity of diaphragm excursion during quiet breathing [Q-v] increased from 12.8 ± 1.6 to 19.4 ± 1.1 mm s^{-1} after training, $P < 0.005$; the velocity of diaphragm excursion during sniffing [S-v] increased from 71.9 ± 9.4 to 110.0 ± 12.3 mm s^{-1} after training, $P < 0.005$).

2.5. Limiting symptom during exercise

Dyspnoea was the reason for terminating the exercise test in five from the nine patients studied prior to the inspiratory muscle training programme. However, on completion of the training programme was only one was limited by dyspnoea (the others were limited by leg fatigue).

2.6. Ultrasound measures as predictors of change post-training

Q-ex was a negative predictor of changes in exercise capacity ($r = -0.80$), i.e. individuals with small diaphragmatic excursions during quiet breathing were most likely to improve their exercise capacity after the training programme. In addition, Q-ex correlated inversely with changes in Q-ex ($r = -0.76$), S-ex ($r = -0.90$) and S-v ($r = -0.77$) so that patients with smaller diaphragm excursions during quiet breathing before training were more likely to increase these measures of diaphragm function with training. A similar finding was found with Q-v which inversely correlated with changes in Q-v ($r = -0.79$), S-ex ($r = -0.68$) and S-v ($r = -0.77$).

Reference

[1] Northridge DB, Grant S, Ford I et al. A novel exercise protocol suitable for use on a treadmill or a bicycle ergometer. Br Heart J 1990;64:313–316.