

ORIGINAL ARTICLE

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Walking performance and economy in chronic heart failure patients pre and post exercise training

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Abstract The effect of a 3-week exercise programme on performance and economy of walking was analysed in 16 male patients with chronic heart failure [mean age 51.8 (SD 6.9) years, height 174.9 (SD 6.3) cm, body mass 75.3 (SD 11.5) kg, ejection fraction 20.8 (SD 5.0)%]. They were submitted to a cardiopulmonary exercise test on a cycle ergometer and a 6-min walking test on a treadmill before and after the period of exercise training. The training programme consisted of interval cycle (five times a week for 15 min), and treadmill ergometer training (three times a week for 10 min) at approximately 70% cycling peak oxygen uptake ($\dot{V}O_{2\text{peak}}$) and supplementary exercises (three times a week for 20 min). Compared to the pre values cycling $\dot{V}O_{2\text{peak}}$ [11.9 (SD 2.9) vs 14.0 (SD 2.3) $\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$], maximal self paced walking speed [0.68 (SD 0.33) vs 1.16 (SD 0.30) $\text{m} \cdot \text{s}^{-1}$], and net walking power [2.16 (SD 0.89) vs 2.73 (SD 0.91) $\text{W} \cdot \text{kg}^{-1}$] had increased ($P < 0.01$) while net energy cost [3.31 (SD 0.66) vs 2.33 (SD 0.38) $\text{J} \cdot \text{kg}^{-1} \cdot \text{m}^{-1}$] had decreased ($P < 0.001$) after the training period. Approximately 42% of the increase of walking speed resulted from a higher walking power output, whereas approximately 58% corresponded to a positive effect on walking economy. The improvement in walking economy was a function of an increase in walking velocity itself and a result of a more efficient walking technique. These results would indicate that in patients with marked exercise intolerance, adequate exercise training programmes could contribute to favourable metabolic changes with positive effects on the economy of motion.

Key words Walking test · Metabolic power · Energy cost · Exercise training · Chronic heart failure

Introduction

Walking capacity is an important factor determining quality of life in patients with chronic heart failure (CHF). Walking tests have been used to investigate the outcome of rehabilitation programme in pulmonary patients (Cockcroft et al. 1981; McGavin et al. 1977; Sinclair and Ingram 1980), and patients with severe CHF (Meyer et al. in press), and have been proposed as simple means for assessing functional capacity in patients with CHF (Ajayi and Balogun 1991; Parameshwar et al. 1989; Pardy et al. 1981). Maximal distance walked has been found to decrease progressively in patients assessed as New York Heart Association class I to III (Lipkin et al. 1986; Riley et al. 1992), and has been shown to be an independent predictor of hospitalization rate and mortality in CHF patients (Bittner et al. 1993). A high correlation between psychosocial adjustment to advanced CHF and walking capacity has been demonstrated (Dracup et al. 1992).

In natural and competitive walking the energy cost of locomotion has been shown to depend on walking technique and speed (di Prampero 1986; Hagberg and Coyle 1984; Menier and Pugh 1968; Zaciorskij 1987). Neuromuscular diseases have been found to lead to significant increases in the energy cost of walking, which have been shown to be partly reversible with exercise training (Olgiatei et al. 1986). In CHF patients with severe exercise intolerance, it is not known how far an increase in walking test performance represents a training induced benefit of metabolic capacity, or a change in walking economy. The aim of the present study was to analyse the effect of a 3-week exercise programme on performance and economy of walking in CHF patients.

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Methods

Subjects

A group of 16 male patients with severe CHF [mean age 51.8 (SD 6.9) years, height 174.9 (SD 6.3) cm, body mass 75.3 (SD 11.5) kg, echocardiographic ejection fraction 20.8 (SD 5.0)%] participated in the study. Of the group, 8 patients were on a heart transplant waiting list. No patient had acute myocardial and/or peripheral ischaemia, serious rhythm disorders, orthopaedic and/or neurological problems, and all were in stable sinus rhythm. Lung function (percentage predicted) was borderline normal, but typical for CHF patients [forced vital capacity 84.7 (SD 17.4)%, forced expiratory volume in 1 s 81.1 (SD 19.5)%]. Longterm medication included ACE inhibitors ($n = 15$), digitals ($n = 13$), nitrates ($n = 12$), beta-blockers ($n = 8$), diuretics ($n = 13$), and phenprocoumon ($n = 14$). The study was approved by the State Ethics Committee, and all the patients gave written informed consent prior to participation.

Test procedure

All the patients were familiarized with the exercise tests and laboratory procedures. Before and after the 3-week exercise training period each patient completed a cardiopulmonary exercise test on a cycle ergometer and a 6-min walking test on a treadmill.

Cardiopulmonary exercise testing

Cardiopulmonary exercise testing was conducted on an electro-dynamically braked cycle ergometer (Lode, Mijnhard, Netherlands). After 3 min unloaded pedalling, the exercise intensity was increased by $12.5 \text{ W} \cdot \text{min}^{-1}$ up to muscle fatigue and/or severe dyspnoea. Oxygen uptake ($\dot{V}\text{O}_2$) and carbon dioxide production ($\dot{V}\text{CO}_2$) were measured breath-by-breath (Oxycon Sigma, Mijnhard, Netherlands). Heart rate and heart rhythm were recorded continuously from a 12-lead electrocardiogram (Cardiovit, Schiller, Switzerland). Blood pressure was measured by plethysmomanometry (Finapres, Ohmeda, USA). At the end of every minute of exercise 20- μl capillary blood was collected from an hyperaemic ear lobe for enzymatic-amperometric blood lactate (BLC) determination from haemolyzed blood (ESAT, Eppendorf).

Walking test

The 6-min walking test was conducted on a treadmill (Q5, Quinton, USA). Cardiopulmonary and BLC data were determined as described for the cycling ergometry protocol. Acute adjustment of $\dot{V}\text{O}_2$ and BLC require a period of constant exercise longer than 1 min. Therefore, all walking test data are presented as mean values calculated from the final 3 min of walking. After a reference phase of 5 min, the patients were encouraged to walk as far as possible without provoking dyspnoea or other symptoms, such as chest pain and vertigo. Treadmill speed was controlled by the patients minute by minute, being aware of their walking time throughout the treadmill protocol.

Metabolic power and energy cost of walking

Net metabolic power was calculated from $\dot{V}\text{O}_2$ and $\dot{V}\text{CO}_2$ above rest values, energy equivalent, net lactate production per unit of time and body mass by: metabolic power (watts per kilogram) = $\dot{V}\text{O}_2$ (millilitres per kilogram per second) · energy equivalent (joules per millilitre) + net lactate (millimoles per litre per second) · O_2 -lactate equivalent (millilitres per kilogram per millimoles per litre) · 21.131 (joules per millilitre). Assuming that an increase in $1.0 \text{ mmol} \cdot \text{l}^{-1}$ in blood lactate concentration is equivalent

to 13.8 J mechanical work per 1.0 kg of body mass, and that $1.0 \text{ ml } \dot{V}\text{O}_2$ provides 5.24 J, the O_2 lactate equivalent is $2.6 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{mmol}^{-1} \cdot \text{l}$. It has been reported that the latter calculation is compatible to a distribution space of lactate of approximately 45% of the body mass (di Prompero 1981; Mader and Heck 1986). The energy cost of walking was calculated by: energy cost (joules per kilogram per metre) = metabolic power (watts per kilogram) · speed⁻¹ (seconds per metre) (Brueckner et al. 1991; di Prampero 1986; Margaria et al. 1963). For analysis of the relationship between speed and metabolic power of walking pre and post-training, the metabolic power was predicted as a quadratic function of speed: metabolic power (watts per kilogram) = $a + b \cdot \text{speed}^2$ (metres per second squared) (Bobbert 1960; Cotes and Meade 1960; Ralston 1958; van der Walt and Wyndham 1973; Zaciorskij 1987; Zarrugh et al. 1974).

Exercise training

The 3-week exercise training programme consisted of cycle ergometer training (five times a week for 15 min each time), treadmill ergometer training (three times a week for 10 min each time), and supplementary exercises (three times a week for 20 min each time). All cycle and treadmill ergometer training sessions were supervised by continuous monitoring of heart rate and heart rhythm. Blood pressure was monitored every three minutes. The $\dot{V}\text{O}_2$, $\dot{V}\text{CO}_2$ and BLC were determined during the last cycling and walking training sessions. All training sessions were performed without chest pain or other symptoms.

The 15-min interval cycle ergometer training was divided into phases of 30-s exercise and 60-s recovery. Recovery intensity was 15 W. For exercise phases, the intensity was 50% of maximal short-time exercise capacity determined by a special steep ramp test (Meyer et al. 1996).

During the interval treadmill walking, the exercise intensity of the 60-s speed phases was adjusted according to the maximal heart rate tolerated during the cycle interval training. Each high speed phase was followed by 60-s low speed walking at $0.4 \text{ m} \cdot \text{s}^{-1}$. The weekly energy costs of cycle and treadmill training were approximately 650 kJ and 270 kJ, respectively.

In addition to ergometer training, three times per week patients performed a 20-min exercise that focused on flexibility, muscular movement co-ordination, isometric contractions of small muscle groups, and inspiratory capacity.

Statistics

The percentage of the variance explained by the quadratic curve fit was determined by correlation and orthogonal regression of the observed and predicted metabolic power for each speed and by squaring of the Pearson-product correlation coefficient. The Wilcoxon test was used to determine intra-individual mean differences. For all statistics, the significance level was set at $P < 0.05$.

Results

The 3-weeks of exercise training significantly increased 6-min maximal self-paced walking speed by 88.5 (SD 48.7)%, the corresponding $\dot{V}\text{O}_2$ by 15.7 (SD 15.6)%, and metabolic walking power by 33.7 (SD 39.5)% ($P < 0.01$). The BLC response to self-paced walking was unchanged, and the energy cost of walking decreased significantly by 27.3 (SD 17.5)% (Table 1) ($P < 0.001$).

Improved walking economy was indicated by the curve being shifted to the right, when metabolic power of walking (watts per kilogram) was predicted as a function of walking speed (v) as $1.410 + 1.346 v^2$ pre-

Table 1 Walking speed, oxygen uptake ($\dot{V}O_2$) and blood lactate concentration (BLC) including rest values and net metabolic power and energy cost above rest values during the last 3 min of the 6-min walking test, pre and post exercise training

	Pretraining		Posttraining		P value
	mean	SD	mean	SD	
Speed ($m \cdot s^{-1}$)	0.68	0.33	1.16	0.30	0.001
$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	10.0	2.2	11.6	2.3	0.01
BLC ($mmol \cdot l^{-1}$)	1.0	0.3	1.1	0.2	n.s.
Power ($W \cdot kg^{-1}$)	2.16	0.89	2.73	0.91	0.01
Energy cost ($J \cdot kg^{-1} \cdot m^{-1}$)	3.31	0.66	2.33	0.38	0.001

training, and $1.122 + 1.122 v^2$ post-training (Fig. 1). Pre and post-training, 81% ($P < 0.001$) and 78% ($P < 0.001$) respectively, of the variance were explained by the quadratic curve fits. There was no difference between predicted and measured data ($P > 0.05$). The predicted metabolic power values calculated from individual values of walking speed were 2.16 (SD 0.80) $W \cdot kg^{-1}$ pre-training and 2.72 (SD 0.81) $W \cdot kg^{-1}$ post-training.

The increase in the self-paced walking speed resulted from a higher level of metabolic power of walking and a positive effect on walking economy. The isolated benefit of walking speed can be estimated by calculating a theoretical metabolic power based on the function of power and speed pretraining and walking speed post-training. According to average values, approximately 42% of the increase in speed resulted from a higher level of walking power, whereas approximately 58% was derived from changes in walking economy. The improvement in walking economy was a result of walking technique and walking speed (Figs. 1–3).

Peak cycle ergometer $\dot{V}O_2$ increased significantly from 11.9 (SD 2.9) $ml \cdot kg^{-1} \cdot min^{-1}$ to 14.0 (SD 2.3) $ml \cdot kg^{-1} \cdot min^{-1}$ ($P < 0.001$).

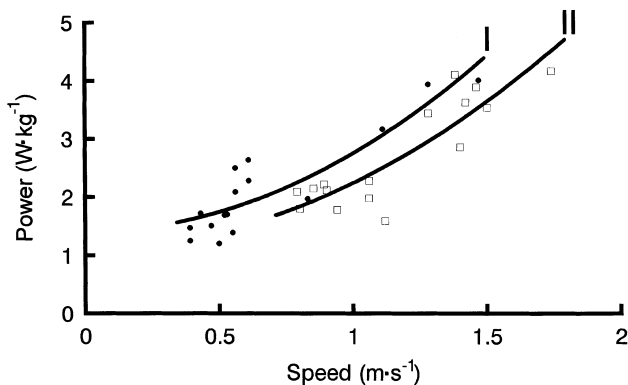


Fig. 1 Measured and predicted metabolic power of walking related to walking speed pre (● = measured, I = predicted) and post (□ = measured, II = predicted) exercise training

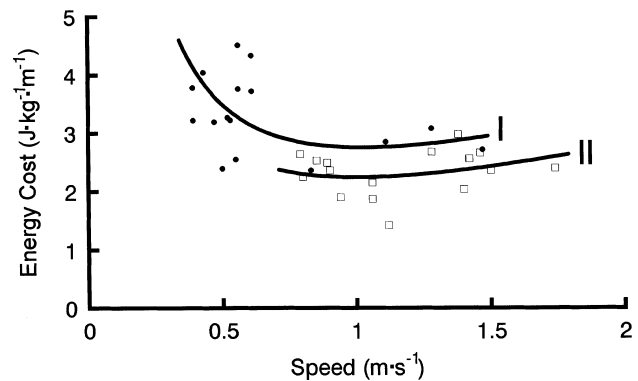


Fig. 2 Measured and predicted energy cost of walking related to walking speed pre (● = measured, I = predicted) and post (□ = measured, II = predicted) exercise training

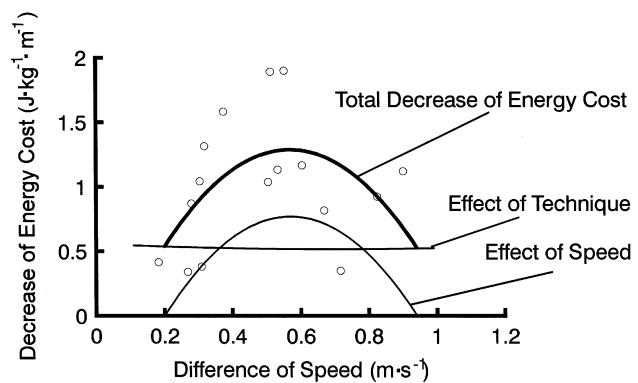


Fig. 3 Relationship between individual reduction of energy cost due to increase of walking speed (*effect of speed*), reduction of energy cost due to walking technique (*effect of technique*) and resulting total decrease of energy cost (*total decrease of energy cost*) in relationship to the increase of walking velocity (*difference of speed*). *effect of speed* = *difference of speed* (6.387 – 5.597 *difference of speed*) – 1.053, ($r^2 = 0.25$, $P < 0.05$) *effect of technique* = *difference of speed* (0.088 *difference of speed* – 0.122) + 0.599, $r^2 = 0.25$, $P < 0.05$). *Total decrease of energy cost* = *effect of speed* + *effect of technique* (○ = measured data, $r^2 = 0.26$, $P < 0.05$)

Discussion

In CHF patients the quality of life strongly depends on the ability to perform basic activities providing for daily needs such as walking. Limiting factors for walking performance are the availability and the demand of metabolic power.

Walking performance

The metabolic power of walking depends on:

- Potential and kinetic energy changes due to the oscillations of the centre of mass in the vertical plane and to deceleration and acceleration at each stride,
- Internal work, meaning energy spent on activity not changing directly the position of the centre of mass,

- C. Muscle contractions for the maintenance of posture, and
 D. Work of respiratory muscles and heart (di Prampero 1986; Zaciorskij 1987).

Direct measurement of all the above-mentioned factors is complicated however, the total metabolic power of walking has been determined via indirect calorimetry (Bobbert 1960; di Prampero 1986; Margaria et al. 1963; van der Walt and Wyndham 1973; Zaciorskij 1987).

The metabolic power of walking has been found to be linearly related to body mass (Bobbert 1960; di Prampero 1986; van der Walt and Wyndham 1973; Zaciorskij 1987). This indicates that obesity is a relevant factor reducing mobility, and argues in favour of specifying metabolic power of walking as a function of body mass.

The metabolic power of walking has also been shown to be affected by the speed of locomotion (Bobbert 1960; Cotes and Meade 1960; Dill 1965; di Prampero 1986; Ralston 1958). The present results have demonstrated (Fig. 1), underlined by numerous empirical analyses of walking, and in agreement with common ideas about the energetics of walking, that the metabolic power of walking can be reasonably described as a quadratic function of walking speed (Bobbert 1960; Cotes and Meade 1960; Ralston 1958; van der Walt and Wyndham 1973; Zaciorskij 1987; Zarrugh et al. 1974). Pre and post-training, the present results (Table 1) agree with generally accepted data about the metabolic power of walking of approximately $2.3\text{--}5.9 \text{ J} \cdot \text{kg}^{-1} \cdot \text{m}^{-1}$ at walking speeds between 0.4 and $1.9 \text{ m} \cdot \text{s}^{-1}$ (di Prampero 1986; Zaciorskij 1987; Zamparo et al. 1992, 1995). Nevertheless, Fig. 1 shows a power speed curve shifted to the right after the exercise training period. Consequently, the increase in walking performance partly represented a significant effect on metabolic capacity accounting for higher metabolic walking power and partly on an optimized walking economy.

Walking economy

The energy cost per distance walked has been generally accepted as a measure of walking economy (Brueckner et al. 1991; di Prampero 1986; Margaria et al. 1963). The energy cost of walking has been shown to depend on walking speed and walking technique (Olgati et al. 1986; Zamparo et al. 1995). Inadequate walking techniques impose a greater disturbing variability of the motor pattern which increases the metabolic power spent on the above-mentioned factors.

In healthy sedentary people the energy cost of natural walking has been found to be characterized by small interindividual variations; however, when the variability is expressed in terms of relative values of overall energy cost, it has been found to be higher than in other types of locomotion (di Prampero 1986). The latter has been attributed to the low ratio between speed of locomotion and body mass compared to other locomotion techniques like running and cycling, etc. (di Prampero 1986).

After the training period the variability in the energy cost of walking was reduced from approximately 20% to 17%, to a level that has been published for non handicapped subjects at a comparable walking speed (di Prampero 1986). This reduced variability in energy cost may partly be an effect of a more stable walking technique, as well as a reduced variability in self-paced speed.

The relationship between walking speed and energy cost of walking pre and post-training is shown in Fig. 2. In agreement with relevant literature (Bobbert 1960; Cotes and Meade 1960; Dill 1965; di Prampero 1986; Ralston 1958), the energy cost of walking was near an optimum between 1.0 and $1.4 \text{ m} \cdot \text{s}^{-1}$.

Obviously, pre and post-training walking economy was significantly different (Table 1, Fig. 2) ($P < 0.001$). In 12 cases, the individual self-paced velocity was below the optimal speed before training, but all the subjects chose a walking speed within the optimal range after the exercise training (Fig. 2). Consequently, the observed 27% reduction in the energy cost of walking would seem to have been a result of improved walking techniques and self paced walking speeds. Comparing the energy cost of walking between 0.7 and $1.5 \text{ m} \cdot \text{s}^{-1}$, the speeds recorded pre and post-exercise training (Fig. 2), the change in walking technique corresponded to an 18% reduction in the energy cost of walking.

The relationship between energy cost and speed (Fig. 2) and individual differences between walking speed pre and post-training enable further analyses. Individual benefits of walking technique on walking economy, the reductions of energy cost due to changes in walking technique and the corresponding values due to increases in walking speed were calculated. The differences between individual effects of technique and corresponding effects of speed appeared to be a function of the increases in walking velocity itself (Fig. 3). The effect due to an increase of walking velocity was maximal at a speed difference of approximately $0.5 \text{ m} \cdot \text{s}^{-1}$. Compared to the benefit of walking velocity, walking technique had higher effects on walking economy at increases of speed below approximately $0.35 \text{ m} \cdot \text{s}^{-1}$ and above approximately $0.8 \text{ m} \cdot \text{s}^{-1}$.

The observed level of technique-related benefits in walking economy was lower than the difference between the energy costs of patients with multiple sclerosis and controls determined at given walking speeds (Olgati et al. 1986). In contrast to the latter, the corresponding differences between patients with hemiplegia and controls were smaller (Zamparo et al. 1995). The small difference between hemiplegics and controls has been explained by an effective compensation between the healthy and the sick limb which enables the hemiplegics to minimize the external energy of level walking by adequate transformation of potential energy into kinetic energy at each step (Zamparo et al. 1995). This type of compensation seemed to be impossible in patients with multiple sclerosis. Multiple sclerosis affects both limbs and has been reported to increase the energy cost of

walking three to four times (Oligati et al. 1986). The CHF is a complex syndrome affecting both the cardiovascular system and skeletal muscle. It has been found that deconditioning of skeletal muscle leads to muscle atrophy, changes in the relationships between type I and type II-B fibres and a reduction of enzymatic capacity (Sullivan et al. 1990) which may impair muscle co-ordination (Meyer et al. in press). Combined with a decondition-related deficit of experience of motion, the latter may induce suboptimal motor patterns. This may explain the observed level of the beneficial effect of technique on the energy cost of walking in CHF patients after the training period.

Consequences for exercise testing and rehabilitation programme

In CHF patients adequate rehabilitation programmes have been shown to enhance exercise capacity, primarily by favourable peripheral changes especially in skeletal muscle (Hambrecht et al. 1995; Meyer et al. in press; Sullivan et al. 1990). The present results demonstrated that in CHF patients with severe exercise intolerance, walking capacity can be dramatically increased by a 3-week rehabilitation programme. It has been reported that approximately 89% higher self-paced walking speed offers a much augmented potential to withstand the physical stresses of normal life and an enormous increase in the quality of life (Guralnik et al. 1995).

Motivation during exercise does not seem to have been a relevant and influencing factor of self-paced walking in the present study. A greater effort is usually reflected by a higher level of BLC, but there were no differences in walking BLC pre and post exercise training. Nevertheless, it remains unclear why the level of BLC was unchanged, despite a higher self-paced walking speed. Lack of changes in BLC might have resulted from a greater concentration of oxidative enzymes and/or number of mitochondria, improved oxygen delivery and/or lower stimulation of muscle glycogenolysis and/or reduced lactate release from skeletal muscle by reduced catecholamine concentrations and/or greater peripheral vasodilatation.

At pretraining walking economy, the observed 34% higher metabolic walking power would have accounted for approximately 42% of the observed increase in walking speed. Thus, after 3 weeks of exercise training, in CHF patients the benefit for self-paced walking which could be attributed to the increase in metabolic power was smaller, compared to the improvement in walking economy. Approximately two-thirds of the improvement in walking economy was a result of a better walking technique. The remaining percentage resulted from an economizing effect of a more adequate maximal self-paced walking speed.

It can be concluded that in deconditioned subjects like CHF patients, standard exercise tests for determination of central haemodynamics and metabolic capacity should be combined with tests of the performance of

basic activities providing for daily needs. Performance of basic activities providing for daily needs does not necessarily represent metabolic capacity. It gives additional information about the economy of motion. Economy of motion may be the dominant factor for improved performance capacity after exercise training programmes. This seems to be especially relevant in patients with severe exercise intolerance. Adequate exercise training programme and their evaluation should contribute to both favourable metabolic changes and positive effects on the economy of motion.

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