Comparison of effects of supervised *versus* self-monitored training programmes in patients with chronic obstructive pulmonary disease

L. Puente-Maestu*, M.L. Sánz**, P. Sánz**, J.M. Cubillo*, J. Mayol**, R. Casaburi***

Comparison of effects of supervised versus self-monitored training programs in patients with chronic obstructive pulmonary disease. L. Puente-Maestu, M.L. Sánz, P. Sánz, J.M. Cubillo, J. Mayol, R. Casaburi. ©ERS Journals Ltd 2000.

ABSTRACT: The effects of two 8 week programmes of reconditioning in chronic obstructive pulmonary disease (COPD) patients were studied.

Forty one subjects (mean±sd) 644.5) yrs; forced expiratory volume in one second (FEV1) 1.09±0.16 L; 40.6±6.2% predicted were randomly assigned either to supervised training on a treadmill, 4 days·week $^{-1}$ (group S; n=21) or walking 3 or 4 km in 1 h 4 days·week $^{-1}$, selfmonitored with a pedometer, with weekly visits to encourage adherence (group SM; n=20). Patients were evaluated with the chronic respiratory diseases questionnaire (CRQ) and two exercise tests on a treadmill: incremental (IT) and constant (CT), above lactic threshold or 70% of maximal oxygen uptake ($V^{\prime}{\rm O}_{2}$, $_{\rm max}$) with arterial blood lactate determinations.

Estimated mean work rate of training was 69±27 W and 25±5 W respectively for groups S and SM. Both types of training produced similar changes in the four dimensions of the CRQ. In group S reconditioning yielded significant (p<0.05) increases in $V_{\rm O_2,\,max}$ and increases in duration, with decreased lactate accumulation, venti-lation, CO₂ output ($V_{\rm CO_2}$), heart rate (HR) and diastolic blood pressure (DBP) at the end of CT. They also adopted a deeper slower pattern of breathing during exercise. The SM group showed significant (p<0.05) increases in duration, lower HR and DBP at the end of CT. Significantly (p<0.05) different effects between S and SM pro-grammes were changes in $V_{\rm O_2,\,max}100\pm101$ mL·min⁻¹ versus 5 ± 101 mL·min⁻¹), duration of the CT (8.1 ± 4.4 min versus 3.9 ± 4.7 min), $V_{\rm CO_2}$ (-94 ± 153 mL·min⁻¹ versus 48 ± 252 mL·min⁻¹), lactate accumulation (-1.3 ± 2.2 mmol·L⁻¹ versus 0 ± 1.2 mmol·L⁻¹ and respiratory rate at the end of CT (-4.3 ± 3.4 min⁻¹ versus -1 ± 4.2 min⁻¹).

Supervised, intense training yields physiological improvements in severe chronic obstructive pulmonary disease patients not induced by self-monitored training. The self-monitored, less intense training, increases submaximal exercise endurance, although to a lesser degree.

Eur Respir J 2000; 15: 517-526.

*Hospital General Universitario Gregorio Marañón, Servicio de Neumologá, Madrid, Spain. **Hospital Virgen de la Torre, Servicio de Neumologá, Madrid, Spain. ***University of California Los Angeles, Harbor-UCLA Medical Center, Division of Respiratory and Critical Care Physiology and Medicine, Torrance, CA, USA.

Correspondence: L. Puente-Maestu, Hospital General Universitario Gregorio Marañón, Servicio de Neumologá, c/ Doctor Ezquerdo 47, 28007 Madrid, Spain. Fax: 34 15868018

Keywords: Chronic obstructive pulmonary disease oxygen uptake rehabilitation submaximal exercise testing

Received: December 29 1998 Accepted after revision November 23 1999

This study was financed by the Fondo de Investigaciones Sanitarias de la Seguridad Social grant 94/0122.

Pulmonary rehabilitation programmes for patients with chronic obstructive pulmonary disease (COPD) are well established as means of alleviating symptoms and improving function in conjunction with other forms of therapy [1, 2]. Exercise training is a key component of rehabilitation programmes. The primary goal of such programmes is to increase functional capacity to the possible maximum or at least to a level that permits comfortable daily living activities. There is some controversy regarding the mechanisms responsible for the increase in functional capacity when large muscle groups are trained. Two distinct mechanisms have been proposed: firstly it would be possible to obtain physiological benefits arising from improved cardiorespiratory and muscle fitness. This, probably, is only achievable if the patient is trained above some intensity threshold [3–6]. The second mechanism consists of improving mobility without apparent changes in cardiorespiratory function associated with an increased tolerance to dyspnoea [7–11].

As a cost-cutting measure, pulmonary rehabilitation could be conducted with only modest supervision. In par-

ticular, it has been asserted that exercise programmes can be conducted in a self-monitored mode [7, 12]. The authors wished to determine whether improvements in function obtained from self-monitored exercise programmes consisted of only psychological benefits or of physiological benefits as well.

For this reason, the aim of this study was to compare two exercise training programmes, one fully supervised at the hospital, and the other self-monitored by the patient, in which participation of hospital staff was minimal. Outcome variables examined included both quality of life measures and physiological measures.

Material and methods

Study design, settings and experimental population

The study was designed as a randomized, controlled, parallel two group study. Once it was verified that the patients met the selection criteria and signed the informed consent, they were randomly assigned to one of the two training methods described below. The randomization method used involved blocks of four patients and was established before the first patient was included. The physicians who sent the patients for rehabilitation were unaware of the randomization sequence.

Patients were referred to the authors' rehabilitation programme by their pneumologists, who decided on other therapies during the study. Selection criteria were: 1) age <75 yrs; 2) severe COPD (history of having smoked at least 10 packs-year, post bronchodilator forced expiratory volume in one second (FEV1) <50% of predicted and FEV₁/forced vital capacity (FVC) <0.7) without significant reversibility (<15% of the initial value, 15 min after the inhalation of 200 µg of salbutamol); 3) declared smoking cessation at least 6 month before enrolling and arterial blood carboxyhaemoglobin <3%; 4) stable phase of their COPD, meaning no exacerbation, for at least 2 months, of acute dyspnoea needing medical assistance, changes in volume or characteristics of sputum, increase in lung sounds (wheezing or ronchi) or increases in the needs of lung medication; 5) grade 2 or more of dyspnoea sensation measured by the a modification of the Medical Research Council scale [13] that scores dyspnoea form 0 (none) to 4 (resting dyspnoea) in ascending categories related to walking function; 6) no evidence of asthma, bronchiectasis, obliterating bronchiolitis, scarring affecting >20% of one hemithorax in the chest radiography, thoracic deformities, fibrothorax, severe cardiomyopathies, ischaemic cardiopathy, severe arrhythmia, type I diabetes mellitus, neuromuscular disorders, severe hepatic or renal diseases; and 7) no physical or psychological impairment impeding exercise testing or training.

Patients were excluded if they showed poor collaboration or skipped >1 week of training and did not want to restart the whole training programme.

All patients signed an informed consent and the protocol was approved by the authors' institution's Committee for Ethics in Human Research. The authors' centre is a tertiary, university hospital.

Interventions

Patients were randomly assigned to one of two 8 week training programmes. The first group was supplied with a pedometer and asked to walk 3 or 4 km in 1 h on level ground, 4 days week⁻¹. They were asked to walk at a pace brisk enough to elicit moderate dyspnoea at least 10 min into the hour of exercise. The length of the step to adjust the pedometer was measured by counting the number of steps needed to cover 10 m on level ground. It was readjusted after the first week of training. Subjects kept a daily record of the number of steps and walked distance shown by the pedometer. Patients came once a week to the clinic to have their records checked and to be encouraged to continue with the training. This group was denominated the self-monitored (SM) group.

The second group was trained on a treadmill, supervised by a physiotherapist, starting at 3 km·h⁻¹ and a slope equal to those giving rise to an oxygen uptake equivalent to 25% of the difference between maximal oxygen uptake (VO₂,max) and the oxygen uptake (VO₂) at which the lactic acidosis threshold was detected (VO₂,LAT) plus VO₂,LAT (Δ)). In the case that VO₂,LAT was not detected,

patients started exercising at the slope and speed at which 70% of $V'_{\rm O_2,max}$ had been reached in the incremental test. From that starting point the authors attempted to increase treadmill distance 2% every week according to tolerance. Patients trained for 60 min·day⁻¹, 4 day·week⁻¹. Session could be split into as many as three periods separated by 10 min rest when needed, but a total of 60 min of exercise was still completed. These patients constitute the supervised (S) group.

Outcome measures

Spirometry and plethysmography were performed in the seated position according to international guidelines [14] with the Masterlab (Jaeger, Hochberg, Germany). The mean dissipation time and the pressure gauges of the cabin and mouthpiece were calibrated every morning. The pneumotachograph was calibrated each day with a 2 L syringe. The values considered in the study are those obtained 15 min after the inhalation of two puffs (200 µg) of salbutamol [15]. In the authors' laboratory the bronchodilator test is standardized and patients are given verbal and written instructions to refrain from using shortacting β -adrenergic drugs and ipratropium bromide for 12 h, and long-acting β -adrenergic drugs and theophyllines for 24 h. Pulmonary transfer factor of carbon monoxide (TL,CO) was measured in the sitting position by the standardized single breath method with a Transferscreen II (Jaeger). Inspiratory and expiratory maximal pressure (MIP and MEP) were measured at residual volume (RV) and total lung capacity (TLC), respectively in the seated position as described by BLACK and HAYATT [16] using a 200 cmH₂O calibrated aneroid manometer (Sibelmed, Barcelona, Spain) and a Vacumed 1002 mouthpiece (Ventura, CA, USA). All measured pressures were maintained for at least 1 s. Maximal pressure manoeuvres were repeated six times separated 1 min. The best inspiratory and expiratory measurements were used.

Quality of life was measured in all patients by the same nurse, using the version of the Chronic Respiratory Questionnaire (CRQ) [17] translated into and validated in Spanish [18]. The Questionnaire was administered just before the exercise tests, not >2 weeks before the training programme and within the first week afterward.

Accuracy and reproducibility of the pedometers were tested in the authors' laboratory, with the sensitivity of the movement detector set at minimum. The patients in the S group carried one of the six different pedometers during the treadmill constant work-rate test; distance measured by the pedometer and the treadmill differed by <10%.

Exercise tests were performed on a treadmill (Laufergometer Junior; Jaeger). Patients were asked to have a light meal the morning of the test and to abstain from caffeinated beverages. The patient wore a facemask with a dead space of 100 mL. The room was conditioned to 25°C. Gas exchange measurements were made using a breath by breath exercise system (Oxycon α, Jaeger). Before each test, the gas analysers of the system were calibrated with a three-point calibration for O₂ and two points for CO₂. The Oxycon a system (Jaeger) measures flow with a turbine that was calibrated with a 2 L syringe at 14, 21 and 28 strokes min⁻¹. The incremental exercise test was performed following a protocol modified from BALKE [19] until symptoms limitation. Before the test, all the subjects performed

a trial to get acquainted with the treadmill. The authors considered as $V'O_2$,max the value measured in the final 10 s of the highest grade the patient could sustain for a full minute. Gas exchange lactic acidosis threshold (LAT) was determined by the V-Slope method [20]. Its measurement was considered valid when it was within $\pm 100~\text{mL} \cdot \text{min}^{-1}$ of the determination made by the ventila-tory equivalents [21] and the test lasted >3 min. Dyspnoea and leg discomfort were measured at the end of the exercise by a Borg scale. Before the exercise test the scale was explained to the patient. A radial artery was anaerobically punctured with a 25 F needle at the end of the test for blood gas analysis. It was immediately tested for blood gases and pH with a blood gas electrode analyser (IL 1306; Instrumentation Laboratory, Lexington, MA, USA).

On a different day than the incremental test, a constant work-rate test at 3 km·h⁻¹ was performed at a slope corresponding to 25% of Δ of the pretraining incremental test. If the authors could not detect the LAT, the treadmill was set with the parameters at which 70% of V'O₂,max had been reached in the pretraining incremental test. The patient started with the slope preset and at 1.5 km·h⁻¹ during 1 min and 3 km·h⁻¹ afterward. A radial artery was cannulated before the constant work rate test. Blood was anaerobically drawn from the cannulated radial artery before and 2 min after the exercise test and was immediately tested for blood gases and pH with a blood gas electrode analyser (IL 1306; Instrumentation Laboratory), for haemoglobin and carboxyhaemoglobin with a co-oximeter (IL-486; Instrumentation Laboratory). Blood for lactate was immediately placed in a tube with ethylene diamine tetra-acetic acid (EDTA), kept in iced water, and then carried to the laboratory within minutes. Lactate was measured by an enzymatic method.

Dyspnoea and leg discomfort were measured at the end of the exercise by a Borg [22] scale. The test finished when the patient was not able to continue despite encouragement or after 25 min.

Training work rate was estimated from the formulas developed by GIVONI and GOLDMAN [23]:

$$\begin{split} \textit{V'}o_2 &= 294^{-1} \!\cdot\! \eta \!\cdot\! W[2.3 + 0.32(V - 2.5)^{1.65} \\ &\quad + G \!\cdot\! (0.2 + 0.07(V - 2.5))] \end{split}$$

Where η is the terrain coefficient and it is one for treadmill and blacktop surfaces, W is body weight in kg, V is speed in km·h⁻¹ and G is incline expressed as percentage. The standard deviation of this regression equation is 0.11 L·min⁻¹. The work rate equivalent to the VO_2 so obtained was calculated by the formula developed by Jones [24]:

Work rate (watts) =
$$(V'o_2 - 0.3)/0.0123$$

Where $V'o_2$ is expressed in L min⁻¹

Statistical analysis

Comparisons of mean responses before and after training were performed by paired t-test. Comparisons of means between groups were carried out with unpaired t-test. These tests were preceded by checking for normality and equality of variance. If the variance equality assumption was violated, the proper correction was applied to the t-test. Means were declared to be significantly different when the probability of two tail type I error was <0.05.

Proportions were compared by the exact unconditional test for difference of two binomial proportions [25]. Significance of the changes for the CRQ scores within groups was sought by determining whether the proportion of patients with a change in score equal or higher than the one determined to be significant by the authors [17, 26] (i.e. 0.5 points per item or 2.5, 2, 3.5 and 2 points respectively for dyspnoea, fatigue, emotional function and mastery) was significantly greater than zero. Correlation was measured with the Pearson correlation coefficient (r). Values of mean responses are presented with their SD within parentheses and correlation with their 95% confidence interval for comparisons with r=0, unless otherwise specified. Statistics were performed with a statistical package (SPSS 7.5; Hispano-Portuguesa SPSS, SL, Madrid, Spain) in a personal computer.

Results

A total of 49 patients were initially enrolled. Of these, 41 completed the assigned programme and produced evaluable results: 21 subjects in the S group and 20 in the SM group. The eight patients who abandoned the study reported scheduling reasons, no adherence to the training programme or unexpected personal affairs. None withdrew because of medical complications. Five of them had been assigned to group SM and three to group S.

The general characteristics of the two groups before training are presented in table 1. In summary, both groups consisted of males with mean weight slightly above the ideal weight, severe airflow limitation (FEV1/vital capacity (VC) $45\pm8\%$ pred), moderate hypoxaemia (oxygen tension in arterial blood (P_{a,O_2}) 64 ± 6 mmHg) without CO_2 retention at rest and a mild decrease in $T_{b,CO}$ ($70\pm23\%$ pred).

Regarding their exercise performance, they showed a substantial decrease in aerobic capacity $[VO_2, max]$ of $61\pm12\%$ pred] [27]. At maximal exercise capacity they had, on average, significant desaturation and hypoventilation (The carbon dioxide tension in arterial blood (Pa, CO_2) at the end of incremental exercise test increased an average of 6 ± 4 mmHg over resting levels).

Training

Both groups of patients adhered to their exercise programmes reasonably well. From group S, eight (38%) missed one or two sessions, three (14%) could not attend for a full week (one because they needed to attend to personal affairs and two due to mild exacerbations of their COPD). A week was added at the end of the training programme of these three patients. One patient (5%) did not attend for 2 weeks on a row and agreed to restart the training for another 8 weeks after a delay of 4 weeks. Most of the patients in the SM group walked >1 h·day⁻¹ and did it more than the 4 days-week-1 requested (table 2). Two (10%) stopped training for a week because of mild exacerbation of their COPD. Ten patients missed 1 visit (50%) and two patients (10%) missed two of the visits to evaluate adherence, mainly because of scheduling problems or short vacations. Nonetheless, each of the patients presented on the next visit their training log for the entire period since their last visit.

Table 1. - Description of the two groups

		Supervised n=21		Self-monitored n=20	
Variable	Units	Mean	SD	Mean	SD
Age	yrs	63.3	±4.3	65.6	±4.7
Height	cm	163.1	± 4.4	164.8	± 4.6
Body mass	kg	68.9	± 7.2	68.6	± 10.2
FEV1	Ľ	1.09	± 0.15	1.09	± 0.17
FEV1 %	% pred	41	±6	40	± 6
FEV1/VC	% pred	44.5	± 7.3	46.1	±7.9
FVC	Ĺ	2.57	± 0.47	2.59	± 0.55
FVC %	% pred	76	±13	75	±11
P_{a,O_2}	mmHg	65.3	±5.5	63.4	± 7.1
Sat O ₂	% pred	92.9	± 2.1	91.9	± 2.4
Pa,CO_2	mmHg	37.9	± 2.3	37.7	± 2.6
Pa,O_2 E	mmHg	58.4	± 8.7	58.3	± 7.3
Sat O ₂ E	% pred	86.4	±5.9	85.3	± 6.2
Pa,CO₂ E	mmHg	43.4	±5.7	44.5	± 3.2
TL,CO	mmol∙min ^{-T} ·kPa ⁻¹	5.3	± 1.2	5.1	± 1.4
TL,CO	% pred	72.4	±21.7	66.9	± 23.1
KCO	mmol·min-1·kPa-1·L-1	1.14	± 0.3	1.1	± 0.4
KCO %	% pred	79.1	± 28.1	77.7	±29.5
$V_{O_2,max}$	$ ext{L-min}^{-1}$	1.24	± 0.24	1.25	± 0.29
VO ₂ ,max %	% pred	61.1	±11.5	62.1	± 12.4

FEV1: forced expiratory volume in one second; FEV1 %: forced expiratory volume in one second as a per cent of predicted [15]; VC: inspiratory vital capacity; FVC: forced vital capacity; FVC %: forced vital capacity as per cent of predicted [15]; P_{a,O_2} : arterial partial oxygen pressure breathing air; Sat O_2 : oxygen saturation of haemoglobin directly measured with a co-oximeter; P_{a,CO_2} : arterial partial carbon dioxide pressure breathing air; E: exercise; $T_{L,CO}$: pulmonary transfer factor for carbon monoxide; KCO: carbon monoxide diffusion constant; $V'_{O_2,max}$: maximal oxygen uptake; $V'_{O_2,max}$ %: maximal oxygen uptake as per cent of predicted [27].

The estimated work rate and total amount of work done along the training programme are reported in table 2. Both were significantly higher for group S (p<0.0001). Mean increase in slope of the treadmill over the course of the training period in group S was 7.7 (2.5)%.

Quality of life

The initial and post-training scores of the CRQ are summarized in table 3. There were no differences between the two groups either before or after training. As can be seen in table 3, scores in all the four dimensions of the CRQ improved significantly. There were no significant differences in the magnitude of change or the proportion of patients who improved the score of each domain by a clinically significant amount. Correlations between changes in each dimension of the CRQ score and both changes in $VO_{2,max}$ and changes in constant work rate duration were calculated for each subject group. No statistically significant correlations were observed.

Lung function and arterial blood gases

In general, there was little change in pulmonary function tests after training (table 4). A modest, but significant increase in FEV1 and MIP and a tendency for MEP to increase (table 4) was found. Those changes were apparent in both groups, without significant differences between them.

Incremental exercise test

Initially both groups were quite similar in their aerobic capacity and other peak ventilatory, cardiovascular and gas exchange measurements (table 5). The group trained under

supervision had a significant (p<0.01) increase in mean $V'_{\rm O_2,max}$ averaging $110\pm101~\rm mL\cdot min^{-1}$ or $9\pm9\%$ of the initial value. $V'_{\rm O_2,LAT}$ increased $90\pm114~\rm mL\cdot min^{-1}$ or $10\pm13\%$, (p<0.01) in the S group and a small but significant (p<0.05) increase in $V'_{\rm O_2,LAT}$ was seen $41\pm86~\rm mL\cdot min^{-1}$ in the SM group as well. $V'_{\rm O_2,LAT}$ could be detected before and after training in 15 patients of group S and 14 in group SM. In response to supervised training patients also changed their ventilatory pattern to deeper, slower breathing (table 5). Accordingly, ventilation also tended to be more efficient with a mean decrease in dead space ($V'_{\rm D}$)/tidal volume ($V'_{\rm T}$) of 0.04 ± 0.07 , p=0.083. Those effects were not seen in the SM group. Leg fatigue score at the end of the test decreased significantly after training in both groups (table 5).

Comparing the responses of the two groups, the mean increase in VO_2 ,max, either in absolute or relative to predicted values and the decrease in breathing rate were statistically greater in the S group (p<0.05).

Constant work-rate exercise test

Again, before training both groups were very similar in their physiological response to the constant work-rate test, as can be seen in table 6. Both groups improved in endurance time, although the increase was larger in group S.

After intense training, group S showed several changes in the variables obtained from the constant exercise test (table 6). First, lactate accumulation decreased (p<0.0001). Second, carbon dioxide excretion at the end of exercise decreased (p<0.01) and $V'_{\rm O_2}$ at the end of the exercise tended to be lower as well. Third, there was a change (p<0.05) in breathing, with a slower and deeper pattern, in the same way as occurred in the incremental test. Fourth,

Table 2. – Training intensity and total work performed along the 8 weeks of the programme

	Work rate W	Frequency days·week ⁻¹	Time min	Total work kJ
S group				
1st week	30 (9)	4	60	436 (133)
2nd week	50 (17)	4	60	721 (249)
3rd week	63 (24)	4	60	911 (349)
4th week	74 (29)	4	60	1069 (430)
5th week	82 (33)	4	60	1179 (489)
6th week	83 (33)	4	60	1200 (488)
7th week	85 (34)	4	60	1228 (503)
8th week	87 (36)	4	60	1254 (524)
Mean	69 (27)	4	60	8001* (3097)
SM group				
1st week	26 (6)	4.4(1)	73 (15)	460 (111)
2nd week	25 (6)	5.1(1)	86 (19)	557 (208)
3rd week	25 (6)	5.5 (1)	86 (22)	452 (99)
4th week	27 (6)	5.4(1)	79 (19)	681 (182)
5th week	25 (5)	5.3 (1)	71 (15)	446 (89)
6th week	26 (5)	5.0 (1)	81 (18)	524 (163)
7th week	26 (6)	5.3 (1)	75 (15)	471 (112)
8th week	24 (5)	5.4(1)	75 (16)	646 (269)
Mean	25 (5)	5.2 (1)	78.3 (6.5)	4237* (834)
p=	< 0.0001	< 0.0001	< 0.0001	0.0005

Data are presented as mean (SD). Work rate is the mean estimated training intensity calculated as described in "Material and methods" section. Frequency is the mean days per week trained according to physiotherapist records (S group) or patients log (SM group). Time is the average time trained per day, according to physiotherapist records (S group) or patients log (SM group). Total work is the weekly total amount of work calculated from the previous parameters. *: mean cumulative sum. S: supervised; SM: self-monitored.

ventilatory demand decreased significantly (p<0.01), with an average reduction in minute ventilation (V'E) at the end of exercise of 3.44.2 L·min⁻¹. This decrease in V'E at the end of exercise was strongly correlated with the decrease in lactate accumulation in the S group r=0.71 (0.42–1). Interestingly, though, changes in V'E were not significantly correlated either with changes in FEV1 nor with changes in FVC. Fifth, there were decreases in heart rate (HR) (p<0.01) and diastolic blood pressure (p<0.05) at the end of exercise. Finally, patients' perceptions at the end of exercise also changed with training. Dyspnoea scores decreased significantly (p<0.05) and leg fatigue showed a trend towards doing so (p=0.066).

In the SM group, the changes were much less marked. The main physiological changes were a decrease in heart rate and diastolic blood pressure at the end of exercise (p<0.05). From the perceptual point of view, leg fatigue scores decreased significantly.

Comparing the changes in response in the two groups, significant differences (p<0.05) were found between changes in lactate accumulation (p<0.01), $V'CO_2$, V'E and breathing rate at the end of exercise (p<0.05).

Discussion

The authors have compared two programmes for conditioning severe COPD patients with a different degree of supervision. Both types of training improved exercise tolerance, but the magnitude and the extent of physiological improvements were larger in patients training under supervision. Some changes, like the modifications of breathing pattern and the decrease in lactate accumulation in the constant work rate test above the lactic acidosis threshold, were unique to supervised exercise training.

Those selected for study were a population of patients with severe COPD not receiving chronic oxygen therapy who demonstrated a substantial decrease in exercise capacity. They are representative of one of the subpopulations of COPD patients in which leg muscle reconditioning is felt to be beneficial.

Two training strategies were compared, the first was a self-monitored programme that can be easily carried out by subjects with moderate to severe dysfunction and requires only a little extra work by a physician, a nurse or a physiotherapist and can be performed even if the patient seldom leaves their domicile. The exercise prescription for the self-monitored programme was designed to include frequency, duration and intensity characteristics that had the potential to induce a physiological training effect. Prescribing 1 h of exercise that covers 3–4 km and instructing the patient to exercise to as much as moderate dyspnoea allows, is thought to be, a "best effort" attempt to get COPD patients to exercise without direct monitoring. The authors feel that practitioners prescribing a self-monitored home exercise programme are unlikely to obtain benefits greater that the ones observed. However, because this study included pedometer measurements, the authors were able to determine that, in fact, the total work of the SM group was less than the supervised group. The second strategy was monitored training, which needs more health care resources and more collaboration on the part of the patient. As the self-monitoring training programme was based on

Table 3. - Chronic Respiratory Questionnaire (CRQ) scores before and after training

	Supervised			Self-monitored		
	Mean pretraining	Mean post-training	No. signif. increase	Mean pretraining	Mean post-training	No. signif. increase
Dyspnoea	16.4±3.1	20.0±4.6	13 (0.62)**	16.7±2.4	20.7±6.1	14 (0.70)**
Fatigue	16.2 ± 4.2	19.5±3.6	14 (0.67)**	18.1±3.5	20.9 ± 3.8	16 (0.80)**
Emot. Func.	31.6 ± 7.2	34.6 ± 7.2	7 (0.33)**	33.3 ± 6.0	38.0 ± 6.8	7 (0.35)**
Mastery	16.0 ± 4.9	19.0 ± 4.3	14 (0.67)**	16.3 ± 3.9	21.7 ± 5.6	17 (0.85)**
Total	80.2 ± 17.3	93.1 ± 16.2	. ,	84.2±13.9	101 ± 17.2	. ,

This table presents the scores (mean±sD) of the four different dimensions of the CRQ questionnaire and the total score before and after training. No patient had a significant decrease of score. No. Signif.: increase: the number of patients with a significant change in score, (the fraction of the total group is in parentheses); Emot. Func.: emotional function. **: p<0.01.

Table 4. - Pulmonary function test values before and after training

		Supervi	Supervised (n=21)		Self-monitored (n=20)	
	Units	Mean pretraining	Mean post-training	Mean pretraining	Mean post-training	
FEV1	L	1.09±0.15	1.16±0.17*	1.09±170	1.15±0.21*	
FEV1 %	% pred	41±6	44±7	40±6	43±8*	
FVC	Ĺ	2.57 ± 0.47	2.65 ± 0.46	2.63 ± 0.51	2.67 ± 0.58	
FVC %	% pred	76±13	79±14	76±10	77±12	
FEV ₁ /FVC	% pred	43±8	45±7*	42±10	44±8*	
TLC	Ĺ	6.44 ± 0.74	6.40 ± 0.79	6.77 ± 0.70	6.67 ± 0.85	
TLC %	% pred	112±11	111±11	110±11	109±13	
FRC	Ĺ	4.48 ± 0.74	4.51 ± 0.75	4.47 ± 0.62	4.46 ± 0.58	
FRC %	% pred	139±23	140±24	142±20	142±26	
TL,CO	mmol·min⁻¹·kPa	5.3 ± 1.21	5.5±1.4	5.1 ± 1.40	5.2 ± 1.8	
Tl,co %	% pred	72 ± 22	75±23	67±23	66 ± 25	
KCO	mmol·min-1·kPa-1·L-1	1.14 ± 0.34	1.18 ± 0.41	1.07 ± 0.36	1.00 ± 0.37	
KCO %	% pred	82±28	84±32	7624	7126	
MIP	cmH ₂ O	63±15	70±12**	58±17	68±13**	
MEP	cmH ₂ O	109 ± 26	117±24	120±26	126±24	
P_{a,O_2}	$ar{mmHg}$	65±6	67±8	63±7	65±8	
S_{a,O_2}	%	93±2.0	93±8.1	92±2.4	92 ± 2.7	
Pa,CO_2	mmHg	38±2	38±3	38±3	38±2	

Values are mean \pm so. FEV1: forced expiratory volume in one second; FVC: forced vital capacity; TLC: total lung capacity; FRC: functional residual capacity; TL,CO: pulmonary transfer factor for carbon monoxide; KCO: diffusion constant (TL,CO/alveolar volume); MIP: maximal inspiratory pressure at residual volume; MEP: maximal expiratory pressure at TLC; Pa,O2: arterial partial oxygen pressure breathing air; Sa,O2: resting oxygen saturation of haemoglobin measured with a co-oximeter; Pa,CO2: resting arterial partial carbon dioxide pressure. **: two tail p<0.01; *: p<0.05, comparing values before and after training within the same group.

walking, the authors decided to train the other group on a treadmill.

The authors anticipated a higher rate of withdrawals from the supervised group, but there were no differences in the dropout rate. The finding could be biased by the awareness of participating in a study (the Hawthorne effect) [28] or selection bias of very co-operative patients.

Other randomized controlled trials have shown that CRQ scores improve after multifactorial training programmes [9, 29]. In the current study mainly with leg exercise

training, improvements in CRQ scores for each of the four areas either as mean values or as the proportion of patients, were manifested and, importantly, the improvement was similar in both groups, even though the physiological effects of the two types of training were markedly different. Others have also noted that the changes in CRQ were not closely correlated with physiological improvement [30, 31]. Other isolated interventions without effects on leg exercise capacity, like weight lifting [32] or behavioural modification [33] also improved quality of life

Table 5. - Incremental exercise test measurements before and after training

		Supervised (n=21)		Self-monitored (n=20)	
	Units	Mean pretraining	Mean post-training	Mean pretraining	Mean post-training
V'O ₂ max	mL·min⁻¹	1243±243	1353±298**	1250±291	1255±278
$V'_{\text{O}_2\text{max}}$ %	% pred	61±11.5	66±14**	62±12	63±10
$V'_{\rm O_2}LAT^{\#}$	$mL \cdot min^{-1}$	886±158	949±172**	944±143	978±155
HR,max	min ⁻¹	136±13	136±9	133±13	132±12
V'E,max	L·min⁻¹	39±6	38±8	38±10	37±8
VE/MVV	%	82±9	79±14	82±13	80±12
Bf,max	min ⁻¹	34±6	31±5**	31±7	31±7
$V_{T,\max}$	mL	1142 ± 180	1239±237*	1223±287	1234±247
$P_{a,O_2}E$	mmHg	58±9	57±8	58±7	56±10
$S_{a,O_2}E$	%	86±6	86±5	85±6	85±6
Pa,CO ₂ E	mmHg	44±6	46±5	45±3	45±5
$V_{\rm D}/V_{\rm T}$	υ	0.37 ± 0.08	0.33 ± 0.06	0.37 ± 0.05	0.35 ± 0.05
Dyspnoea E	Borg scale	6.2 ± 1.4	6.4 ± 2.1	6.5 ± 1.5	7.1±1.6
Leg fatigue E	Borg scale	3.5 ± 1.9	1.8±1.6**	2.7 ± 1.9	1.8±1.6*

Values are mean \pm sp. $V'O_2$ max: maximal oxygen uptake; $V'O_2$ LAT: the oxygen uptake at which the lactic acidosis threshold was detected; HRmax: heart rate at the $V'O_2$ max as percent of predicted (calculated as 220-age); V'E,max: maximal minute ventilation; MVV: maximal voluntary ventilation; B'f,max: breath frequency at the V'E,max; V'T,max= tidal volume at the V'E,max; P_a ,O₂: arterial partial oxygen pressure; E: values at the end of the exercise test; S_a ,O₂: oxygen saturation of haemoglobin directly measured with a co-oximeter; P_a ,CO₂: arterial carbon dioxide pressure; VD: dead space; VT: tidal volume. **: two tail p<0.01; *p<0.05; *: based in 15 patients in the supervised group and 14 in the self-monitored group.

Table 6. – Constant exercise test measurements before and after training

		Supervis	Supervised (n=21)		Self-monitored (n=20)	
	Units	Pretraining	Post-training	Pretraining	Post-training	
Time	min	9.4±4.7	17.4±4.3**	8.5±3.9	12.4±5.1**	
V'_{0} 2E	$\mathrm{mL}{\cdot}\mathrm{min}^{-1}$	1252±274	1177±285	1154±300	1184±318	
V'CO ₂ E	$\mathrm{mL}{\cdot}\mathrm{min}^{-1}$	1121±323	1028±277*	1015±321	1063±320	
HR Ē	min ⁻¹	132±12	125±13*	130±13	128±9*	
HRE%	% pred	84±7	80±8**	83±9	82±8	
V'E E	$L \cdot min^{-1}$	39±7	34±7**	39 ± 6.4	38±7	
V'E E/MVV	%	82±14	72±11**	84±12	82±10	
$\mathbf{B}f\mathbf{E}$	min ⁻¹	32±4	27±4**	31±5	30±4	
<i>V</i> т Е	L	1.21 ± 0.21	128±0.19*	1.29 ± 0.25	1.31 ± 0.33	
SBP E	mmHg	179±29	174±23	172±15	165±17	
DBP E	mmHg	95±11	91±8*	92±8	89±9*	
ΔLa	$\text{mmol}\cdot \text{L}^{-1}$	3.2 ± 1.8	$1.6\pm0.8**$	2.7 ± 1.8	2.6 ± 1.0	
Dyspnoea E	Borg scale	6.8 ± 1.7	5.5±2.3*	6.5 ± 1.7	6.5 ± 2.7	
Leg fatigue E	Borg scale	3.3 ± 2.1	2.2 ± 2.0	2.8 ± 2.1	2.1±1.9	

Values are mean \pm sD. VO₂: oxygen uptake; E: values at the end of the exercise test; VCO₂: carbon dioxide output; HR: heart rate; V'E: minute ventilation; MVV: maximal voluntary ventilation; Bf: breath frequency; VT: tidal volume; SBP: systolic blood pressure; DBP: diastolic blood pressure; Δ La: difference between measurements of arterial blood lactate made 2 min after exercise and resting before exercise. **: two tail p<0.01; *: p<0.05.

as measured by specific questionnaires. An explanation for these results could be an effect on CRQ scores by factors other than physiological benefit such as, for example, the perception as a positive value of a more personal medical assistance, the expectation of a new treatment, a positive effect of having "something to do" for a few hours every day, or the awareness of participating in a study. In any case, quality of life and exercise capacity measurements are independent outcomes of rehabilitation programmes and therefore both should be included in the assessment of such interventions.

The authors saw a small but significant improvement in FEV1 and maximal mouth pressures in both groups with no changes in resting blood gases. This finding has occasionally been described previously [34, 35]. It is believed that it could be a consequence of a better use of bronchodilator treatment because of more information and perhaps a better adherence to medications motivated by psychological reasons related to being included in a research programme. A training effect could also play a role in the case of maximal pressures.

Patients presented a response to incremental exercise consistent with severe COPD [36]: low ventilatory reserve, high V'D/V'T, relatively high HR for the $V'O_2$ max, but low in comparison with normal, desaturation and CO_2 retention. The increase in carbon dioxide tension in arterial blood (Pa, CO_2) and the low ventilatory reserve points towards ventilation as the factor limiting exercise in these patients.

In the S group patients there was an improvement in exercise tolerance in the incremental test. In the constant work rate tests, for most of the patients the work rate was above the VO_2LAT and so, at end-exercise, they reached approximately the same VO_2max , ventilation and Pa,CO_2 values as in the incremental work load test. After training there was a striking decrease in ventilatory needs in relation to a marked decrease in lactate accumulation coupled with a deeper, slower more efficient pattern in the S group. Change in the breathing pattern has been shown after training [35, 37]. A more efficient ventilation has also been described [38, 39]. To the authors' knowledge,

though, it has not been previously described as a relationship dependent on the intensity of training.

All the changes described above were associated with a substantial increase in exercise tolerance among the patients of the S programme. However, the SM group, trained at lower intensity, also showed an increase in endurance because of motivation or other reasons. Several studies have shown increased exercise tolerance without apparent physiological changes [1, 8, 10, 39, 40]. The authors' SM programme, although it could not induce the array of physiological changes apparent after the supervised high intensity training, elicited a decrease in HR and diastolic blood pressure at the end of the constant work load exercise. In the S group, decrease in diastolic blood pressure could be related to less acidosis and lower release of catecholamines [21], however the same effect, albeit smaller, was seen in the SM group in spite of the same lactate accumulation and presumably the same acidaemia as in the pretraining exercise. Thus, the effect could have other causes such as a beneficial effect of increased daily exercising, more adherence to diet or treatment in the hypertensive patients by the same reasons given before for the increase in FEV1.

Several supervised rehabilitation programmes employing either outpatient or hospital based rehabilitation have been published [1, 6, 8–10, 29, 31, 34, 35, 38, 41–43]. These studies varied considerably in the parameters of exercise prescription, but most of them ranged 6–12 weeks in duration. In those programmes in which supervised exercise sessions lasted 20 min, with sessions more than twice per week and $VO_{2\text{max}}$ was measured as an outcome variable, a consistent improvement in both $VO_{2\text{max}}$ and endurance of a submaximal work rate after training was reported [6, 35, 38, 41, 42]. In the supervised training programme, patients were trained for 1 h, 4 days·week-1 during 8 weeks and improvement in both these outcome variables was seen.

Several home-care or community based programmes have also been published [7, 12, 29, 39, 40, 44, 45]. However it is useful to distinguish between home programmes

in which frequent visits by rehabilitation specialists were made to supervise the programme and those in which no such home supervision was offered. In the former category, Dutch investigators [29, 45] prescribed leg exercise 24 min·day⁻¹. Training exercises were taught by a physiotherapist and practised during twice weekly visits to a local physiotherapist during the first 12 weeks. Improvement in aerobic capacity, walking distance and dyspnoea were obtained. Though achieving evidence of physiological benefit, these home based programmes are resource intensive and cannot be considered unsupervised. In contrast home-based programmes with little or no supervision [7, 12, 39, 40] have generally yielded no evidence of physiological improvement in exercise tolerance (e.g. improved V'O₂max), though improvement in more effort dependent measures (e.g. 6 min walking distance) is sometimes seen.

In summary, exercise training can induce several physiological adaptations resulting in an increased aerobic capacity and exercise tolerance. These changes apparently depend on reaching a certain exercise intensity. Thus to elicit them, a rigorous programme of exercise training with supervision is needed. On the other hand, unsupervised regular walking at a brisk pace has beneficial effects on exercise tolerance and health perception but does not elicit the physiological changes induced by supervised training. Determining whether these physiological benefits justify the increased costs of the supervised programme will require a cost/benefit analysis.

References

- 1. Casaburi R. Exercise training in the chronic obstructive lung disease. *In*: Casaburi R, Petty TL, eds. Principles and practice of pulmonary rehabilitation. Philadelphia, USA, WB Saunders, 1993; pp. 204–225.
- Celli BR, Snider GL, Heffner J, et al. Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease. Committee of the Scientific Assembly of Clinical Problems. Am J Respir Crit Care Med 1995; 152 (Suppl.): 1–46.
- Davis JA, Frank MH, Whipp BJ, Wasserman K. Anae-robic threshold alterations caused by endurance training in middle-aged men. *J Appl Physiol* 1979; 46: 1039–1046.
- Yerg JE. Seals DR, Hagberg JM, Hollozsy JO. Effect of endurance exercise training on ventilatory function in older individuals. *J Appl Physiol* 1985; 58: 791–794.
- Casaburi R, Patessio A, Ioli F, Zanaboni S, Donner CF, Wasserman K. Reductions in exercise lactic acidosis and ventilation as results of exercise training in patients with obstructive lung disease. *Am Rev Respir Dis* 1991; 143: 9–18
- Punzal PA, Ries A, Kaplan RM, Prewitt LM. Maximum intensity exercise training in patients with chronic obstructive pulmonary disease. *Chest* 1991; 100: 618–623.
- McGavin CR, Gupta SP, Lloyd EL, McHardy GJR. Physical rehabilitation for the chronic bronchitic: results of a controlled trial of exercise at home. *Thorax* 1977; 32: 307–311.
- 8. Chester EH, Belman MJ, Bahler RC, Baum GL, Schey G, Buch P. Multidisciplinary treatment of chronic pulmonary insufficiency. *Chest* 1977; 72: 695–702.
- 9. Goldstein RS, Gort EH, Stubbing D, Avendano MA, Guyatt

- GH. Randomised controlled trial of respiratory rehabilitation. *Lancet* 1994; 344: 1394–1397.
- Sinclair DJM, Ingram CG. Controlled trial of supervised exercise training in chronic bronchitis. *BMJ* 1980; 280: 519–521.
- 11. Belman MJ, Wasserman K. Exercise training and testing in patients with chronic obstructive pulmonary disease. *Basics Respir Dis* 1981; 10: 1–6.
- Busch AJ, McClements JD. Effects of a supervised home exercise program on patients with severe chronic obstructive pulmonary disease. *Phys Ther* 1988; 68: 469–474.
- Medical Research Council. Committee on research into chronic bronchitis: instruction for use on the questionnaire on respiratory symptoms. Devon, UK, W.J. Holman, 1966.
- Quanjer PhD. Standardised lung function testing. Report Working Party "Standardisation of lung function tests". Bull Eur Physiolpatol Respir 1983; 19 (Suppl. 5): 22–27.
- Roca J, Sanchis J, Agusti-Vidal A, et al. Spirometric reference values for a Mediterranean population. Bull Eur Physiopatol Respir 1982; 18: 101–102.
- Black LF, Hyatt RE. Maximal respiratory pressure: normal values and relationships to age and sex. Am Rev Respir Dis 1969; 99: 696–702.
- Guyatt GH, Berman LB, Townsend M, Pugsley SO, Chambers LW. A measure of quality of life for clinical trials in chronic lung disease. *Thorax* 1987; 42: 773–778.
- Güell R, Casan P, Sangenis M, et al. Traducción española y validación del cuestionario de calidad de vida en pacientes con enfermedad obstructiva crónica (EPOC). Arch de Bronconeumol 1995; 31: 202–210.
- Balke B, Ware R. An experimental study of physical fitness of Air Force personnel. US Armed Forces Med J 1959; 10: 675–688.
- Beaver WL, Wasserman K, Whipp BJ. A new method for detecting the anaerobic threshold by gas exchange. *J Appl Physiol* 1986; 60: 2020–2027.
- Wasserman K, Hansen JE, Sue DY, Whipp BJ, Cassaburi R. Principles of exercise testing and interpretation. 2nd Edn. Philadelphia, USA, Lea and Febiger, 1994.
- Borg GAV. Psychophysical bases of perceived exertion. *Med Sci Sports Exerc* 1982; 14: 377–381.
- Givoni B, Goldman RF. Predicting metabolic energy cost. *J Appl Physiol* 1972; 30: 429–433.
- Jones NL. Clinical exercise Testing. 4th Edition. Philadelphia, USA, WB Saunders Company, 1997.
- Barnard BA. Significance test for 2 by 2 tables. Biometrika 1947; 34: 123–128.
- Guyatt GH, Walter S, Norman G. Measuring the change over time: assessing the usefulness of evaluative instruments. *J Chron Dis* 1987; 40: 171–178.
- Hansen JE, Sue DY, Wasserman K. Predicted values for clinical exercise testing. Am Rev Respir Dis 1984; 129 (Suppl.): S49–S55.
- 28. Ulmer FC. The Hawthorne effect. *Educ Dir Dent Aux* 1976; 1: 28.
- Wijkstra PJ, van der Mark TW, Kraan J, van Altena R, Koëter GH, Postma DS. Effects of home rehabilitation on physical performance in chronic obstructive pulmonary disease. *Eur Respir J* 1996; 9: 104–110.
- Wijkstra PJ, van Altena R, Kraan J, Otten V, Postma DS, Koëter GH. Quality of life in patients with chronic obstructive pulmonary disease improves after rehabilitation at home. *Eur Respir J* 1994; 7: 269–273.
- Bendstrup KE, Ingemann Jensen J, Holm S, Bengtsson B.
 Outpatient rehabilitation improves activities of daily living, quality of life and exercise tolerance in chronic

- obstructive pulmonary disease. Eur Respir J 1997; 10: 2801–2806.
- Simpson K, Killian K, McCartney N, Stubbing DG, Jones NL. Randomised controlled trial of weightlifting exercise in patients with chronic airfiow limitation. *Thorax* 1992; 47: 70–75.
- Atkins CJ, Kaplan RM, Timms EM, Reinsch S, Lofback K. Behavioural exercise programs in the management of chronic obstructive pulmonary disease. *J Consult Clin Psychol* 1984; 52: 591–603.
- 34. Mungall IPF, Hainswoth RA. Objective assessment of the value of exercise training to patients with chronic obstructive airways disease. *Q J Med* 1980; 193: 77–85.
- Casaburi R, Porszasz J, Burns MR, Carithers ER, Chang RSY, Cooper C. Physiologic benefits of exercise training in rehabilitation of patients with severe chronic obstructive pulmonary disease. *Am J Respir Crit Care Med* 1997; 155: 1541–1551.
- Gallagher CG. Exercise limitation and clinical exercise testing in chronic obstructive pulmonary disease. *Clin Chest Med* 1994; 15: 305–326.
- Woolf CR, Suero JT. Alterations in lung mechanics and gas exchange following training in chronic obstructive lung disease. *Dis Chest* 1969; 55: 37–44.
- 38. Degre S, Sergysels R, Messin R, *et al.* Hemodynamic responses to physical training in patients with chronic lung disease. *Am Rev Respir Dis* 1974; 110: 395–402.
- 39. Clark CJ, Cochrane L, Mackay E. Low intensity peripher-

- al muscle conditioning improves exercise tolerance and breathlessness in COPD. *Eur Respir J* 1996; 9: 2590–2596
- 40. Cambach W, Chadwick-Straver RVM, Wagenaar RC, van Keimpema ARJ, Kemper HCG. The effects of a community-based pulmonary rehabilitation programme on exercise tolerance and quality of life: a randomised controlled trial. *Eur Respir J* 1997; 10: 104–113.
- Ries AL, Kaplan RM, Limberg TM, Prewitt LM. Effects of pulmonary rehabilitation on physiologic and physicosocial outcomes in patients with chronic obstructive pulmonary disease. *Ann Intern Med* 1995; 122: 823–832.
- Vallet G, Ahmanidi S, Serres I, et al. Comparison of two training programs in chronic airway limitation patient: standardised *versus* individualized protocols. Eur Respir J 1997; 10: 114–122.
- O'Donell DE, McGuire M, Samis L, Webb K. General exercise training improves ventilatory and peripheral muscle strength and endurance in chronic airflow limitation. *Am J Respir Crit Care Med* 1998; 157: 1489–1497.
- Bass H, Withcomb JF, Forman R. Exercise training: therapy for patients with chronic obstructive pulmonary disease. *Chest* 1970; 57: 116–121.
- 45. Strijbos JH, Postma DS, van Altena R, Gimeno F, Koëter GH. A comparison between outpatient hospital-based pulmonary rehabilitation program and home-care pulmonary rehabilitation program in patients with COPD. A follow up of 18 months. *Chest* 1996; 109: 366–373.