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Effects of home rehabilitation on physical performance in patients with chronic obstructive pulmonary disease (COPD)

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Effects of home rehabilitation on physical performance in patients with chronic obstructive pulmonary disease (COPD). P.J. Wijkstra, Th.W. van der Mark, J. Kraan, R. van Altena, G.H. Koëter, D.S. Postma. ©ERS Journals Ltd 1996.

ABSTRACT: We investigated whether 12 weeks of rehabilitation at home in patients with chronic obstructive pulmonary disease (COPD) had a beneficial effect on lactate production, metabolic gas exchange data, workload of the inspiratory muscles, and dyspnoea during a maximal bicycle ergometer test. A second aim was to assess whether a change in dyspnoea was related to a change of inspiratory muscle workload.

Forty three COPD patients with severe airways obstruction were included in the study: mean forced expiratory volume in one second (FEV_1) 1.3±0.4 L (44% predicted), mean FEV_1/inspiratory vital capacity (IVC) 37±8%. Twenty eight patients started a rehabilitation programme, whilst 15 patients received no rehabilitation. Rehabilitation was carried out at home; patients were supervised by a general practitioner, a physiotherapist and a nurse. Exercise tolerance was measured by means of a 6 min walking distance test (6MWD) and maximal workload (W_{max}) during an incremental symptom-limited cycle ergometer test. Inspiratory muscle workload at W_{max} was assessed with the Tension Time Index (TTI), and dyspnoea at W_{max} with the Borg scale.

After 12 weeks, the rehabilitation group showed a significantly larger increase in 6MWD (from 438 to 447 m) and in W_{max} (from 70 to 78 W) compared with the control group. A significant improvement in oxygen consumption ($V'O_2$) (from 1.0 to 1.1 L), lactate level (from 3.7 to 3.1 mEq·L^{-1}), dyspnoea (from 6.0 to 4.5) and TTI (from 0.10 to 0.08) at W_{max} occurred in the rehabilitation group during the programme. The reduction in TTI was not significantly correlated with the fall in dyspnoea, as assessed by the Borg scale.

We conclude that 12 weeks of rehabilitation at home in COPD patients increases symptom-limited $V'O_2$ in combination with an increased W_{max}. At this significantly higher W_{max}, there was a reduction in dyspnoea, lactate level and inspiratory muscle workload. The reduction in dyspnoea was not related to a decreased inspiratory muscle workload. This study shows that rehabilitation at home can produce beneficial physiological improvements during exercise in patients with chronic obstructive pulmonary disease.

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Several studies have already shown that pulmonary rehabilitation increases the exercise tolerance of patients with chronic obstructive pulmonary disease (COPD) [1–11]. In contrast to most studies that were carried out in a clinical setting [1–9], we have developed a home-based rehabilitation programme. In a previous study, we have already shown that rehabilitation at home increases exercise tolerance and quality of life [12]. In this study we report upon the same group of patients, but we now address the effects of home rehabilitation on physiological parameters during a maximal exercise test and the effects on 6 min walking distance.

Until now, little has been known about the physiological changes during exercise after rehabilitation. The study of CASABURI and co-workers [5] showed that exercise training reduces ventilatory requirement in COPD patients by a reduction in ventilation accompanied by a decrease in lactate. However, COPD patients with severe airways obstruction might not reach their anaerobic threshold during exercise. Therefore, it will be unlikely that these patients have a reduction in ventilation as a result of exercise training [13, 14]. In this study, we investigated the effects of training on lactate production and metabolic gas exchange data in COPD patients with severe airways obstruction. Furthermore, we investigated whether training in these patients might facilitate a specific element of the ventilatory requirement during exercise, *i.e.* reducing the workload of inspiratory

muscles, assessed by the Tension Time Index (TTI) [15].

In addition to increased exercise tolerance, several studies found reduced dyspnoea after rehabilitation, despite the fact that the change in dyspnoea was not related to physiological changes [8,10,11,16]. Two studies have shown that dyspnoea is positively related to maximal inspiratory pressure ($P_{I,max}$) and the ratio of inspiratory time and total respiratory cycle time (t_I/t_{tot}) [17, 18].

Therefore, we investigated whether reduced dyspnoea after home rehabilitation, including inspiratory muscle training, was related to an improved $P_{I,max}$ and/or reduced t_I/t_{tot}. Changes in $P_{I,max}$ and t_I/t_{tot} reflect differences in inspiratory muscle workload, as assessed by the TTI (TTI=inspiratory pressure (P_I)/$P_{I,max}$ × t_I/t_{tot}) [15]. In contrast to our previous study [12], we now report upon dyspnoea during exercise as assessed by the Borg scale; we hypothesized that a reduced TTI during exercise might lead to a decrease in dyspnoea.

Patients and methods

Patients

We studied 45 patients (all smokers or ex-smokers, using inhaled corticosteroids, inhaled anticholinergic and/or inhaled β-agonists) with known COPD, according to the American Thoracic Society (ATS) criteria [19]. All patients were in a clinically stable condition (no exacerbations during the last 4 weeks). Entry criteria were: 1) forced expiratory volume in one second (FEV_1) <60% predicted; and 2) FEV_1/inspiratory vital capacity (IVC) <50%, both after two inhalations of 40 μg ipratropium bromide. All patients showed severe airways obstruction (mean FEV_1 44% pred, mean FEV_1/IVC 38%) and less than 10% reversibility (mean increase in FEV_1 of 0.15 L, 5% pred). Patients with evidence of ischaemic heart disease, intermittent claudication, musculoskeletal disorders or other disabling diseases were excluded. The study was approved by the Medical Ethics Committee of the University Hospital Groningen and all patients gave informed consent.

Study design

The patients were hospitalized for 2 days for their initial evaluation. Stratified randomization was used to achieve an approximate balance of important characteristics in all groups [20]. The patients were stratified for FEV_1 % predicted (< or ≥ 45% pred), maximal workload of the cycle ergometer test (< or ≥ 70 W), and the limiting factor in exercise capacity (ventilatory limitation or nonventilatory limitation) [21]. After this stratification, the patients were allocated at random to a 12 week home rehabilitation programme or to a control group. Two groups of 15 patients each started a rehabilitation programme for 18 months, whilst one group of 15 patients formed a control group. During the first 12 weeks, both rehabilitation groups received the same programme. They then received a different follow-up of physiotherapy. The patients of both rehabilitation groups were pooled, since the effects of rehabilitation during the first 12 weeks had been investigated. The following measurements were carried out before and 12 weeks after rehabilitation: 1) spirometry; 2) cycle ergometer test; and 3) 6 min walking distance (6MWD).

Rehabilitation programme

The patients carried out their home rehabilitation programme and were supervised by a multidisciplinary team: pulmonologist (RvA), local physiotherapist, local nurse, and general practitioner. Before the start of the study, the co-ordinating doctor (PJW) visited all physiotherapists, nurses and general practitioners to instruct them about the rehabilitation programme.

The physiotherapy programme consisted of relaxation exercises, breathing retraining, upper limb training, target-flow inspiratory muscle training (IMT), and exercise training on a home-trainer. Relaxation exercises were carried out according to Jacobson's technique of progressive muscle relaxation [22]. Breathing retraining consisted of education on COPD, pursed-lip breathing, expiratory abdominal augmentation, and synchronization of thoracic and abdominal movement [23]. Upper limb training was carried out according to the principles of proprioceptive neuromuscular facilitation (PNF), consisting of two exercises for each arm with the same weight and co-ordinated with breathing during exhalation [24]. IMT was carried out on an incentive flowmeter (Respirex, DHD medical products, New York, USA) with an added resistance according to DEKHUIJZEN *et al.* [25]. During IMT, the patients had to generate 70% of their maximal inspiratory pressure during 3 s, whilst the unloaded expiration was 4 s. Patients performed IMT twice a day for 15 min.

Exercise training was carried out on a home-trainer, following the method of ALLISON *et al.* [26]. The home-trainer was a mechanically-braked bicycle, equipped with an odometer. The workload was adjusted to maximal workload baseline (W_{max}) of the individual patients. Patients were instructed to keep their speed at 60 rotations·min^{-1}. They started exercise training for 4 min at 60% of their W_{max} in the cycle ergometer test. The time span was gradually extended to 12 min and the workload to a maximum of 75% of the W_{max}. All exercises were taught by the physiotherapist and practised during twice weekly visits to a local physiotherapist for 12 weeks. Patients had to practise twice a day for 30 min at home, following an individualized protocol. Patients performed relaxation exercises and breathing retraining on alternating days, and IMT, upper limb training and exercise training every day.

In addition to this physical training, patients were supervised once a month by a nurse and a general practitioner. The nurse visited the patients at home to give patients and family members a better insight into the potential disabilities and handicaps due to COPD. Information

was given about the pulmonary disease, various strategies for treatment, use of the medication, ways of coping with the disease, and the role of a rehabilitation programme in this coping strategy. Once a month, the patient visited the general practitioner, who supervised clinical status and maintenance treatment. The control group did not participate in the rehabilitation programme. In both groups, exacerbations were treated with oral corticosteroids and antibiotics, whilst their maintenance treatment was not changed. All patients, including the control group, received a diary to fill in the time they spent on daily activities, whilst the patients of the rehabilitation group also had to fill in the time they spent on exercises.

Outcome measurements

Baseline lung function characteristics

Total lung capacity (TLC), residual volume (RV), FEV_1, IVC, transfer factor of the lungs for carbon monoxide divided by alveolar volume ($T_{L,CO}/V_A$), volume-pressure (*V-P*) relationship, and static compliance (C_{st}) were measured at the initial evaluation. Static lung volumes were determined in a constant volume whole-body plethysmograph (Jaeger, Würzburg, FRG). FEV_1 and IVC were measured by means of a pneumotachograph (Jaeger, Würzburg, FRG). $T_{L,CO}/V_A$ was measured by the single-breath method. *V-P* diagrams of the lungs were recorded using an oesophageal balloon and a spirometer. C_{st} was calculated from the *V-P* diagram. Reference values are those of the European Coal and Steel Community (ECSC) [27].

Cycle ergometer test

Before the incremental symptom-limited cycle ergometer test (Jaeger, Würzburg, FRG), a brachial artery catheter was inserted and blood was periodically sampled for analysis of pH, arterial carbon dioxide tension (P_{a,CO_2}), arterial oxygen tension (P_{a,O_2}), and bicarbonate (HCO_3^-) (Ciba Corning 278, Medfield, USA). Patients respired through a mouthpiece and wore a noseclip during the incremental symptom-limited cycle ergometer test. Minute ventilation (V'_E), oxygen uptake (V'_{O_2}) and carbon dioxide output (V'_{CO_2}) were measured every 30 s from analysis of the expirate by a computerized system (EOS Sprint; Jaeger, Würzburg, FRG). Cardiac frequency (f_C) was monitored simultaneously (Marquette Electronics Inc., Milwaukee, WI, USA), and arterial oxygen saturation (S_{a,O_2}) was recorded continuously by an ear oximeter (Biox IIA, Biox Technology Inc., CO, USA).

After 1 min of unloaded pedalling, work rate increased by 10 W every minute. Blood samples were drawn at rest and then every 2 min just before work rate increased. The last samples were drawn after 2 min of recovery. Blood was also sampled for the determination of lactate level at rest and after 2 min of recovery. This sample was pipetted into an iced perchlorate solution and subsequently centrifuged [28]. Patients were instructed to stop when they could not continue due to dyspnoea or general fatigue.

The maximum workload (W_{max}) was defined as the highest work level reached and maintained for a full minute. To assess the level of dyspnoea, the intensity of this symptom was scored by the patients at rest and at W_{max} on a modified Borg category scale [29]. If patients tolerated an oesophageal balloon, pleural pressure was also measured during the test. The balloon, containing 0.5 mL of air and positioned in the middle of the oesophagus 40 cm from the anterior nares, was connected to a pressure-transducer (Hewlett Packard GmbH, No. 782018, Homburg, Germany) and the pressures generated were recorded. Inspiratory muscle workload at rest and at W_{max} was assessed by the TTI (TTI=(P_I/sniff oesophageal pressure (P_{oes}))×(t_I/t_{tot}) [21], in which P_I is the actual inspiratory pressure, sniff P_{oes} the maximal inspiratory oesophageal pressure, t_I the duration of the inspiration, and t_{tot} the duration of the total respiratory cycle. Sniff P_{oes} was assessed during a maximal sniff manoeuvre before the cycle test from RV [30], carried out at least five times; the maximal value was used in the analysis. Before each measurement, the pressure transducer was calibrated with a Gambro reference instrument (Gambro AB KO7046, Lund, Sweden); ambient pressure was used as zero level.

Six minute walking distance (6MWD)

After two training sessions to become familiar with the test, patients walked indoors as far as possible for a period of 6 min, without encouragement [31].

Statistical analysis

The Kolomogorov-Smirnov (K-S) test was used to compare distributions of the variables with standard normal distributions. Baseline outcome measures between the two groups were analysed using Student's unpaired t-test. The results after 12 weeks compared using baseline within each group were analysed with the paired t-test. Changes of the different variables during 12 weeks between both groups were investigated using Student's unpaired t-test. Correlation between the variables was investigated by Pearson's correlation coefficient. Significance level was set at p less than 0.05.

Results

Two patients dropped out of the rehabilitation group: one patient due to a cerebral tumour and one due to arthritis. The rehabilitation group, therefore, consisted of 28 patients and the control group of 15.

Outcome measures

Baseline lung function characteristics showed no significant differences between the rehabilitation and the

Table 1. – Baseline characteristics of the study population

	Rehabilitation group	Control group
Patients n	28	15
Gender M:F	23:5	14:1
Age yrs	64 (5)	62 (5)
$FEV_{1,b}$ L	1.2 (0.3)	1.2 (0.3)
$FEV_{1,a}$ L	1.3 (0.4)	1.4 (0.3)
FEV_1 % pred	44 (11)	45 (9)
FEV_1/IVC %	39 (8)	36 (7)
IVC % pred	84 (16)	94 (15)*
TLC % pred	118 (14)	114 (11)
RV/TLC % pred	151 (24)	133 (18)*
C_{st} L·kPa^{-1}	4.3 (3.1)	5.3 (3.0)
$T_{L,CO}/V_A$ % pred	65 (23)	65 (25)
pH	7.42 (0.02)	7.42 (0.02)
P_{a,O_2} kPa	9.2 (1.0)	9.5 (0.9)
P_{a,CO_2} kPa	5.5 (0.5)	5.4 (0.6)

All values are expressed as mean, and SD in parenthesis. M: male; F: female; $FEV_{1,b}$: forced expiratory volume in one second before bronchodilation with two inhalations of 40 µg ipratropium bromide; $FEV_{1,a}$: FEV_1 after bronchodilation; % pred: expressed as a percentage of the predicted value; FEV_1/IVC: FEV_1 expressed as a percentage of the slow inspiratory vital capacity; TLC: total lung capacity; RV: residual volume; C_{st}: static lung compliance; $T_{L,CO}/V_A$: transfer factor of the lungs for carbon monoxide divided by alveolar volume; P_{a,O_2}: arterial oxygen tenson; P_{a,CO_2}: arterial carbom dioxide tension. *: p<0.05, unpaired test.

control group, except for IVC and RV/TLC % pred (table 1). There was also no significant difference between both groups for smoking status or drug therapy. In the control group, FEV_1 and IVC were significantly (p<0.05 and p<0.01, respectively) lower after 12 weeks compared to baseline (table 2). The decrease in IVC in the control group was significantly (p<0.05) larger than the decrease in the rehabilitation group.

Cycle ergometer test

At baseline, 39 patients (26 in the rehabilitation group and 13 in the control group) had a ventilatory limitation in their exercise capacity: an increase in P_{a,CO_2} (at rest 5.5±0.6 kPa (mean±SD), at W_{max} 6.1±0.7 kPa), a small decrease in P_{a,O_2} (at rest 9.2±0.9 kPa, at W_{max} 9.0±1.5 kPa) and a stable alveolar to arterial oxygen difference ($P_{A\text{-}a,O_2}$) (at rest 4.4±1.1 kPa, at W_{max} 4.2±1.3 kPa) [21]. Furthermore, their ventilation at maximal exercise was close to their maximal voluntary ventilation (MVV = 37.5 × FEV_1) [32]. Two patients in each group showed a diffusion limitation in exercise capacity (rise in $P_{A\text{-}a,O_2}$ >2 kPa) [21]. Twenty three patients in the rehabilitation group stopped the test because of dyspnoea, one because of leg fatigue, and four because of a combination of both. Ten patients in the control group stopped because of dyspnoea, one because of fatigue, and four because of a combination of both. No differences were observed between the two groups at baseline (table 3).

Table 2. – Spirometry

	Rehabilitation group		Control group	
	Baseline	12 weeks	Baseline	12 weeks
IVC L	3.4 (0.9)	3.3 (0.8)	3.9 (0.8)	3.5 (0.9)**##
FEV_1 L	1.3 (0.4)	1.3 (0.3)	1.4 (0.3)	1.2 (0.3)*

All values are expressed as mean and SD in parenthesis. *: p<0.05, paired t-test; **: p=0.001, paired t-test; #: p<0.05, unpaired t-test between the changes in both groups. For abbreviations see legend to table 1.

The rehabilitation group showed an improved W_{max} (p<0.05) of 10% (from 70 to 78 W) after 12 weeks (table 3). W_{max} decreased by 9% in the control group, the difference between the two groups being significant (p<0.01). The symptom-limited V'_{O_2} (V'_{O_2}-SL) in the rehabilitation group was also significantly higher after 12 weeks compared to the control group (p<0.05). The dyspnoea score of the rehabilitation group at W_{max} was lower (p<0.01) after 12 weeks compared to baseline; there was no significant difference from baseline in the control group.

The rehabilitation group showed a significant decrease in TTI both at rest (p<0.01) and at W_{max} (p<0.05) after 12 weeks. This group had a decrease in maximal lactate production (p<0.05) of 0.6 mEq·L^{-1} after 12 weeks compared to baseline. Decreases in TTI and lactate were found at a significantly higher W_{max} compared to baseline (78 *vs* 70 W). Since not all patients could swallow the oesophageal balloon, TTI was measured in 30 patients only (20 in the rehabilitation group and 10 in the control group). The decrease in the TTI at W_{max} in the rehabilitation group was due to a decrease in the ratio

Table 3. – Results at maximal workload during bicycle ergometer test

	Rehabilitation group		Control group	
	Baseline	12 weeks	Baseline	12 weeks
W_{max} W	70 (30)	78 (31)*	79 (27)	71 (28)##
f_C beats·min^{-1}	120 (13)	122 (17)	126 (15)	121 (16)*
f_R breaths·min^{-1}	30 (6)	29 (6)	29 (4)	28 (3)
V'_E L·min^{-1}	30.4 (9.8)	31.1 (10.0)	32.7 (8.5)	29.9 (9.8)
V'_{O_2} L·min^{-1}	1.0 (0.3)	1.1 (0.4)*	1.1 (0.3)	1.0 (0.3)#
V'_{CO_2} L·min^{-1}	0.9 (0.4)	1.0 (0.4)	1.0 (0.4)	0.9 (0.3)
VE_{O_2}	30.7 (4.4)	29.5 (4.4)	30.3 (4.0)	29.8 (4.7)
RQ	0.9 (0.1)	0.9 (0.1)	0.9 (0.1)	0.9 (0.1)
pH	7.37 (0.04)	7.36 (0.04)	7.35 (0.05)	7.34 (0.03)
P_{a,CO_2} kPa	6.2 (0.6)	6.0 (0.7)	6.0 (0.9)	5.9 (0.8)
P_{a,CO_2} kPa	8.9 (1.4)	8.9 (1.4)	9.2 (1.7)	9.1 (1.9)
Lactate mEq·L^{-1}	3.7 (1.7)	3.1 (1.3)*	4.1 (1.9)	3.4 (1.7)
TTI rest	0.05 (0.06)	0.03 (0.01)	0.03 (0.02)	0.03 (0.02)
TTI	0.10 (0.03)	0.08 (0.03)*	0.09 (0.04)	0.09 (0.03)
Dyspnoea	6.0 (2.6)	4.7 (1.8)**	5.9 (2.5)	5.7 (2.4)

All values are expressed as mean and SD in parenthesis. W_{max}: maximal workload sustained for one minute; f_C: cardiac frequency; f_R: respiratory frequency; V'_E: minute ventilation; V'_{O_2} oxygen uptake; V'_{CO_2}: carbon dioxide output; VE_{O_2}: ventilatory equivalent for oxygen; RQ: respiratory quotient; lactate: lactate production 2 min postexercise; dyspnoea: perceived breathlessness on the Borg scale; TTI: Tension Time Index = (P_I/sniff P_{oes} × t_I/t_{tot}). *: p<0.05, paired t-test; **: p<0.01, paired t-test; #: p<0.05, unpaired t-test; ##: p<0.01, unpaired t-test between the changes in both groups.

t_I/t_{tot} (from 0.38 to 0.33; p<0.05) and an increase of sniff P_{oes} (from 8.0 to 8.4 kPa, p<0.05). Changes of sniff P_{oes} (from 8.0 to 8.1 kPa) and the ratio t_I/t_{tot} (from 0.38 to 0.37) were not significant in the control group.

Although both dyspnoea and TTI at W_{max} decreased in the rehabilitation group, the changes were not significantly correlated (r=0.30) to each other.

Minute ventilation (V'_E) measured after 12 weeks at identical baseline workloads, showed a significant decrease in the rehabilitation group (from 31.8 (9.9) to 29.4 (9.6) $L \cdot min^{-1}$; p<0.01). In the control group there was a nonsignificant decrease in V'_E assessed at the same workload from 34.9 (9.0) to 32.4 (9.0) $L \cdot min^{-1}$. Whilst only six patients from the control group reached their baseline W_{max}, 23 patients from the rehabilitation group reached their baseline W_{max}.

Six minute walking distance (6MWD)

There was no significant difference in the 6MWD between the two groups at baseline. The 6MWD increased significantly in the rehabilitation group from 438 (84) to 447 (87) m. In contrast, the control group showed a decrease from 472 (121) to 444 (141) m, the difference between the two groups being significant (p<0.05).

Discussion

This study showed that home rehabilitation of COPD patients with severe airways obstruction improved physiological parameters during maximal exercise tests. We found an improved V'_{O_2}-SL, together with a decreased lactate, TTI, and dyspnoea at a significantly higher W_{max} after 12 weeks of training. The decrease of the TTI was due to a fall in the t_I/t_{tot} ratio and an increase in maximal inspiratory pressure (sniff P_{oes}). However, the fall in TTI was not correlated with the decrease in dyspnoea. The rehabilitation group also showed an improved 6 min walking distance.

This is the first controlled study carried out at home in which an improved exercise tolerance and V'_{O_2}-SL was found in association with a decrease in lactate and TTI. Whilst the finding of improved exercise tolerance agrees with other home rehabilitation studies [10, 11, 16], physiological improvements have only been shown in hospital-based in-patient and out-patient studies [2, 5, 6]. In contrast to CASABURI and co-workers [5], who found that a reduction in lactate was related to a decrease in ventilation (r=0.75), we did not find this relationship. This is probably due to patient selection. Firstly, the patients in the study by CASABURI and co-workers [5] had a better FEV1 (1.9 L, 56% pred) compared to our study (1.3 L, 44% pred). Secondly, the patients in the study by CASABURI and co-workers [5] showed a normal P_{a,O_2} at rest in contrast to our patients (11.0 and 8.9 kPa, respectively). Finally, CASABURI and co-workers [5] included patients only if their end-exercise lactate level exceeded 3 $mEq \cdot L^{-1}$, whilst in our study only 17 out of 45 patients developed comparable lactate levels (six patients in the control group and 11 in the rehabilitation group). Even in these 11 patients from the rehabilitation group, whose end-exercise lactate level exceeded 3 $mEq \cdot L^{-1}$, there was no significant decrease in V'_E at W_{max} and no relationship with the decrease in lactate.

Thus, the majority of our patients were not able to exercise at a work rate that resulted in lactic acidosis [13, 14]. This was also reflected in the postexercise value of the respiratory quotient (table 3). As a consequence of the minor reduction in lactate, our patients did not show a decrease in ventilation at W_{max}. Although V'_E assessed at their identical W_{max} baseline, decreased significantly after training, the same decrease in V'_E was shown in the control group. However, a good comparison between both groups is nearly impossible, because only six out of 15 patients from the control group reached their W_{max} baseline, in contrast to 23 patients from the rehabilitation group. The decrease in W_{max} in the control group might be responsible for the observed decrease in lactate level.

We found a reduced TTI after training, which means that rehabilitation can facilitate a specific element of the ventilatory requirement, *i.e.* the inspiratory muscle workload. With regard to the relevance of the small decrease in TTI in the rehabilitation group, two points are important. Firstly, the TTI in the rehabilitation group was assessed at a significantly higher W_{max}, which suggests that the decrease in TTI may have been larger when it was assessed at comparable workload. Secondly, the W_{max} of the control group decreased after 12 weeks, whilst there was no change in the TTI. Therefore, we believe that the small, though significant, decrease in TTI in the rehabilitation group is relevant. Although the TTI at W_{max} in the control group is less than the TTI in the rehabilitation group at baseline, it did not reach significance.

In the present study, we used sniff P_{oes} in the TTI as maximal inspiratory pressure, whilst BELLEMARE and GRASSINO [15] used transdiaphragmatic pressure (P_{di}). However, according to the study of LAROCHE *et al.* [33], sniff P_{oes} is a valid assessment of inspiratory muscle strength and shows a good correlation with P_{di}.

The decrease of TTI was due to both a decrease of t_I/t_{tot} and an increase of sniff P_{oes}. Because the patients carried out a comprehensive rehabilitation programme, it cannot be established which part is responsible for these changes. The improved inspiratory muscle strength may be due to IMT, because the intensity of whole body exercises is probably inadequate to increase inspiratory muscle strength [34]. Furthermore, it has been shown that a combination of rehabilitation and IMT leads to a significantly larger increase of inspiratory muscle strength than rehabilitation alone [25]. Though we included COPD patients in whom the ventilatory pump limits exercise capacity and who, therefore, might benefit from IMT [35], the changes in sniff P_{oes} caused by IMT were not impressive. SMITH *et al.* [36] and GUYATT *et al.* [37] suggested that resistance training in which breathing pattern and flow rate are controlled may have positive effects. Although we controlled the flow rate and breathing pattern during IMT, we found a smaller improvement of

inspiratory muscle strength than DEKHUIJZEN *et al.* [25]. This may be due to the fact that DEKHUIJZEN *et al.* [25] assessed inspiratory muscle strength by $P_{I,max}$, whilst we used the sniff P_{oes}. Moreover, the patients in the study by DEKHUIJZEN *et al.* [25] were supervised by the physiotherapist during their performance of IMT. In our study, IMT was taught by the physiotherapist but training at home was unsupervised. Nevertheless, sniff P_{oes} was significantly improved in the rehabilitation group, suggesting that unsupervised IMT at home can be successful.

The rehabilitation group showed significantly less dyspnoea at W_{max} after 12 weeks of training. Though several rehabilitation approaches may result in reduced dyspnoea [38], exercise training appears to be the best intervention, as it affects both psychological and physiological aspects of dyspnoea. Tolerance and adaptation to exercise is most probably the mechanism by which training improves dyspnoea, as pulmonary and cardiac function generally do not change [1, 8, 16]. We observed, next to reduced dyspnoea, a decrease in TTI after training. A decreased input from muscle receptors may account for peripheral desensitization to dyspnoea [38]. This finding is in accordance with studies in healthy volunteers and in patients with a variety of diseases, which showed that dyspnoea, assessed by a Borg score, was related to t_I/t_{tot} [17, 18]. However, we could not find a significant correlation between changes in dyspnoea and t_I/t_{tot} or TTI, suggesting that other factors, such as individual behaviour, personality and emotional state, may have influenced the extent of breathlessness [38].

The control group had a decrease in 6MWD, suggesting that exercise tolerance may deteriorate when COPD patients do not participate in a rehabilitation programme. Further controlled long-term rehabilitation studies are needed to confirm this observation.

Finally, the unexpected decrease both in FEV_1 and IVC after 12 weeks in the control group needs further discussion. This decline in lung function might have been the result of better medical treatment in the rehabilitation group than in the control group. However, the maintenance treatment in both groups did not change during these 12 weeks and there was no difference in the mean number of exacerbations during 12 weeks between the two groups (rehabilitation group: 26 exacerbations in 28 patients; control group: 11 exacerbations in 15 patients). At present, we cannot explain the large decrease in lung function in our control group. Follow-up of the patients may show whether this is an incidental finding.

In summary, we conclude that home rehabilitation of patients with COPD may be successful by improving maximal workload, symptom-limited oxygen uptake, and maximal inspiratory pressure, together with a decrease of lactate, inspiratory muscle workload (TTI), and dyspnoea during maximal exercise. The interpretation of these moderate, though significant, improvements in a small number of patients is difficult, but taken together they indicate that home rehabilitation of patients with COPD can achieve benefits traditionally associated with intensive hospital-based programmes.

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