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Short term effect of intermittent negative pressure ventilation in COPD patients with respiratory failure

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ABSTRACT: Ten patients with stable chronic obstructive pulmonary disease (COPD) and hypercapnic respiratory failure were randomly submitted to intermittent negative pressure ventilation (INPV) 6 h per day for 5 consecutive days by either a cuirass or pneumo wrap ventilator. The effects were assessed by measurements of spirometry, blood gases, maximal inspiratory (MIP) and expiratory (MEP) pressures, 12 minutes walking distance test (12 mwd), sensation of dyspnoea by a visual analogue scale (VAS) and diaphragmatic electromyographic activity (Edi). Edi was recorded during INPV sessions in only 7 patients. The same measurements apart from Edi were also performed in 8 matched control patients randomly submitted to conventional physiotherapy. During INPV, Edi activity was reduced, at least temporarily down to 50% of baseline values. Comparison of baseline with post INPV values showed no changes in thoracic gas volume (TGV), forced expiratory volume in one second (FEV_1), FEV_1/forced vital capacity (FVC), arterial oxygen partial pressure (Pao_2) and MEP; significant improvements were seen in MIP, vital capacity (VC), VAS, and 12 mwd only in patients submitted to INPV. A significant improvement in $Paco_2$ was observed in both groups of patients. We conclude that INPV may be effective in improving the functional reserve of the inspiratory muscles in selected COPD patients with hypercapnic respiratory failure and signs of inspiratory muscle dysfunction.

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Patients with chronic obstructive pulmonary disease (COPD) often show signs of compromised performance of the inspiratory muscles (IM) such as dyspnoea, tachypnoea, paradoxical thoraco-abdominal motion during inspiration, hypercapnia, reduction of vital capacity (VC) and exercise performance, decrease of IM strength and endurance. All these signs have been attributed to incipient or actual IM fatigue [1, 2]; although the importance of fatigue in this situation is unresolved it may contribute to the pathogenesis of respiratory failure [3–10].

Recent studies have suggested that a period of rest of the IM by mechanical ventilation may be useful to recover from fatigue. Intermittent negative pressure ventilation (INPV) with body ventilators, while requiring no airway intubation, seems promising in improving the functional reserve of the IM but experience in this field is still limited. A definite treatment schedule has not yet been defined and conflicting results have been reported [1, 11–15]. Cropp and Di Marco [12] observed an improvement in IM strength and endurance in COPD patients after INPV administered for 3–6 h per day for 3 consecutive days; the benefits lasting no longer than 5 days. However, Gutierrez *et al.* [13] found that an 8 h per week schedule with INPV in patients with respiratory failure was effective in long term treatment, while Pluto *et al.* [14] found no significant changes in arterial carbon dioxide partial pressure ($Paco_2$), maximal inspiratory pressure (MIP) and physical capacity in 5 patients with COPD after 8 weeks of INPV for 4–7 h per day.

We have investigated the effects of treatment with INPV administered 6 h per day for 5 consecutive days in a randomized, controlled study in hypercapnic COPD patients with signs of impaired performance of IM.

Patients

Studies were carried out in 18 consecutive male patients with severe COPD meeting the following criteria: arterial oxygen partial pressure (Pao_2) < 60 mmHg, $Paco_2$ > 50 mmHg, MIP at functional residual capacity (FRC) < 60 cmH_2O, at least one of the following signs which have been attributed to IM fatigue [1]: dyspnoea, tachypnoea, abdominal paradox, chest-abdomen asynchrony.

All patients were in a stable condition at the time of the study with no exacerbation of respiratory symptoms during the preceding month. Malignancy, asthma or other relevant associated diseases were excluded. All patients were tested and treated while taking their usual medical therapy such as oral beta agonists, methylxanthines and long term oxygen. Informed consent to participate in the study was given by all patients.

Methods

Static lung volumes were measured by means of a body plethysmograph (Bodytest, Jaeger, Wurzburg, West Germany); dynamic lung volumes by means of a pneumotachograph with volume integrator (Jaeger PT 1020). Blood gas analysis was performed by a Radiometer ABL30 (Copenhagen, Denmark) blood gas analyser on arterialized blood obtained from the ear lobe.

Respiratory muscle strength was assessed by measuring MIP at residual volume (RV) and FRC and maximal expiratory pressure (MEP) at total lung capacity (TLC) according to the method of BLACK and HYATT [16]. The pressure developed during a maximal respiratory effort against a closed mouthpiece with a 0.6 mm leak, was measured by a 143 PC 05D Micro Switch (Freeport, Ill, USA) differential pressure transducer (range of linearity: ±350 cmH_2O) connected to the mouthpiece. The best of three efforts was recorded.

Patients' subjective evaluation of dyspnoea was recorded at rest and after a mild effort (walking on a treadmill (Laufergotest, Jaeger, Wurzburg, West Germany) 100 m at 1.5–2.5 $km \cdot h^{-1}$) using a visual analogue scale (VAS) [17]. Exercise performance was assessed by the 12 min walking distance test (12 mwd) according to MCGAVIN and coworkers [18].Verbal encouragement was given continuously during the test, but the patient was free to stop when desired. Three practice attempts were performed in the 3 days preceding the initial evaluation, the best of which was considered as the baseline value. Results are expressed as actual distance walked in metres.

All measurements likely to be influenced by motivation and learning were performed under the supervision of the same operator (A.N.) unaware of the patient's treatment.

After baseline measurements, patients were assigned randomly to either a study group (group I) or a control group (group II). Ten subjects in group I received INPV by either a cuirass (Thompson Maxivent, Puritan Bennett Corporation, Boulder Co, USA) or a pneumo-wrap (170 C Respirator, Lifecare Lafayette, Co, USA) ventilator. The ventilators were set to intermittently deliver external negative pressure between -20 and -35 cmH_2O at a frequency slightly greater than the patient's own breathing frequency. Patients received INPV for 2 sessions per day, each session lasting 3 h with a 2 h interval for lunch, for 5 consecutive days.

In order to assess the actual degree of IM rest in 7 patients submitted to INPV, the diaphragmatic electromyogram (Edi) was recorded with surface electrodes placed in the 6th and 7th intercostal spaces on the anterior axillary line during the first session of treatment. The signal was filtered between 50 and 1600 Hz and was then rectified and integrated with a leaky integrator. Control measurements were made during spontaneous breathing in the supine position with the ventilator switched off for about 15 minutes, thereafter the negative pressure was applied and Edi was recorded for about 1 h. Edi was measured in arbitrary units, as the mean of 10 consecutive respiratory cycles during quiet breathing and at the moment in which Edi became relatively constant. Changes in Edi were expressed as percentage of the value obtained during spontaneous breathing. A 50% reduction of Edi from the basal value in at least 5 consecutive respiratory cycles was considered as evidence of actual rest of the diaphragm.

Eight subjects in group II received conventional physiotherapy 1 h per day as coordination exercises [19].

Measurements were repeated 24 h after discontinuation of the last session of treatment. Clinical observations were performed by physicians aware of the therapy to which the patient had been submitted, but not necessarily involved in the study.

Results are expressed as mean (standard deviation). Data were analysed using Student's paired t-test. A value of $p<0.05$ was considered significant.

Table 1. – Characteristics and criteria of inclusion of patients in study (mean, SD)

		Group I n=10	Group II n=8
Age	yrs	58 8	62 10
Weight	kg	63 14	63 11
Height	cm	160 8	166 5
MIP (FRC)	cmH_2O	34 13	41 16
$Paco_2$	kPa	7.5 0.5	7.5 0.9
	(mmHg)	(56) (4)	(56) (7)
Pao_2	kPa	6.8 0.5	6.5 0.8
	(mmHg)	(51) (4)	(49) (6)

MIP (FRC): maximal inspiratory pressure at functional residual capacity; $Paco_2$ and Pao_2: arterial carbon dioxide and oxygen partial pressures, respectively.

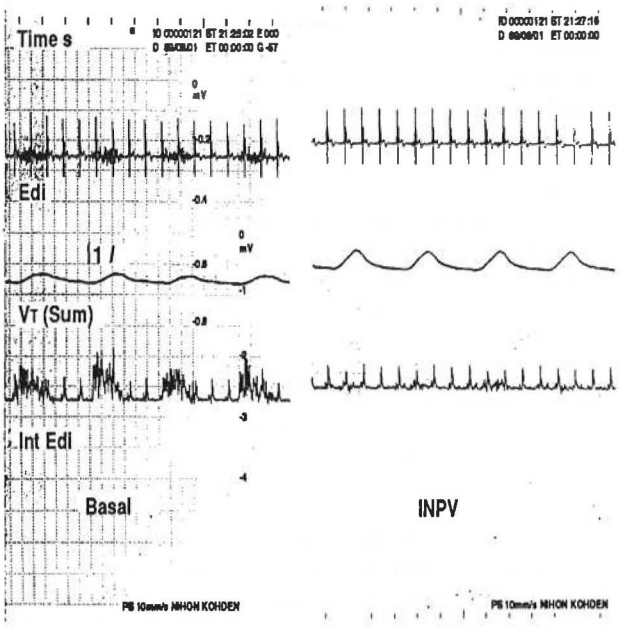

Fig. 1. – Recording of integrated Edi in a representative subject before (left) and during INPV (right). Top–bottom: Time (s) diaphragmatic electromyogram (Edi), tidal volume (*l*) from sum signal of Respitrace (inspiration upward), integrated Edi.

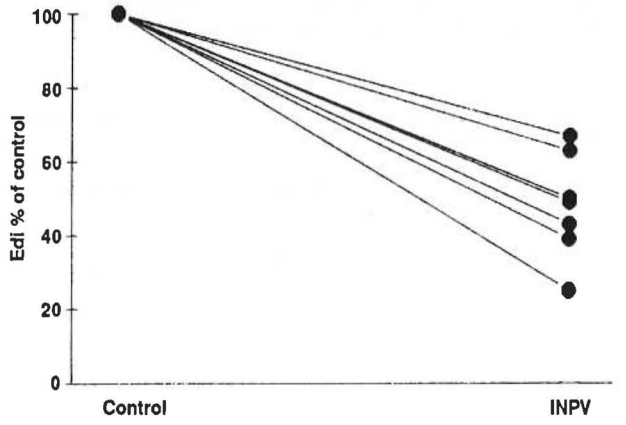

Fig. 2. – Mean change in Edi (% of control values obtained during spontaneous breathing) during an INPV session in 7 patients.

Results

The characteristics of the 2 groups of patients and the baseline values of the criteria used for admission are shown in table 1.

There were no significant differences between control and study (INPV) group for the physiological measurements, drug and oxygen therapy or smoking habits.

Complete recordings of Edi were obtained in only 7 patients of group I. All of these patients showed a marked reduction in Edi during INPV. In some patients quite a large variability occurred among the different respiratory cycles, but at least 5 consecutive breathing cycles in which a 50% reduction of Edi from control during spontaneous breathing could be identified, in all 7 patients. A typical tracing is shown in figure 1. Figure 2 depicts the mean changes in Edi in individual patients during one run with INPV.

The comparison of pre and post treatment measurements of lung function is shown in table 2. After the period of study there were no significant changes in thoracic volumes as assessed by thoracic gas volume (TGV), in expiratory flows as assessed by forced expiratory volume in one second (FEV_1), and FEV_1/forced vital capacity (FVC) in both groups while a significant increase in VC was observed only in group I.

Table 2. – Lung function studies before and after the treatment (mean, SD).

	FEV_1 *l*	FEV_1/FVC %	TGV % pred	VC
Group I				
before	0.77	45	196	56
	0.39	8	60	18
after	0.78	42	204	63*
	0.52	10	75	14
Group II				
before	0.79	51	141	46
	0.38	8	18	10
after	0.78	52	175	50
	0.40	6	40	13

*: Significantly greater than baseline value ($p<0.05$); FEV_1: forced expiratory volume in one second; FVC: forced vital capacity; TGV: thoracic gas volume; VC: vital capacity.

The effects of treatment on arterial blood gases and respiratory muscle strength are shown in table 3.

Post treatment measurement of MIP at FRC could be performed in only 7 patients of group I and in 6 patients of group II. A significant improvement in MIP both at FRC and at RV was only found in group I. Individual data of MIP at RV are shown in figure 3. $Paco_2$ significantly decreased in both groups. Individual data of $Paco_2$ are shown in figure 4, bottom. Only 5 out of 10 patients of the study group showed a decrease from the basal value greater than 8.5%, while none of the patients of the control group showed such an improvement.

No other significant changes were observed in control patients. Table 4 depicts the effects of treatment on the subjective perception of dyspnoea as assessed by VAS and on the exercise capacity of patients as assessed by 12 mwd. VAS and 12 mwd significantly improved only in group I. Individual data of 12 mwd are shown in figure 4, top.

No correlation was found between the decrease in Edi during INPV and the degree of improvement in individual patients.

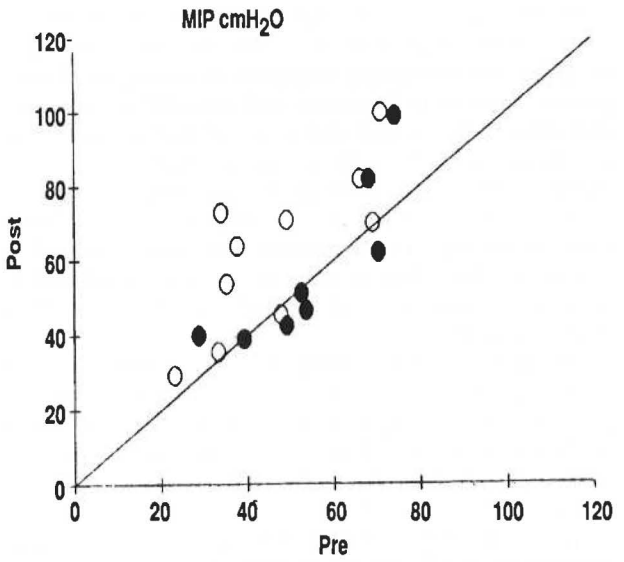

Fig. 3. – Comparison of baseline and post treatment values of MIP. Data points represent individual subjects. Open circles are patients treated with INPV. Solid circles are control patients. The solid line is the line of identity. (Abbreviations in the text).

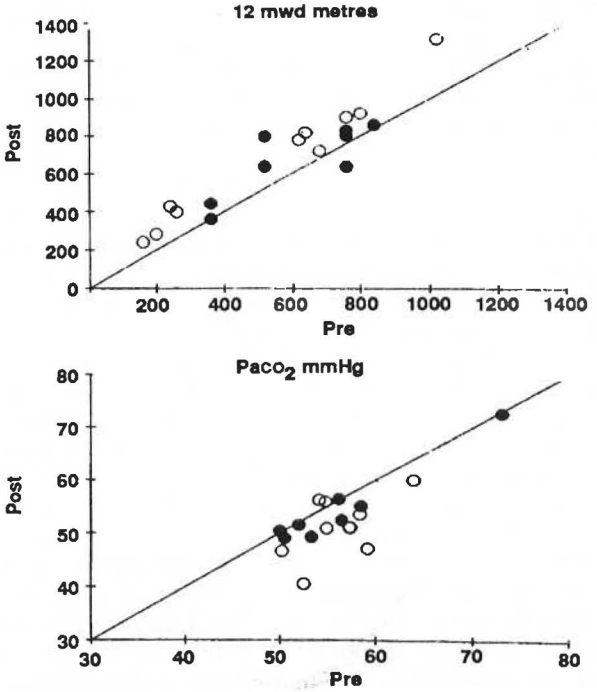

Fig. 4. – Comparison of baseline and post treatment values of $Paco_2$ (bottom) and 12 mwd (top). Symbols and abbreviations as in figure 3. Two patients treated with INPV showed identical results in $Paco_2$.

Table 3. – Effects of treatment on blood gases and respiratory muscle strength mean±(SD).

		Group I		Group II	
		before	after	before	after
Pao_2	kPa	6.8 (0.5)	7.1 (0.9)	6.5 (0.8)	6.5 (0.8)
	mmHg	51 (4)	53 (7)	49 (6)	49 (6)
$Paco_2$	kPa	7.5 (0.5)	6.8** (0.8)	7.5 (0.9)	7.3* (1.1)
	mmHg	56 (4)	51 (6)	56 (7)	55 (8)
pH		7.39 (0.03)	7.40* (0.02)	7.38 (0.02)	7.39 (0.02)
MIP (FRC)	cmH_2O	34 (13)	42* (12)	41 (16)	41 (18)
MIP (RV)	cmH_2O	47 (17)	62* (22)	54 (16)	58 (22)
MEP	cmH_2O	96 (42)	90 (33)	83 (29)	99 (36)

*: Significantly different from baseline value ($p<0.05$); **: significantly different from baseline value ($p<0.01$); MIP (RV): maximal inspiratory pressure at residual volume; MEP: maximal expiratory pressure. For other abbreviations see legend to table 1.

Discussion

This small randomized controlled study indicates that short term INPV treatment by cuirass or pneumo wrap ventilators may improve IM performance as assessed by MIP, $Paco_2$, subjective evaluation of dyspnoea, and exercise tolerance in COPD patients with respiratory failure. The results suggest that short term rest of the IM may be useful in temporarily reversing some of the processes leading to impaired IM function in COPD.

The treatment schedule of INPV was similar to that employed by CROPP and DI MARCO [12]. They ventilated COPD patients for 3–6 h per day on 3 consecutive days and found a mean increase in MIP of 10 cmH_2O and a three-fold increase of sustained voluntary hyperventilation. Five days after discontinuation of assisted ventilation the maximum duration time declined to baseline values. Their patients were selected on the basis of a reduced maximal voluntary ventilation. Our patients were selected on the basis of reduced ventilatory function and

of clinical signs which have been attributed to IM fatigue [1]. GUTIERREZ *et al.* [13] administered INPV for 8 h one day a week and BRAUN and MARINO [11] treated their patients 4–10 h per day showing a long term improvement of indices of IM function. RABINOVITCH *et al.* [15] found improved EMG signs of diaphragmatic fatigue with only 1 h of treatment. By contrast, PLUTO *et al.* [14] found no significant improvement in IM function after comparable periods of INPV. These differences may reflect different criteria for patient selection or, as recently, outlined by, ROCHESTER [2], different levels of IM fatigue, our patients having to be considered as suffering from actual, rather than incipient IM fatigue. The reduction in Edi activity observed in our 7 patients (figs. 1 and 2) gives objective information on the degree of diaphragmatic rest during INPV. This is in keeping with the studies of ROCHESTER *et al.* [20] and LEVY *et al.* [21]. On the other hand, RODENSTEIN *et al.* [22] using a Drinker tank respirator, found that short runs of INPV in naive patients did not result in changes in diaphragmatic EMG (by a bipolar oesophageal electrode), whereas in a patient habituated to INPV they found an immediate and nearly complete cessation of inspiratory activity. As for this it should be stressed that our patients showed a reduction in Edi after at least 20 minutes of adaptation to INPV. Furthermore, GUTIERREZ *et al.* [13] observed transdiaphragmatic pressure swings of zero in two of their patients during INPV, confirming that the diaphragm is put at rest also from a mechanical point of view.

In our patients, rest of the IM significantly improved MIP (table 3, fig. 3). Indices of obstruction such as FEV_1 and FEV_1/FVC and indices of the geometrical abnormalities associated with hyperinflation such as TGV were unchanged by INPV, therefore changes in MIP could not be ascribed to changes in muscle length, curvature or mechanical arrangement [23].

The increase in MIP observed in patients submitted to INPV could be ascribed to motivation and learning effects. This is very unlikely however, because no change in MIP was observed in the control group. The measure of MIP is a global index of the force generating ability of the IM as a whole, not specifically applicable to one muscle group. In COPD patients, IM dysfunction may be due not only to mechanical disadvantage [24] but also to the metabolic and biochemical derangements due to the disease.

Intermittent rest of IM could allow them to recover from metabolic derangements without running the risk related to excessive rest leading to disuse atrophy.

Group I showed a significant reduction in $Paco_2$ (table 3, fig. 4, bottom). Our results confirm those of CROPP and DI MARCO [12]. Their control group, however, which received no treatment did not show the slight but significant improvement of $Paco_2$ we found in our control group. This may be due to the physiotherapeutic regimen to which our control group patients were submitted.

Although the role of IM dysfunction in the development of hypercapnia in COPD patients is not clear, there are reports indicating that these factors are related [6]. Improvement in dyspnoea, diaphragmatic excursion, and maximal expiratory pressure has been reported in controlled studies [19] employing breathing exercises, similar to those performed by the patients of group II including abdominal-diaphragmatic breathing, headdown posture, pursed-lip breathing and controlled respiratory frequency. MIP, a true index of IM performance, was not measured in those studies: in our study MIP did not change with similar techniques suggesting the absence of effect on IM function. The improvement of $Paco_2$ achieved by our control patients may have occurred as a result of other changes induced by the physiotherapeutic regimen such as improved coordination or a more relaxed posture.

The lack of improvement of Pao_2 (table 3) is not surprising, as it is well known to be determined also by perfusion and ventilation/perfusion mismatching which might not be influenced by INPV treatment. On the other hand, other authors have observed an improvement of this parameter too [12].

All of our patients showed a reduction of dyspnoea as assessed by VAS and an increase in 12 mwd (table 4, fig. 4, top). Even though the improvements in these indices might be related in part to a learning effect, they were observed only in group I. 12 mwd is dependent on the physical capacity of patients and may be used as an index of their ability to cope with daily activity. After treatment, patients of group I but not of group II gave the observer a suggestion of improvement of the clinical signs of IM dysfunction. Visual inspection confirmed increased abdominal motion, better rib-cage-abdominal synchrony and reduction or reversal of abdominal paradox in all patients. PLUTO and coworkers [14] who found no significant improvement in IM strength, observed reduced dyspnoea in their patients. It is unlikely that the improvements observed after INPV could be ascribed to medical therapy including oxygen as these were similar in both groups and there were no changes in expiratory flows and lung volumes. In comparison to INPV we used a coordination exercise treatment which is not known to induce an improvement in exercise tolerance in such a short term. A true placebo that is the submission of control patients to negative ventilators at "zero" pressure would be more appropriate, nevertheless with our ventilators even a minimum possible negative pressure (-2, -3 cmH_2O) was able to reduce transdiaphragmatic pressure swings in normals [25]. Since, however, the patients of group II were not submitted to true placebo therapy and patients of group I did not receive conventional physiotherapy, a placebo effect in group I cannot be excluded. The effects observed after INPV declined in about 2 weeks. The repetition of the same treatment schedule again induced improvements of the same parameters [26].

No relevant side effects were observed during INPV. Furthermore, in a parallel study [27], we observed no change in mean pulmonary artery pressure and cardiac output during cuirass ventilation in COPD patients with pulmonary hypertension, so INPV may therefore be a more suitable form of ventilation than intermittent positive pressure ventilation given by intubation which is known to reduce cardiac output, and requires airway intubation [28].

In conclusion, in selected COPD patients with respiratory insufficiency and signs of inspiratory muscle dysfunction, intermittent negative pressure ventilation seems to be useful in reducing inspiratory muscle activity, allowing their rest and improving their functional reserve without any adverse effect.

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Effet à court terme d'une ventilation sous pression négative intermittente, chez les patients BPCO en insuffisance respiratoire. N. Ambrosino, T. Montagna, S. Nava, A. Negri, S. Brega, C. Fracchia, L. Zocchi, C. Rampulla.

RÉSUMÉ: Dix patients atteints de bronchopneumopathie obstructive chronique (BPCO) et insuffisance respiratoire hypercapnique, ont été soumis de façon randomisée à une ventilation sous pression négative intermittente (INPV) 6 heures par jour pendant 5 jours consécutifs, soit à la cuirasse, soit par un ventilateur pneumo wrap. Les résultats ont été appréciés en mesurant la spirométrie, les gaz du sang, les pressions inspiratoires maximales (MIP) et expiratoires maximales (MEP), le test de distance de march pendant 12 minutes (12 mwd), la sensation de dyspnée au moyen d'une échelle visuelle analogique (VAS) et l'activité électromyographique diaphragmatique (Edi). Edi n'a été enregistrée pendant les séances de INPV que chez 7 patients seulement. Les mêmes mesures à part l'Edi, ont été réalisées également chez 8 patients contrôle pairés soumis au hasard à une physiothérapie conventionelle. Pendant INPV, l'activité Edi est diminuée, tout au moins temporairement, jusqu'à 50% des valeurs basales. La comparaison des valeurs basales avec celles faisant suite à INPV, n'a pas montré de modification de CPT, VEMS, VEMS/CVF,

Pao_2 et MEP; par contre, des améliorations significatives ont été observées pour MIP, CV, VAS, et 12 mwd, uniquement chez les patients soumis à INPV. Une amélioration significative de la $Paco_2$ a été observée dans les deux groupes de patients. Nous concluons que INPV peut être efficace pour améliorer la réserve fonctionnelle des muscles inspiratoires chez des patients BPCO sélectionnés, ayant une insuffisance respiratoire hypercapnique et des signes de dysfonction musculaire inspiratoire.
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