Four-week Negative Pressure Ventilation Improves Respiratory Function in Severe Hypercapnic COPD Patients*

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Studies on respiratory muscle resting by negative pressure ventilation (NPV) in patients with stable COPD have given conflicting results. Probable explanations lie in criteria of patients’ selection, method of NPV application, and lack of supervision of respiratory muscle rest. Thirteen hypercapnic patients with COPD were, therefore, randomly assigned to either a NPV group or a control group. The NPV was applied by an airtight jacket (pneumosuit), 5 h a day, 5 consecutive days a week for 4 weeks. Both NPV group and control group performed in-hospital pulmonary rehabilitation program for a 4-week period. Arterial blood gases, spirometry, maximal inspiratory pressure (MIP) and maximal expiratory pressure (MEP), breathing pattern, and electromyogram (EMG) of the diaphragm and parasternal intercostal muscles were measured on the preintervention day and, at the end of the second and fourth weeks of treatment (days 13 and 27, respectively). The short-term effect of NPV on EMG suppression was also checked throughout the ventilator sessions in three different days (1, 12, and 26, respectively). A 6-min walking test (WT) and level of dyspnea by a modified Borg scale were evaluated on preintervention and the last day. Negative pressure ventilation resulted in a significant reduction in EMG activity of both diaphragm and parasternal muscles, associated with significant increase in MIP, tidal volume, and ventilation, and increase in PaO$_2$ and decrease in PaCO$_2$. A significant relationship between change in MIP and change in PaCO$_2$ was observed ($r = 0.72$, $p < 0.01$). Improvement in 6-min WT and dyspnea sensation was also observed, both being the sole changes in the control group. These data seem to indicate a beneficial role of respiratory muscle rest in improving respiratory function. Adequate supervision by personnel familiar with the equipment is likely to contribute to successful treatment. (Chest 1994; 105:87-94)

**EMG** = electromyographic activity; **EMGd =** EMG from diaphragm muscles; **EMGint =** EMG from intercostal muscles; **MEP =** maximal expiratory pressure; **MIP =** maximal inspiratory pressure; **NPV =** negative pressure ventilation; **P$_{a}$ =** pressure per breath; **RV =** residual volume; **VC =** vital capacity. **WT =** walking test.

Recent studies have given conflicting results as to whether negative pressure ventilation (NPV) is helpful in resting the respiratory muscles in patients with stable severe COPD. Based on these discrepancies, the need for respiratory muscle resting by NPV in these patients has been recently questioned. Possible explanations for the different outcomes of NPV lie in the criteria for selection of patients, method and application of NPV, and acceptance and commitment of the patient to the procedure. In addition, in many articles, no evidence was given that respiratory muscles were actually being rested during either short-term or long-term studies.

In the present article, we report the results of a 4-week-period of NPV treatment on patients with chronic hypercapnic COPD in whom the effect on electromyographic (EMG) activity of the inspiratory muscles was adequately monitored. The results indicate that respiratory muscle rest, as assessed in terms of suppression in EMG activity, is associated with improvement in muscle strength, breathing pattern, and arterial blood gas values.

**Materials and Methods**

**Patients**

The study was performed on 13 patients (mean ± SD age, 63.7 ± 5.7 years) with chronic obstructive pulmonary disease (COPD), according to the American Thoracic Society criteria. All patients were hypercapnic (mean ± SD PaCO$_2$ = 60.6 ± 5.9 mm Hg) and had severe airflow obstruction. They were all free of active cardiovascular diseases. At the time of the study, all patients were in a clinically stable state. Informed consent was obtained from each subject.

**Functional Evaluation**

Routine spirometry obtained with subjects in a seated position and arterial blood gas values were measured as previously described. The normal values for lung volumes are those proposed by the European Community for Coal and Steel. Maximal static inspiratory (MIP) and expiratory (MEP) pressures at FRC and total lung capacity (TLC), respectively, were measured using a differential pressure transducer (Statham SC 1001; Hato Rey, PR). The subject comfortably seated, wearing a noseclip, performed maximal inspiratory and expiratory efforts near FRC and TLC, respectively, against an obstructed mouthpiece, with a small leak (internal diameter, 0.6 mm) to minimize oral pressure artifacts. The maneuvers were repeated until three measurements sustained for at least 1 s with less than 5 percent variability were recorded. The highest value obtained was used for analysis.

After baseline routine testing during room-air breathing, the
ventilatory pattern, respiratory drive, and mouth occlusion pressure were evaluated with subjects put in a comfortable supine position. In the apparatus we used, the inspiratory line was separated from the expiratory one by a one-way valve (Hans-Rudolph, Kansas City, Mo) connected to a pneumotachograph (Fleisch type 3). The flow signal was integrated into volume. From the spirogram we derived the following: inspiratory time (Ti), expiratory time (Te), total time of the respiratory cycle (Ttot), and tidal volume (VT). Mean inspiratory flow (VT/Ti), duty cycle (Ti/Ttot), respiratory frequency (f = 1/Ttot × 60), and instantaneous ventilation (VE = VT × f) were also calculated.

The EMG activity of the respiratory muscles was recorded as previously described.10-13 The EMG of the chest wall muscles was recorded from the second parasternal intercostal (EMGint) and diaphragm (EMGd) muscles via large surface electrodes. The EMGd was recorded from the lower anterolateral rib cage as described by Gross et al.14

Muscle action potentials ("raw") were differentially amplified, filtered between 100 and 1000 Hz, to remove as much ECG as possible, without significantly filtering EMG. Both the filtered EMG signal and mouth pressure recording were displayed on a single-beam storage oscilloscope (Tektronix 5115, Tektronix Inc., Beaverton, Ore). Electromyographic activity was full-wave rectified and integrated over time (time constant, 100 ms) using a third-order, low-pass filter to provide a measurement of change in average electrical activity as a function of time, referred to as "moving time average" (XMTA). Inspiratory activity was quantified both as peak of activity and as rate of rise of activity (slope). The former was directly measured in arbitrary units (X) and the latter was obtained by dividing X by the inspiratory time (X/TPi).

Owing to the variability of the impedance between diaphragm and electrodes, absolute values (nV) are not comparable among different subjects. To overcome this problem and to obtain a reference value, EMG activity was measured while the subject, connected to the pneumotachograph, performed an inspiratory capacity (IC) maneuver breathing in up to the TLC.15 This maneuver was repeated at least three times and in each subject both IC and the intensity of the recorded diaphragmatic EMG were closely reproducible (less than 5 percent variability). The mean level of this EMG activity was taken as a reference; all successive measurements have been expressed as a percentage of this reference value obtained at TLC. As EMG activity of an inspiratory muscle may include cardiac muscle activity, we checked cardiac artifacts to manually gate ECG, when necessary, so that it would not contribute to the EMG.

The output of CO2 meter, the flow signal, the integrated flow signal, the pressure signal, and the moving time average were recorded continuously on a multichannel chart recorder.

Negative pressure ventilation was provided by a pressure-cycled pump (Emerson model 33CBE; J.H. Emerson Co, Cambridge) connected to an airtight jacket (pneumosuit). The delivered negative pressure was checked by a lead placed into the jacket and connected to a pressure transducer (Statham P23 1D).

A 6-minute walking test (WT)16 was performed after each patient had been instructed to cover as much ground as he could on foot in 6 min, walking along a hospital corridor. Verbal encouragement was given continuously during the test, but patients were free to stop when they wished.

Assessment of dyspnea level was performed by means of a modified Borg scale;17 that ranged from zero to ten and included verbal descriptors.

**Data Analysis**

Results were compared by Mann Whitney U test for unpaired samples and Wilcoxon test for paired samples. The significance of the change induced by NPV was assessed by two-way analysis of variance and Friedman test. A p value < 0.05 was considered to be significant.

**Protocol**

Patients were randomly assigned to either NPV group (six patients) or control group (seven patients). The NPV group underwent a rehabilitation program plus a 4-week period of NPV 5 h a day divided into two runs of 150 min each, on 5 consecutive days a week; the control group underwent 4 weeks of in-hospital rehabilitation program. The rehabilitation program consisted of a morning period of coordination exercises, treadmill and floor walking, and an afternoon period of unsupported arm exercises.

**Preintervention Day.** On a preintervention day, each NPV group patient was put into the pneumosuit, with the pump turned off. After a 10-min adaptation period, baseline evaluation began. Respiratory cycles and EMG were continuously recorded over a 15-min time period. To assess the necessary negative pressure for inducing the maximum EMG suppression, after baseline assessment EMG activity was replicated during intermittent NPV with the patient connected to a mouthpiece. The ventilator was turned on and its frequency, Ti and Tr, were adjusted so as to closely approximate to the subject's spontaneous timing components. A first step was performed at −10 cm H2O; then at −20, −30, and −35 cm H2O. A greater negative pressure (−40 cm H2O) was applied but it was considered not to be useful since it was associated with a lower EMG activity suppression (increase in EMG) and

**Table 1—Pulmonary Function Data, Maximal Respiratory Pressures, 6-min WT, and Dyspnea Score in NPV Group and Control Group on Preintervention Day (C) and Day 27 of Treatment (27)**

<table>
<thead>
<tr>
<th></th>
<th>Age, yr</th>
<th>VC, % pred</th>
<th>FEV1, % pred</th>
<th>FEV1/VC, %</th>
<th>FRC, % pred</th>
<th>RV, % pred</th>
<th>MIP, cm H2O</th>
<th>MEP, cm H2O</th>
<th>PaO2, mm Hg</th>
<th>PaCO2, mm Hg</th>
<th>6-min WT, m</th>
<th>Dyspnea Score</th>
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<td>NPV group</td>
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<tr>
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<td>50.5</td>
<td>23.3</td>
<td>38</td>
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<td>173.8</td>
<td>42.2†</td>
<td>111</td>
<td>51†</td>
<td>62†</td>
<td>335†</td>
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<td></td>
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<td>(13.0)</td>
<td>(35.5)</td>
<td>(6.0)</td>
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<td>27</td>
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<td>124.8</td>
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<td>(5.3)</td>
<td>(56.6)</td>
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<tr>
<td>C</td>
<td>64.0</td>
<td>53.0</td>
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<td>150.1</td>
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<td>(13.2)</td>
<td>(14.1)</td>
<td>(28.0)</td>
<td>(7.1)</td>
<td>(4.5)</td>
<td>(106.6)</td>
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<td>151.2</td>
<td>169.2</td>
<td>45.3</td>
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<td>451.2</td>
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<td>(4.3)</td>
<td>(4.5)</td>
<td>(12.9)</td>
<td>(16.2)</td>
<td>(10.4)</td>
<td>(28.9)</td>
<td>(5.4)</td>
<td>(5.1)</td>
<td>(69.2)</td>
<td>(1.5)</td>
<td></td>
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</tbody>
</table>

*Values are mean ± SD.
†NPV group (27 vs C); p<0.05.
‡p<0.02
§p<0.01.
|Control group (27 vs C); p<0.05.

NPV Improves Respiratory Function in COPD Patients (Gigliotti et al)
Table 2—Breathing Pattern and Electromyographic Activity of the Diaphragm and Intercostal Muscles in NPV Group and in Control Group on the Preintervention Day (C) and on the Day 13 and 27 of Treatment

<table>
<thead>
<tr>
<th>NPV Group</th>
<th></th>
<th>Control Group</th>
<th></th>
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<tbody>
<tr>
<td></td>
<td>Day 13</td>
<td>Day 27</td>
<td></td>
</tr>
<tr>
<td>Vt, L</td>
<td>0.49±0.16</td>
<td>0.52±0.08</td>
<td>0.60±0.15</td>
</tr>
<tr>
<td>Ti, s</td>
<td>1.08±0.29</td>
<td>1.11±0.14</td>
<td>1.13±0.15</td>
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<tr>
<td>Te, s</td>
<td>2.01±0.53</td>
<td>1.96±0.43</td>
<td>1.99±0.65</td>
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<tr>
<td>Tt/Tot, s</td>
<td>3.06±0.80</td>
<td>3.07±0.54</td>
<td>3.12±0.71</td>
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<tr>
<td>Vt/Ti, L/s</td>
<td>0.45±0.04</td>
<td>0.47±0.04</td>
<td>0.52±0.09</td>
</tr>
<tr>
<td>Ti/Ttot</td>
<td>0.35±0.03</td>
<td>0.36±0.04</td>
<td>0.37±0.06</td>
</tr>
<tr>
<td>Ve, L/min</td>
<td>9.40±0.51</td>
<td>10.23±0.90</td>
<td>10.90±0.68</td>
</tr>
<tr>
<td>l, breaths/min</td>
<td>20.50±3.30</td>
<td>20.18±4.30</td>
<td>18.40±4.30</td>
</tr>
<tr>
<td>EMGd, % TLCs</td>
<td>33.10±7.50</td>
<td>25.10±3.10</td>
<td>21.10±4.90</td>
</tr>
<tr>
<td>EMGint, % TLCs</td>
<td>35.70±12.3</td>
<td>27.60±5.27</td>
<td>21.08±6.20</td>
</tr>
</tbody>
</table>

*Values are mean ± SD. Vt = tidal volume; Ti = inspiratory time; Te = expiratory time; Tt/Ttot = total time of respiratory cycle; Vt/Ti = minute ventilation; f = respiratory frequency; EMGd = electromyographic activity of diaphragm; EMGint = electromyographic activity of intercostal muscles; NPV = negative pressure ventilation.

Sometimes mild obstructive apnea episodes. With negative pressure limited to -35 cm H₂O, no patients complained about discomfort over the duration of the run. Patients were initially requested to breathe synchronously with the ventilator, then to relax. The best negative pressure was selected on the basis of the maximal produced EMG suppression and VT (just greater than the control value), then it was applied in the scheduled NPV sessions. In all patients, applied pressure ranged between -25 and -35 cm H₂O.

Study Period: Electromyographic activity was evaluated either before (prerun, days 1, 13, 27) or during (run-in, days 1, 12, 26) NPV session. The run-in EMG suppression was assessed as follows: for each session, after an initial 15-min adaptation period, a period of 5 min every 15 min was considered, that is, 20 min per hour were analyzed; the relevant averaged peak EMG activity was expressed as percentage of the prerun average peak activity.

In the preintervention day and at the end of the second and fourth week of treatment (days 13 and 27, respectively) patients underwent spirometry, arterial blood gas determinations, breathing pattern, and MEP and MEP. Also, 6-min WT and level of dyspnea were evaluated on the preintervention day and day 27 of treatment.

RESULTS

Functional data for the two groups of patients are summarized in Table 1. Both NPV and control groups exhibited severe airflow obstruction (FEV₁/VC), hyperinflation (FRC), a marked reduction in MIP, and a moderate decrease in MEP, as compared with our laboratory reference values; all patients were hypercapnic (62.2 ± 6 mm Hg and 59.8 ± 4.5 mm Hg, for NPV and control groups, respectively) and hypoxemic (51.5 ± 5.5 mm Hg and 53.5 ± 7.1 mm Hg, for NPV and control groups, respectively). No difference in terms of spirometric parameters was observed between the two groups. Dyspnea score and 6-min WT were also similar in them.

Breathing characteristics and EMG activity of the diaphragm (EMGd) and parasternal intercostal mus-
icles (EMGint) for NPV and control groups on the preintervention day are shown in Table 2, the differences between the groups being not significant.

At the cessation of the study period, the NPV group exhibited a significant increase in MIP (p < 0.05), PaO₂ (p < 0.02), 6-min WT (p < 0.05), and significant reduction in PaCO₂ (p < 0.05) and dyspnea score (p < 0.01) (Table 1 and Figs 1 and 2). Significant changes in 6-min WT and dyspnea score (p < 0.05 for both) were the sole changes noticed in the control group.

Changes in breathing pattern on second and fourth week (days 13 and 27, respectively) in the NPV group are summarized in Table 2 and Figure 3; VT (p = 0.02), VE (p = 0.0049), and VT/TI (p = 0.025) significantly increased. Prerun EMG activity (p = 0.0001) and EMGint (p = 0.0025) decreased throughout the study period (Table 2).

The short-term effect of NPV on EMG suppression (run-in effect) is shown in Figure 4: on day 1, NPV produced a marked suppression in EMGd and a slighter one in EMGint (mean ± SD residual activity, 45.3 ± 6.2 percent, p < 0.001, and 74.8 ± 22.3 percent, p < 0.05, respectively); on day 12, a further reduction was observed in EMGint (52.8 ± 19 percent, p < 0.005) while EMGd mean residual activity (38 ± 6.3 percent, p < 0.001) did not significantly differ from that of day 1. On day 26, no substantial changes were observed in mean (± SD) residual values for EMGd (38 ± 6.3 percent, p < 0.001) and EMGint (47.2 ± 7.2 percent, p < 0.005); in fact, while four of the six patients exhibited a further EMGd suppression, in the remaining two patients (patients 2 and 3), EMG activity was found to increase. In turn, the amount of EMGd suppression recorded in each of the three study days did not statistically differ from that recorded in the remaining two (Friedman test). Conversely, EMGint suppression recorded on days 12 and 26 was significantly greater than that recorded on the day 1 (F = 6.21, p < 0.05).

Figure 5 depicts the plot between PaCO₂ decrease and MIP increase, as measured at the end of second and fourth week of NPV treatment. A direct linear relationship was found (r = 0.73, p < 0.01), the changes in MIP predicting 52 percent of the variance of the changes in PaCO₂.

In the control group, the rehabilitation program did not result in any changes in terms of the above variables (Table 2).

**Discussion**

In this study, we provide evidence that in patients with stable COPD with chronic hypercapnia, NPV is able to produce amelioration in respiratory muscle strength, breathing pattern, arterial blood gases, along with a consistent suppression in EMG activity of the diaphragm and the parasternal intercostal muscles. These results are consistent with some reports, but at variance with others.

Differences between our and other studies showing no benefit from NVP could depend on two major reasons. Criteria for selection of the patients is the first reason. We studied hypercapnic COPD patients while other observations mostly dealt with normocapnic COPD. The observation of a significant improvement in the patient with the highest PaCO₂ and a review of the literature suggested to Celli et al that there may be a subset of patients with COPD who benefit from NPV, and one discriminating factor might be the presence of severe hypercapnia. Adequate ventilator use and supervision is the second reason. In the present study, patients were kept in
hospital throughout the period of treatment and under the control of personnel accustomed to the use of negative pressure ventilators and the techniques employed. In contrast, other studies\(^1,5,5\) did not provide either precise information about supervision or evidence of respiratory muscle resting, while in some other studies\(^7,10\) supervision was not scheduled.

**Respiratory Function**

The observed increase in MIP occurred without any changes in mechanical loading (FEV\(_1\), FRC). Recent observations on motivational central voluntary fatigue\(^{26}\) may support the hypothesis that a generally improved well-being with NPV may improve the ability to voluntarily generate maximal inspiratory effort. The possibility of a learning effect does not have to be excluded either, even if the same ability to increase MIP was not observed in the control group, which exhibited, however, similar improvement in well-being and dyspnea. The improvement in arterial blood gases is somewhat consistent with other studies\(^1,5,9,12,23,25,27,28\) showing a decrease in PaCO\(_2\) and an increase in PaO\(_2\) after either short-term\(^5,28\) or long-term\(^5,22,23,27\) NPV treatment in patients with COPD. Fernandez et al\(^{28}\) have recently observed that NPV treatment produced an increase in mean alveolar ventilation and a fall in Vd/Vt ratio. These results suggested to the authors the possibility that NPV could produce a redistribution in pulmonary blood flow, thereby improving arterial blood gas values. Chronic CO\(_2\) retention appears to be associated with shallow breathing, *ie*, low VT.\(^{29}\) Considering that even

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**Figure 3.** Individual changes in Vt and Ve with NPV.

**Figure 4.** Suppression of the electrical activity of the diaphragm (EMGd, left panel) and parasternal intercostal muscles (EMGint, right panel) during NPV session (run-in) on day 1, 12, and 26 of treatment. The EMG values are expressed as percentage of the prerun value. Individual data are shown.
a slight increase in $V_t$ increases alveolar ventilation enough to produce a reduction in $P_{\text{aCO}_2}$, it is not surprising that the observed decrease in $P_{\text{aCO}_2}$ was associated with the increase in $V_t$ (Figs 2 and 3). On the other hand, the possibility has also been raised that short-term ventilatory treatment decreases oxygen consumption and $CO_2$ production and modifies the respiratory center responsiveness to hypoxemia and hypercapnia. Nevertheless, the significant relationship between reduction in $P_{\text{aCO}_2}$ and increase in MIP (Fig 5), which is in general agreement with that observed in patients with COPD, seems to indicate the role of NPV-induced amelioration in inspiratory muscle strength on improving arterial blood gas values. At variance, the possibility that an improvement in arterial blood gas values may have increased muscle strength should not be excluded, consistent with the observation that acute hypoxemia and/or hypercapnia affect ventilatory muscle performance. Nevertheless, the effects of chronic hypercapnia and/or hypoxemia on respiratory muscle performance are not known; hence, any inference on the role of arterial blood gas amelioration on respiratory muscle improvement is a matter of speculation.

The relationship we found between MIP and $P_{\text{aCO}_2}$ extends the results of Cropp and Di Marco, Ambrosino et al, and Marino and Braun, who demonstrated that resting respiratory muscles may improve their force and arterial blood gas levels; those changes, however, were not related to each other.

Based on a hypothesis put forward recently by Rochester, the association we found among improvement in muscle strength, breathing pattern, and arterial blood gas values could be explained in terms of an integrated central response: muscle weakness and increase in pulmonary resistance decrease MIP and increase the pressure per breath ($P_{\text{P}}$), respectively, thereby increasing $P_{\text{P}}$/MIP ratio. Increase in $P_{\text{P}}$/MIP ratio is the trigger for the integrated response of the respiratory system which results in a reduction in $P_{\text{aCO}_2}$ and a lower $V_t$, thereby increasing $P_{\text{aCO}_2}$. Accordingly, increase in muscle force with NPV could be a reason for the associated improvement in breathing pattern and arterial blood gas values.

**EMG Activity**

**Short-term Effects (Run-In EMG Suppression):** Rochester et al showed that a short session of NPV by a tank could produce suppression of diaphragmatic EMG activity in patients with COPD, a datum consistent with our and other recent reports. Similarly, in a study by Roderstein et al, 1-h NPV, but not a shorter period, resulted in a substantial EMGd suppression. A behavioral response to NPV treatment may account for magnitude and rapidity in EMGd suppression. Nevertheless, the observation that in the present study the maximal reduction in EMG activity of inspiratory muscles occurred at relatively high values of negative pressure delivered (−25 to −35 cm H$_2$O) could indicate that an adequate value of negative pressure is necessary to relieve inspiratory muscle workload. In addition, the observation that the suppression in EMG activity recorded on day 12 was greater than that recorded on day 1 might suggest a learning effect. However, NPV was able to produce a consistent suppression in EMG activity even on day 1, when patients were not accustomed to this type of ventilatory treatment. Also, the observation that in patients 2 and 3 the maximal EMGd suppression was recorded on day 12, while the rate of EMGd suppression on day 26 went back to the level of day 1, seems to indicate that a learning effect, important as it was, did not play a crucial role in EMG suppression.

**Prerun EMG Decrease: In the present study,** prerun EMG activity progressively decreased throughout the study period and it was associated with increase in MIP and amelioration in arterial blood gas values (Table 2). These findings confirm previous data of ours obtained after a 7-day period with iron lung. Theoretically, the meaning of this association lies in the role that both chemical afferents and mechanical afferents arising from lung and/or chest wall are thought to play on the neural respiratory drive (EMG). Suppression of either chemical and neurochemical reflexes has been proven to decrease EMG activity during sleep, while during wakefulness behavioral effects are likely to substantially contribute to EMG inhibition. The present data, however, do not permit us to

![Graph](http://journal.publications.chestnet.org/pdaccess.ashx?url=/data/journals/chest/21688/)
establish the precise reason for the progressive reduction in EMG activity we noticed.

Conclusions

After treatment, both groups of patients showed a reduction of dyspnea and increase in 6-min WT. It is a common finding that despite the lack of significant changes in pulmonary function and hemodynamics, rehabilitation programs produce an increase in exercise tolerance. The latter is attributed to a learning effect, increased motivation, improved mechanical skills, and desensitization to dyspnea.

In summary, our data indicate that application of NPV for 5 h a day on 5 consecutive days weekly for 4 weeks may result in an improvement in inspiratory muscle strength, breathing pattern, arterial blood gas values, and decrease in EMG activity of the respiratory muscles. Based on all these findings, we believe that (1) increase in muscle strength is likely to play a crucial role in improving ventilation and arterial blood gas values, and (2) patients may achieve a progressive reduction in EMG activity over a 4-week NPV rehabilitation program. Respiratory muscle "deactivation," however, should be regularly controlled in order to ensure that an adequate resting level is achieved.

References


