Suppression of Ventilatory Muscle Activity in Healthy Subjects and COPD Patients with Negative Pressure Ventilation*

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We evaluated the ability of NPV to suppress EMGd and EMGint in seven patients with severe COPD and five normal subjects. Subjects were studied either without (A) or with mouthpiece and nose clip (B). Electromyographic suppression was assessed comparing EMG activity during NPV with the control activity without a mouthpiece and prior to the initiation of the NPV run. In normal subjects, in A, NPV resulted in a partial suppression of EMGd in B, prior to NPV, EMGd rose compared with A prior to NPV. In patients, in A, NPV resulted in a suppression of both EMGd and EMGint. In B, prior to NPV, both EMGd and EMGint rose compared with A prior to NPV. Thus, it seems that NPV is able to produce a consistent reduction in inspiratory muscle EMG activity. This variable NPV ability would have to be assessed for better selection criteria for patient candidates in a rehabilitation program. (Chest 1991; 99:1186-92)

The contribution of respiratory muscle fatigue to ARF has been established in recent years.1 In patients with COPD, the relationship between respiratory muscle fatigue and CRF probably is less clear. It has been postulated that in CRF, fatigued respiratory muscles may benefit from resting by NPV.2-10 In patients with COPD, recent and past studies support this hypothesis2-3,6,8,10 even if some observations do not.11,12 For NPV to be effective in resting respiratory muscles, consistent suppression of phasic EMGd is warranted.14 Nonetheless, in most of the aforementioned studies, EMG activity was not evaluated; thus, respiratory muscle resting was not proved. On the other hand, the few studies aimed at providing evidence of respiratory muscle resting with NPV gave conflicting results. In the two studies of Rodenstein et al.,13,15 neither healthy subjects nor naive patients with severe COPD exhibited an immediate consistent decrease in EMGd with NPV; conversely, an immediate reduction in EMGd was observed by Rochester et al.2 in normal subjects during resistive breathing and in trained patients as well.

Contributing to this field, the present investigation was undertaken to evaluate the ability of NPV to suppress ventilatory muscle activity both in normal man and in patients with COPD.

MATERIALS AND METHODS

We studied five normal subjects and seven patients suffering from COPD, according to the ATS criteria.16 All patients were free of active cardiovascular disease and were in a clinically stable state.

Routine spirometry, and arterial blood gas value analysis obtained with subjects in a seated position were measured as previously described.17 The normal values for lung volumes are those proposed by the European Community for Coal and Steel.18

After baseline routine testing, during room-air breathing the ventilatory pattern was evaluated with subjects put in a comfortable supine position. Each subject breathed through a Fleisch type 3 pneumotachograph. The flow signal was integrated into volume. From the spirometer we derived, breath by breath, time and volume components of the respiratory cycle: Ti, Te, Ttot, and Vt. Respiratory frequency (RF = 1/Ttot)60) and instantaneous ventilation (Vt = Vt/RF) also were calculated.

The EMG of the respiratory muscles was recorded as previously described.19,20 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.21

Muscle action potentials ("raw") were differentially amplified, filtered between 100 and 1,000 Hz, to remove as much ECG as possible, without significantly filtering EMG. The filtered EMG signal was displayed on a single-beam storage oscilloscope (Tektronix 5115). The EMG 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
Table 1—Baseline Functional Data in the Two Groups of Subjects*

<table>
<thead>
<tr>
<th></th>
<th>Age (yr)</th>
<th>VC†</th>
<th>RV†</th>
<th>FRC†</th>
<th>TLC†</th>
<th>FEV1†</th>
<th>PaO2 (mm Hg)</th>
<th>PaCO2 (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>COPD patients</td>
<td>(3.6)</td>
<td>(3.8)</td>
<td>(14.7)</td>
<td>(8.17)</td>
<td>(4.5)</td>
<td>(4.7)</td>
<td>62.8</td>
<td>48</td>
</tr>
<tr>
<td>Normal subjects</td>
<td>(3.6)</td>
<td>(10.3)</td>
<td>(2.7)</td>
<td>(4.5)</td>
<td>(2.7)</td>
<td>(12.1)</td>
<td>35.2</td>
<td>48</td>
</tr>
</tbody>
</table>

*Values are means ± 1 SE.
†Percent of the predicted value.

function of time, referred to as "moving time average."20,21 This method of analysis allows the description of the time course of inspiratory muscle activity which shows a definable rate of increase, reaching a peak of amplitude and then rapidly decreasing. Inspiratory activity was quantified as peak of activity and directly measured in arbitrary units. All EMG activity measurements performed during NPV were expressed as percentage of the control value.

In the normal subjects and in two COPD patients, EMGd simultaneously was recorded by means of a bipolar esophageal electrode (Disa 13 K 63) passed through the nose. The esophageal electrode was positioned to obtain an optimal and reproducible signal to noise ratio at VT and maximal inspiration and then fixed at the nose with tape. In the normal subjects, we also looked at esophageal EMGd activity during expiration (PHI). The PHI was measured as percentage of the duration of Te, according to the method of Rodenstein et al.11

End tidal CO2 was recorded by means of an infrared capnograph (Datex) and the SaO2 by an ear oximeter (Oxi, Radiometer, Copenangen). Measurements were performed under control conditions and during intermittent NPV provided by a pressure cycled pump connected with an airtight jacket ("poncho"). The delivered negative pressure was checked by a lead placed in the jacket and connected with a pressure transducer (Statham P23ID).

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

The protocol was as follows. To start with, each subject performed the test without wearing a mouthpiece (A), and EMGd, EMGint and SaO2 were recorded; ten minutes of quiet breathing were recorded and the last 5 min were analyzed and used as a control. Initially, the ventilator's f, and Ti and Te were adjusted to closely approximate to the subject's spontaneous timing components. The first step was performed at −10 cm H2O; then at −20 and −30 cm H2O. A greater negative pressure (−40 cm H2O) was also applied but it was considered not to be useful since it was associated with decrease in EMGd activity suppression (increase in EMC), clinical symptoms of hyperventilation and sometimes mild obstructive apnea episodes.

By limiting the negative pressure to −30 cm H2O, no patient complained about discomfort over the duration of the run (30 min).

With increasing values of delivered negative pressure, Ti and Te slightly lengthened, so that the final f slightly decreased. Subjects were initially requested to breathe synchronously with the ventilator, then to relax. At the end of each negative pressure step, the ventilator was turned off and the following 5-min period of quiet breathing allowed PetCO2 and SaO2 to return to the control values. Each pressure step lasted at least 10 min and the last 5 min were analyzed.

In order to record the time and volume components of the respiratory cycle, and PetCO2, after a 30-min rest period the same sequence was performed while subjects were wearing a mouthpiece and nose clip (B).

Results were compared by the Wilcoxon test for the patient group and by the paired Student t test for the normal group. A p value <0.05 was considered to be significant.

RESULTS

Functional data for patients and normal subjects are summarized in Table 1. Patients exhibited moderate to severe airflow obstruction (percent predicted mean FEV1 = 35.2 percent ± 4.7 SE), and hyperinflation (percent predicted mean FRC = 157 percent ± 8.17 SE); mean PaCO2 was 48 mm Hg ± 3.85 SE and mean PaO2 62.8 mm Hg ± 2.8 SE. Patients 2, 3 and 4 had chronic hypercapnia (PaCO2 = 51.6 to 62 mm Hg) and moderate hypoxemia (PaO2 = 54.5 to 58 mm Hg).

Breathing characteristics (Ve, Vt, RF) and PetCO2 for both normal subjects and patients, and SaO2 (in patients) under control conditions and during NPV (−10, −20, −30 cm H2O) are shown in Tables 2 and 3, respectively. In normal subjects (Table 2) at −20

Table 2—Simultaneous Measurements of Breathing Pattern, PetCO2 and EMGd Activity under Control Conditions and during NPV in 5 Normal Subjects*

<table>
<thead>
<tr>
<th></th>
<th>(\dot{V}_{E}) (L/min)</th>
<th>Vt (L)</th>
<th>RF (cycles s)</th>
<th>PetCO2 (mm Hg)</th>
<th>EMGd (AU)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>9.78</td>
<td>0.81†</td>
<td>11.96†</td>
<td>43†</td>
<td>4.82†</td>
</tr>
<tr>
<td></td>
<td>(0.98)</td>
<td>(0.06)</td>
<td>(0.36)</td>
<td>(1.03)</td>
<td>(1.4)</td>
</tr>
<tr>
<td>−10 cm H2O</td>
<td>9.2</td>
<td>0.72</td>
<td>12.7</td>
<td>40.2</td>
<td>2.6</td>
</tr>
<tr>
<td></td>
<td>(1.3)</td>
<td>(0.09)</td>
<td>(0.3)</td>
<td>(2.2)</td>
<td>(1.07)</td>
</tr>
<tr>
<td>−20 cm H2O</td>
<td>9.9</td>
<td>0.79</td>
<td>12.2</td>
<td>38†</td>
<td>1.54†</td>
</tr>
<tr>
<td></td>
<td>(1.43)</td>
<td>(0.06)</td>
<td>(0.89)</td>
<td>(1.9)</td>
<td>(0.84†)</td>
</tr>
<tr>
<td>−30 cm H2O</td>
<td>11.8</td>
<td>1.13†</td>
<td>10.5†</td>
<td>35.1†</td>
<td>0.992†</td>
</tr>
<tr>
<td></td>
<td>(1.2)</td>
<td>(0.11)</td>
<td>(0.16)</td>
<td>(2.0)</td>
<td>(0.50)</td>
</tr>
</tbody>
</table>

*Values are means ± 1 SE.
†p<0.05.
‡p<0.025.
Table 3—Simultaneous Measurements of Breathing Pattern, PetCO₂, HbSaO₂ and EMGd Activity under Control Conditions and during NPV in 6 Out of 7 Patients with COPD*

<table>
<thead>
<tr>
<th></th>
<th>VE (L/min)</th>
<th>VT (L)</th>
<th>RF (cycles s⁻¹)</th>
<th>PErCO₂ (mm Hg)</th>
<th>HbSaO₂ (%)</th>
<th>EMGd (AU)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>11.0</td>
<td>0.69†</td>
<td>15.9</td>
<td>46†</td>
<td>90.5†</td>
<td>10.3†</td>
</tr>
<tr>
<td>-10 cm H₂O</td>
<td>11.8</td>
<td>0.78</td>
<td>(1.5)</td>
<td>(3.2)</td>
<td>(2.0)</td>
<td>(3.02)</td>
</tr>
<tr>
<td>-20 cm H₂O</td>
<td>11.0</td>
<td>0.78</td>
<td>(1.3)</td>
<td>(3.6)</td>
<td>(1.0)</td>
<td>(1.4)</td>
</tr>
<tr>
<td>-30 cm H₂O</td>
<td>11.4</td>
<td>0.82†</td>
<td>(1.1)</td>
<td>(3.6)</td>
<td>(0.3)</td>
<td>(1.22)</td>
</tr>
</tbody>
</table>

*Values are means ± 1 SE.
†p<0.05.

cm H₂O negative pressure, a slight but significant (p<0.05) reduction in PetCO₂ was observed; at -30 cm H₂O, a greater decrease in PetCO₂, a significant increase in VT (140 percent from control) and a decrease in RF (p<0.05 for all comparisons), with a tendency for VE to increase, resulted.

In six out of the seven patients (Table 3), NPV at -10 and -20 cm H₂O pressure did not result in any significant change in VE, VT, PetCO₂ and SaO₂, while NPV at -30 cm H₂O caused VT and SaO₂ to significantly increase and PetCO₂ to decrease (p<0.05 for all comparisons). Patient 4 could not wear the mouthpiece enough to allow baseline tracings to be recorded.

In the two groups, wearing the mouthpiece resulted in a clear-cut increase in EMGd and EMGint activities, compared with the activity recorded without the mouthpiece (normal subjects, 200 percent ± 28.6 SE for EMGd; COPD patients, 165 percent ± 18.8 SE and 196 percent ± 27.4 SE for EMGd and EMGint, respectively). Figure 1 shows, in a representative case (patient 1), the time course of EMGd and EMGint without (A) and with (B) the mouthpiece during NPV. It is evident from the figure, that NPV (-10 and -20 cm H₂O) not only suppressed the EMG increase produced by the use of the mouthpiece (CB), but it even (-30 cm H₂O) caused a further ≥50 percent EMGd and EMGint decrease of the value recorded without the mouthpiece (CA).

In the normal subjects, suppression of EMGd activity is depicted in Table 2 and Figure 2. In particular, Figure 2 shows the time course of EMGd with NPV in individual subjects. In each of them, NPV resulted in a substantial suppression of electrical activity of the diaphragm at -30 cm H₂O, either without (A, [left panel]) or with (B, [right panel]) the mouthpiece. The mean residual EMGd activity at -30 cm H₂O was 26.8 percent ± 18.3 SE in A, and 20.6 percent ± 9.3 SE in B. In particular, at -10 and -20 cm H₂O, subject 5 showed a poor adaptation to NPV, but a complete suppression of EMGd activity was observed at -30 cm H₂O. In normal subjects, we did not record EMGint activity during NPV since the signal under control conditions was too low to permit any reliable measurement.

In general, in patients, a progressive suppression in EMGd activity was found when the negative pressure delivered was increased (Table 3). From Figure 3, it is evident that NPV resulted in a consistent percentage of suppression of EMGd activity either while patients were breathing without (A, [left panel]) or with (B, [right panel]) the mouthpiece; the mean residual EMGd activity at -30 cm H₂O was 29.8 percent ± 6.0 SE in A, and in B it was 37 percent ± 11.7 SE. In particular, EMGd reduction was slight in patient 6, more evident in patients 3, 4 and 7 and marked in patients 1, 2 and 5. Patient 4 does not appear on the right panel of the Figure 4 because of the mentioned reasons. The evolution of EMGint in the patients is shown in Figure 4. In A (left panel), patients 2, 4 and 6 exhibited a slight suppression of the phasic activity of intercostals muscles, while in patients 1, 3, 5 and 7 the suppression was progressively more marked; in the patients as a group, mean residual EMGd activity
Figure 2. From control (C) time course of peak EMGd during NPV in individual normal subjects either without (A, left panel) or with (B, right panel) mouthpiece and nose clip. EMGd is in percentage of pre-NPV value.

at −30 cm H2O was 32.9 percent ± 10.6 SE of the pre-NPV value. In B (right panel), EMGint consistently decreased (mean residual EMGint being 31.3 percent ± 8.2 SE at −30 cm H2O). The suppression in EMGint progressively rose with increasing negative pressure, with the exception of patient 7 who exhibited a more marked suppression at both −10 and −20 cm H2O than he did at −30 cm H2O.

In three out of the seven patients, after the greater negative pressure step and EMGd suppression were reached (from −10 to −30 cm H2O; up run), without informing the patients, pressure was made to progressively increase (−30 to −10 cm H2O; down run); this was associated with a progressive increase in EMGd activity (Table 4).

In two complementary studies we noticed a good agreement between EMGd, and EMGd, during NPV. The EMGd/EMGd, ratio did not substantially change over the runs (Table 5). In addition, we were not able to detect any substantial change in PIIA with NPV.

Figure 3. From control (C) time course of peak EMGd during NPV in individual COPD patients either without (A, left panel) or with (B, right panel) mouthpiece and nose clip. EMGd is in percentage of pre-NPV value.
**Discussion**

Normal Subjects

A significant increase in $V_T$ and reduction in PETCO$_2$ and consistent progressive suppression in EMGd were observed with NPV.

Our data partially agree with a previous study of Rodenstein et al. who observed that in normal subjects there was a consistent increase both in $V_T$ and $V_E$ with a tank ventilator. In the present study, subjects promptly adapted to the ventilator frequency—maintained, however, as similar as possible to their spontaneous frequency—and showed a significant increase in $V_T$, along with a slight increase in $V_E$; this latter was not appreciable in all the subjects and was apparently too small to account for the significant reduction we noticed in PETCO$_2$. This pattern could be explained if one considers that even a slight increase in $V_T$ increases $V_A$ enough to produce a reduction in PETCO$_2$.23

In the study of Rodenstein et al., subjects immediately locked their spontaneous frequency to the ventilator's frequency, and they were requested to relax; however, the amount of EMGd suppression showed a great interindividual variability and EMGd values were, on average, similar to the control values. This great interindividual variability was explained by the different ability of each subject to relax and voluntarily suppress EMGd activity. Rodenstein et al. also observed a significant increase in PIH of the diaphragm, which was thought to be due to the NPV-induced hyperventilation, as it occurs in a passively hyperventilated cat.24 With regard to our results, consistent with other studies,2,25,26 we noticed a suppression in EMGd activity, already marked at $-10$ cm H$_2$O (subjects 1 and 3) and more substantial at $-30$ cm H$_2$O.

Two principal reasons could be involved in our observations: (1) In unconscious man, a reduction in EMGd activity could depend on CO$_2$ reduction caused by the ventilator-induced hyperventilation.23 However, in awake and unanesthetized subjects the effect of decreasing CO$_2$ on the suppression of EMG activity is less evident,2 compared with the magnitude of EMG suppression we observed. In addition, as was the case in the study of Rochester et al.,2 we did not find any significant correlation between PETCO$_2$ reduction and EMG suppression with NPV. Indeed, in some instances, constant values of PETCO$_2$ accompanied substantial suppression in EMG activity. This observation agrees with the data of Collett et al. obtained, however, in different experimental conditions. Unlike Rodenstein et al.,23 we did not observe

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**Table 4—EMGd Suppression in Three out of the Seven Patients with NPV during Either Up Runs or Down Runs**

<table>
<thead>
<tr>
<th>n</th>
<th>Control</th>
<th>$-10$ cm H$_2$O</th>
<th>$-20$ cm H$_2$O</th>
<th>$-30$ cm H$_2$O</th>
<th>$-20$ cm H$_2$O</th>
<th>$-10$ cm H$_2$O</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>100</td>
<td>41.5</td>
<td>25.7</td>
<td>11.5</td>
<td>34.3</td>
<td>50.8</td>
</tr>
<tr>
<td>4</td>
<td>100</td>
<td>80.7</td>
<td>66.6</td>
<td>52.6</td>
<td>49.1</td>
<td>100.0</td>
</tr>
<tr>
<td>7</td>
<td>100</td>
<td>45.0</td>
<td>31.5</td>
<td>27.0</td>
<td>33.7</td>
<td>51.7</td>
</tr>
</tbody>
</table>

*EMGd values are expressed as percentage of the control values.*

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**Suppression of Ventilatory Muscle Activity (Gigliotti et al)**
any consistent increase in PIIA with NPV; the much smaller increase in VT we observed could explain the discrepancy between our observations and those of Rodenstein et al.13 The lack of changes in PIIA suggests to us that EMG suppression could mostly depend on the significant contribution of behavioral effects (voluntary relaxation). Such behavioral response has been thought to account for both magnitude and rapidity of inhibition in EMGd.15 In the study of Rochester et al.,2 the ability of NPV to suppress EMG varied among the normal subjects, and some of them exhibited a substantial reduction in EMG activity when in the tank; in addition, in the two subjects in whom the test was duplicated on different days, a marked variability in EMG suppression was observed. The large variability of the response in normal subjects could be explained by the different ability either to adapt to the ventilator or to voluntarily relax the respiratory muscles. This different ability might account for the discordances among the present and other studies.5,13

**COPD Patients**

At −30 cm H2O, but not at −10 and −20 cm H2O, patients exhibited a significant, but slight increase in VT and SaO2, along with reduction in PtcCO2.

As hypothesized for the normal subjects, the decrease in PtcCO2 could be explained by the slight increase in VT; accordingly, the expected increase in VA with an amelioration of ventilation-perfusion ratio could account for the increase in SaO2.8,11

Not unlike the normal subjects, patients showed a consistent and progressive suppression in EMGd activity (Fig 3), with increasing NPV negative values up to −30 cm H2O. A similar pattern was found for EMGint. In addition, in patient 3 a greater suppression of EMGint accompanied a smaller suppression in EMGd. In this study, negative pressures up to −30 cm H2O were not uncomfortable for the patients, while −40 cm H2O was. Discomfort was associated with a decrease in EMG suppression (EMG increase), clinical symptoms of hyperventilation and mild apneas episodes. Increase in EMG activity over NPV, either behavioral or apnea-induced, might decrease the efficacy of respiratory muscle rest therapy.14

In this article, the magnitude of EMG suppression was greater than that observed in the study of Rodenstein et al.,15 where EMGd suppression during the relaxation period averaged 30 percent of the control value. However, not unlike the results of Rodenstein et al.,15 but in sharp contrast with the study of Rochester et al.,2 in no patient did activation of the ventilator produce an immediate total suppression of EMG activity. Some differences exist, however, between the study of Rochester et al.2 and our studies. First, in the study of Rochester et al.,2 patients were more hypoxemic and hypercapnic than ours; however, blood gas values do not seem to account for the magnitude and rapidity of EMG inhibition with NPV.15 Second, unlike our patients, those of Rochester et al.2 were accustomed to the tank ventilator. Nonetheless, in our study the employment of a negative pressure, similar in magnitude to that used by Rochester with cuirass, caused a not dissimilar marked suppression in inspiratory muscle EMG activity. The observation that the maximal suppression in EMG activity occurred at the maximal level of comfortable negative pressure delivered (−30 cm H2O), could indicate that either an adequate value of negative pressure is necessary to relieve the inspiratory muscle work load, or a partial learning effect was playing a role, during the −10 and −20 cm H2O steps. The fact that in three patients (No. 1, 4 and 7) up run NPV resulted in a progressive suppression in EMGd activity, while during the down run NPV EMGd activity progressively increased (Table 4) might support the first of the two hypotheses.

In the study of Rochester et al.,2 the use of the mouthpiece and nose clip caused a rapid and sharp increase in EMGd activity after the normal EMG activity had been suppressed by the tank respirator. Under a stimulus of a mouthpiece, EMGd was approximately the same irrespective of whether the patients were in or out of the tank. In the present study, when mouthpiece and nose clip were used to measure VT, consistent pre-NPV increase in EMGd and EMGint activity was noticed in all subjects. Nevertheless, unlike the study of Rochester et al.,2 NPV (−1) and −20 cm H2O) was able to suppress this mouthpiece-imposed EMGd increase, and the −30 cm H2O level caused a further suppression, greater than 50 percent of the baseline value recorded without the mouthpiece (Fig 1). This discrepancy could probably be explained by the fact that our subjects adapted for a longer time to breathe with the mouthpiece.

This observation also might suggest that the inspiratory inhibitory activity of the respirator may depend, at least in part, on the magnitude of increase in the inspiratory drive.

In summary, our data seem to indicate that an adequate negative pressure is able to produce substantial suppression of inspiratory muscle EMG activity. In the conditions of the study, the observed results

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**Table 5—Mean EMGd/EMGd Ratio during NPV in the Normal Subjects and in Two COPD Patients**

<table>
<thead>
<tr>
<th></th>
<th>Control −10 cm H2O</th>
<th>−20 cm H2O</th>
<th>−30 cm H2O</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal subjects</td>
<td>100</td>
<td>102.5</td>
<td>102.6</td>
</tr>
<tr>
<td></td>
<td>(7.1)</td>
<td>(2.5)</td>
<td>(12.5)</td>
</tr>
<tr>
<td>COPD patients</td>
<td>100</td>
<td>103.4</td>
<td>90.1</td>
</tr>
<tr>
<td></td>
<td>(3.8)</td>
<td>(5.5)</td>
<td>(7.2)</td>
</tr>
</tbody>
</table>

*EMGd/EMGd ratio is expressed as percentage of the control value. Values are means ± 1 SE.*
are likely to be attributed mostly to behavioral effect. Conditions of increased neural respiratory drive might play an additional role.

The careful monitoring of the EMG activity suppression during NPV might be useful to select patients who might benefit from long-term treatment with this type of ventilator. However, two important questions arise: First, the study protocol was of short duration and the ability to suppress EMG activity with a poncho might not be sustainable over longer time runs. Second, should only patients found to have suppressible EMG be considered appropriate candidates for such trials? To address this question, one would have to be sure that partial EMG suppression at a given negative pressure is more effective for the effects of rehabilitation than no suppression at the same level. This is a point of practical interest which needs investigating.

References
3 Braun NMT, Faulkner J, Hughes RL, Roussos C, Sahgal V. When should respiratory muscles be exercised? Chest 1983; 84:76-84.