Thoracoabdominal Pattern of Breathing in Neuromuscular Disorders*

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**Study objective:** To assess abnormalities in thoracoabdominal pattern of breathing (TAPB) in neuromuscular disorders during spontaneous breathing, intermittent positive pressure ventilation (IPPV) with and without abdominal (AB) binder, and immediately after IPPV.

**Design:** Repeated measures design: Pre-IPPV spontaneous breathing, IPPV, IPPV with AB binder, and post-IPPV spontaneous breathing. In protocol 1, ventilator pressure was held constant at the individual value habitually adopted in sessions of IPPV. In protocol 2, it was increased stepwise from 5 to 30 cm H2O.

**Setting:** University hospital, Department of Pediatrics, Intensive Care, and Neuro-Ventilatory Rehabilitation.

**Patients:** Thirty-one patients with spinal muscular atrophy (SMA) and 19 patients with myopathy, mean age (±SD) 9.7±3 years.

**Measurements:** Tidal volume (Vt), percent thoracic contribution to Vt (%RC), the phase angle between the thoracic and the AB volume changes and the labored breathing index, which is an index of asynchrony taking into account both the phase relationships and relative volumes of rib cage and AB compartments.

**Results:** We observed marked abnormalities in TAPB during spontaneous breathing, especially in the SMA group. %RC, labored breathing index, and phase angle displayed nearly normal values during IPPV. IPPV pressures of 25 to 30 cm H2O were necessary to increase %RC above 80%. AB binding decreased Vt, but led to larger thoracic volumes, especially in patients with SMA. Thoracic contribution to Vt and thoracic volume after IPPV were higher than baseline levels.

**Conclusions:** The quantitative assessment of TAPB enhances the ability to estimate pulmonary function in neuromuscular disorders, and the efficiency of mechanical ventilation.

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**Key words:** abdominal binding; Deuchenne’s muscular dystrophy; intermittent positive-pressure ventilation; labored breathing index; myopathy; neuromuscular disorders; spinal muscular atrophy; thoracoabdominal pattern of breathing

**Abbreviations:** AB=abdominal; f=breathing frequency; FVCsp=forced vital capacity in supine position; FVCap=forced vital capacity in sitting position; IPPV=intermittent positive pressure ventilation; LBI=labored breathing index; QDC=qualitative diagnostic calibration; RC=rib cage; %RC=percent thoracic contribution to tidal volume; RIP=respiratory inductive plethysmograph; SMA=spinal muscular atrophy; TAPB=thoracoabdominal pattern of breathing; Vt=tidal volume

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Patients with neuromuscular disorders generally display abnormal thoracoabdominal patterns of breathing (TAPB) characterized by the asynchrony of the rib cage (RC) and the abdominal (AB) displacements as well as a diminished contribution of either compartment to tidal volume (Vt). These abnormalities have been described in patients with tetraplegia, Duchenne’s muscular dystrophy, and more rarely, congenital myopathy, and spinal muscular atrophy (SMA). In patients with SMA, the abnormal TAPB is often caused by the paralysis of the thoracic ventilatory muscles in presence of normal diaphragmatic activity. Because of the paralysis of the intercostals, the rib cage is not stabilized nor expanded during inspiration, and the inspiratory negative intrathoracic pressure created by the diaphragm contraction causes the RC compartment to move inward. By contrast, in patients with myopathy, the diaphragm may be partially paralyzed, the intercostal muscles assume the primary role in decreasing intrathoracic pressure, and the diaphragm and the intraabdominal content are drawn passively outward into the chest, leading to paradoxical inward AB motion. However, the par-
tial paralysis of the diaphragm may also reduce the imbalance between the forces applied to the RC and AB compartments. Most often, Vr is diminished because of the weakness of the respiratory muscles and the asynchrony of the RC and AB compartments.

In addition to this mechanical effect, the inability to clear secretions by coughing, which normally results from the activation of intercostal and AB muscles, further aggravates the ventilatory disorders. Spontaneous periodic deep breaths (yawns and sighs), which normally spread surfactant and reinflate atelectatic zones, are absent. The atelectasis and the increase in the surface tension of the alveolar lining layer resulting in breathing at low lung volumes further decrease the lung distensibility. Furthermore, the retraction of the rib cage has a detrimental effect on alveolar development in children. For many days, daily sessions of preset pressure ventilation (intermittent positive pressure ventilation, IPPV) have been recommended to counteract the above pathophysiologic processes. This approach is also referred to as mouth (or oral) positive pressure ventilation and should be differentiated from the respiratory therapy (intermittent positive pressure breathing) used in obstructive or postsurgery patients. IPPV is generally achieved with AB binders to force the RC inflation.

However, the quantitative assessment of TAPB during spontaneous breathing and mechanical ventilation has been rarely addressed. For this reason, the effects of IPPV on TAPB are unclear, and the parameters of artificial ventilation are determined empirically. This study analyzed the TAPB in children with SMA or myopathy, before, during and after IPPV. All these patients were highly familiarized with IPPV. In our first protocol, our measurements were done in the normal conditions of IPPV sessions, ie, the settings of the ventilator were those of their habitual IPPV sessions. In the second protocol, the pressure of the ventilator was varied to study its effects on TAPB. In both protocols, TAPB was studied with or without AB binding.

Table 1—Subject Characteristics*

<table>
<thead>
<tr>
<th>Protocol</th>
<th>Pathology</th>
<th>No.</th>
<th>Category</th>
<th>Age, yr</th>
<th>Ht, cm</th>
<th>Wt, kgf</th>
<th>FVC0, %</th>
<th>FVC60, %</th>
<th>IPPV pressure, cm H2O</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>SMA</td>
<td>11</td>
<td>11 Type II</td>
<td>9.9±3.5</td>
<td>123±23</td>
<td>23±9</td>
<td>48±28</td>
<td>41±29</td>
<td>19.1±2.8</td>
</tr>
<tr>
<td>Myopathy</td>
<td>5</td>
<td>3 CMD, 2 CM</td>
<td>9.4±2.9</td>
<td>120±16</td>
<td>15±5</td>
<td>30±12</td>
<td>30±12</td>
<td>19.0±4.2</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>SMA</td>
<td>19</td>
<td>19 Type II</td>
<td>9.0±2.8</td>
<td>122±14</td>
<td>20±5</td>
<td>49±23</td>
<td>44±21</td>
<td>18.7±2.6</td>
</tr>
<tr>
<td>Myopathy</td>
<td>14</td>
<td>9 CMD, 2 DMD, 2 CM</td>
<td>11.2±3.4</td>
<td>128±18</td>
<td>24±9</td>
<td>36±13</td>
<td>36±13</td>
<td>18.6±2.3</td>
<td></td>
</tr>
</tbody>
</table>

*Values are means±SD, kgf=kilogram-force; CMD=congenital muscular dystrophy; CM=congenital myopathy; DMD=Duchenne’s muscular dystrophy; UND=undetermined category. IPPV pressure indicates the pressure habitually adopted with AB binding. Differences between groups were not significant. In protocol 2, one subject with SMA classified as type II was borderline severe.

Materials and Methods

Subjects

Fifty subjects (24 boys, 26 girls), mean age 9.7±3.1 yr, mean height 123.8±16.9 cm, mean weight 21.6±8.6 kg with spinal muscular atrophy—or myopathy—participated in this study. Patient characteristics for each of the two protocols are given in Table 1. The diagnoses were based on clinical and laboratory examinations including nerve conduction studies, electromyograms, and muscle biopsies. All the patients underwent one or two 30-min daily sessions of IPPV for several years. Volume values were accidentally lost in 1 subject with myopathy in protocol 1.

Apparatus

The patients were ventilated with a ventilator (Bird Mark 7; Bird Products Corp, Palm Springs, Calif). IPPV was administered through a mouthpiece, facial mask, or the tracheotomy cannula. The airflow was triggered by inspiratory depression set at 2 cm H2O. The flow of the ventilator was adjusted to maintain breathing frequency around 16 breaths per minute.

A respiratory inductive plethysmograph ([RIP] Respirac Plus; Non-Invasive-Monitoring-Systems; Miami Beach, Fla) was used for respiratory measurements. The coils of the RIP were placed on the chest above the nipple line and on the abdomen at the umbilical level. The RIP was calibrated using the semiquantitative single-position method (Qualitative Diagnostic Method13-15 [QDC]) and then against the integrated flow of a pneumotachometer (Fleisch No. 00; Harvard Biosciences; Les Ulis, France) connected with a differential pressure transducer (Schlumberger; Velizy, France; ±2 millbar). The pneumotachometer was attached to a mouthpiece or a facial mask and maintained manually by the experimenter throughout the calibration period. The volume signals from the RIP and the pneumotachometer were recorded on a strip chart recorder, and the calibration was done by using a 1-L syringe with a minimum of ten breaths. The signals from the RIP were processed by a microcomputer (Software Respi-Events; Non-Invasive-Monitoring Systems; Miami Beach, Fla) to produce total breath duration, breathing frequency (f), Vr, and thoracic contribution to Vr expressed as the percent Vr/Vt ratio, and denoted %RC. This index was negative when thoracic-abdominal paradox was present. The volume of the thoracic compartment (hereafter designed as “thoracic volume”) was estimated by the product %RC×Vt.

The labored breathing index (LBI) provided by the processing software was used as a global index of thoracal-abdominal coordination taking into account both the phase relationships and relative volumes of rib cage and abdominal compartments. LBI was calculated as follows: the integrals of the absolute values of the derivatives of the inspiratory limbs of RC and AB, irrespective of their
phase relationship, were calculated. Then the sum of these two values was divided by the integral of the derivative of the inspiratory limb of Vt. The LBI was equal to one in case of perfect coordination of the thoracic and AB movements, and above one otherwise (values below 1.2 are considered normal). The phase angle provided by the processing software was also used as index of thoracoabdominal asynchrony. Contrary to LBI, the phase angle is independent of the absolute or relative contributions of the RC and the AB area. The phase angle was computed from the Lissajous loop of the AB and thoracic signals over a tidal breath, assuming that these signals approximate sinus waves. In fact, the AB and RC signals are generally rather square or triangular, but previous studies have shown that the error due to the assumption that they are sinusoidal is less than 9%. Phase angles comprise between 0° (perfect inphase movement) and 180° (paradoxical motion).

Procedures

All the patients were studied in the supine position. After their braces were removed, the patients were fitted with RIP transducers. Once breathing was stabilized, breathing was recorded for 5 min for QDC calibration.

Protocol 1: After QDC calibration, spontaneous breathing was recorded for 5 min. Then, IPPV was applied for two successive 5-min phases: the first one without AB binding, and the second one with AB binding, at the habitual individual pressure level. This pressure comprised between 15 and 25 cm H2O (Table 1). Finally, 5 min of spontaneous breathing without AB binding were recorded.

Protocol 2: After 5 min of recording spontaneous breathing, pressure was increased by 5 cm H2O steps from 5 to 25 cm H2O. Each pressure level was maintained for 1 min. A supplementary step of 30 cm H2O was performed in 9 patients with SMA and 9 patients with myopathy. After the final stage was completed (the 25 or 30 cm H2O stage), the ventilator was set at the habitual individual pressure (comprised between 15 and 25 cm H2O, Table 1), and a 5 min period of IPPV was recorded with AB binding. Finally, spontaneous breathing without AB binding was recorded for 5 min.

Statistics

Results are expressed as mean ± SD. All variables were analyzed separately. In protocol 1, we performed analyses of variance (ANOVA) with groups (two levels: SMA and myopathy) as a between-subjects factor, and phases (four levels: initial spontaneous breathing, IPPV without AB binding, IPPV with binding, and final spontaneous breathing) as a repeated measures factor. In protocol 2, we used ANOVAs with pressure as a within-subject factor. To take the heterogeneous correlations among the repeated measurements into account, p values were adjusted using the Greenhouse and Geisser Epsilon when appropriate. Partial comparisons were done for the pre-post changes and the effects of binding. The Statistical Softwares (Superanova and Staview; Abacus Concepts; Berkely, Calif) were used for these analyses.

RESULTS

Protocol 1

Baseline Values: The subjects displayed high levels of LBI and phase angle during spontaneous breathing (Fig 1). The %RC during spontaneous breathing was low in patients with SMA, and within normal limits in patients with myopathy. However, this and all the remaining differences between groups were nonsignificant.

Breathing Pattern During IPPV Without Binding:

The %RC increased up to 58% in the SMA group, and 65% in the myopathy group. The thoracic volume rose to 400 mL in patients with SMA and 300 mL in patients with myopathy. During IPPV, LBI and phase angle reached nearly normal levels (Fig 2).

Effects of AB Binding: The partial comparison between IPPV with and without binders showed a nonsignificant decrease in Vt. However, we observed large and significant increases in %RC and thoracic volume caused by binding, Fischer’s ratio (F; 1,
14) = 124.38, p < 0.0001, and F(1,13) = 4.96, p < 0.004, respectively. Because the interaction of binding with group factor was not significant, it is unclear whether these benefits of binders for increasing %RC and thoracic volume concerned the two groups or the SMA group only (Fig 1).

**Short-term Effects of IPPV:** The comparison between pre-IPPV and post-IPPV values did not yield significant results in either group, nor globally for the whole sample of subjects.

**Protocol 2**

As a rule, the results of protocol 1 were confirmed by protocol 2 (Fig 3), but several nonsignificant effects in protocol 1 were significant in protocol 2. This was certainly due to the larger sample of subjects in the latter protocol.

**Baseline Values:** The subjects in the two groups displayed low Vr and %RC, and high levels of LBI and phase angle, especially in SMA. However, the differences between groups were not significant (Figs 3 and 4). These data confirmed the observations of protocol 1.

**Effects of Pressure Changes:** Vr increased linearly from 200 mL to about 900 mL in both groups as a function of pressure. The %RC displayed an asymptotic pattern, but thoracic volume increased linearly over the whole range of pressure. Clearly, the increase in Vr for high pressures was mainly due to the increasing contribution of the AB compartment. The thoracic volume also increased, but proportionally less than the AB volume. LBI decreased toward normal values for IPPV pressures higher than 20 to 25 cm H2O (Fig 3). Phase angle displayed a similar trend. These effects were similar in the two groups. Arterial oxygen saturation (SaO2) increased in both groups as a function of pressure. In the SMA group, SaO2 increased on average from 97.6 ± 1.7 to 98.2 ± 1.9. In the myopathy group, SaO2 increased from 96.0 ± 3.3 to 98.2 ± 1.9. The increase was significant on the whole sample of patients, F(6,84) = 3.31, p < 0.02, but not the interaction between pressure and group.

**Effects of AB Binding:** Within each group, Vr with AB binding (ie, at approximately 19 cm H2O of IPPV pressure, Table 1) was similar to Vr without binding at 15 cm H2O (Fig 3). The differences in thoracic volume with and without binders at the same pressure were also calculated individually. These increases averaged 63 mL in the SMA group and 34 mL in the myopathy group. The benefit of binding (Table 2) was small in patients with myopathy in comparison with baseline levels of thoracic volume. In fact, the increase in %RC

**Figure 2.** Thoracoabdominal pattern of breathing during IPPV without and with AB binding, and after IPPV in patients with SMA (n=11) or myopathy (n=4). Values are means±SEM.
Variables were significantly higher between pre-IPPV and thoracic compartment. The breathing decrease in the AB contribution to $V_r$, leading to a decrease in $V_t$, rather than to the increase of the thoracic compartment. With binding, LBI and phase angle attained nearly normal values.

**Short-term Effects of IPPV:** The comparison between pre-IPPV and post-IPPV periods of spontaneous breathing showed that %RC and thoracic volume were significantly higher immediately after IPPV. In the SMA group, the respective increases in these two variables were 28 mL and 14.7% respectively. In the myopathy group, the corresponding increases were 13.6 mL and 3.78%. This was confirmed by a significant prepost comparison, $F(1,32)=4.41$, $p<0.04$ and $F(1,32)=4.85$, $p<0.035$, respectively, but the interaction between the prepost factor and group factor was not significant for either variable. Prepost changes in $V_r$ were not significant. This confirmed the tendencies observed in protocol 1. Prepost-changes in LBI and phase angle were marginally significant ($F(1,32)=3.60$, $p<0.07$ and $F(1,32)=3.34$, $p<0.08$, respectively.

**Relationships Between Pulmonary Function Indices**

All the initial spontaneous data collected in the first phase of each protocol (which was identical for all the subjects) were pooled together for this analysis. Spontaneous ventilatory data are summarized in Table 2. The only significant difference was found for %RC: the patients with SMA had a smaller %RC, $F(1,48)=4.98$, $p<0.04$. In addition, a $\chi^2$ test revealed that abnormal

![Graph showing tidal volume and thoracic volume changes](image)

![Graph showing ventilator pressure and laborous breathing index changes](image)

**Table 2—Summary Data of Ventilatory Variables During Spontaneous Breathing**

<table>
<thead>
<tr>
<th>Pathology</th>
<th>$f$, br/min</th>
<th>$V_t$, mL</th>
<th>%RC</th>
<th>Thor Vol, mL</th>
<th>LBI</th>
<th>Phase Angle, degree</th>
</tr>
</thead>
<tbody>
<tr>
<td>SMA, n=31</td>
<td>20.2±4.6</td>
<td>229±66</td>
<td>13±37</td>
<td>26±82</td>
<td>2.1±1.0</td>
<td>110±51</td>
</tr>
<tr>
<td>Myopathy, n=19</td>
<td>23.2±6.9</td>
<td>215±95</td>
<td>38±37</td>
<td>63±78</td>
<td>1.7±1.0</td>
<td>90±12</td>
</tr>
</tbody>
</table>

*Values are means±SD. Thor vol=thoracic volume calculated as the $V_t$ by %RC product. Phase angle=between thoracic and AB displacements.
TAPB characterized by LBI > 1.3 and phase angle > 30° were more frequent in SMA than myopathy (p < 0.036).

As expected, forced vital capacity in supine position (FVC0) and forced vital capacity in sitting position (FVC90) were closely related in the two groups (r² = 0.91 and r² = 0.80, respectively, Fig 4). This suggested that these indices were redundant in our sample of patients, and only FVC0 was considered for the remaining analyses. FVC0 was a poor predictor of LBI and phase angle (Fig 4). In fact, the inspection of individual data showed that subjects with nearly normal FVC0 displayed no thoracoabdominal asynchrony. The subjects with low FVC0 may exhibit either high or low LBI and phase angle. This suggested that FVC per se was not a good predictor of abnormal TAPB.

The relationship between LBI and phase angle was analyzed by pooling together the data of all the phases of each protocol (Fig 5). No statistical test was performed on this sample because several data were collected for each subject. However, the visual inspection of the scatterogram in Figure 5 suggested that LBI and phase angle displayed a curvilinear relationship. The normal values of LBI and phase angle were generally collected during mechanical ventilation, although several subjects also displayed normal values during spontaneous breathing. Relatively high values of phase angle were associated with nearly normal LBI when the paradoxical volume change of one compartment was small compared to Vt.

Discussion

The purpose of this study was to quantitatively assess the abnormalities in the TAPB during spontaneous breathing or mechanical ventilation in subjects with SMA or myopathy. The first protocol reproduced their habitual IPPV session, whereas in the second protocol, IPPV pressure was manipulated. In fact, these two protocols yielded similar results. Accordingly, the observations of the second protocol may be generalized to the normal IPPV sessions with constant pressure. We observed that the marked abnormalities in TAPB associated with SMA and myopathy were not present during IPPV, but reappeared after IPPV, despite small but significant short-term effects. Pressures as high as 25 to 30 cm H₂O were necessary to increase the thoracic contribution to Vt above 80%. Finally, we observed that AB binding caused small but significant increases in thoracic volumes. However, the absolute values of this variable, when compared with the spon-
taneous levels, suggested that AB binding in patients with myopathy was of little benefit.

Most, but not all the subjects displayed abnormal TAPB during spontaneous breathing. As a rule, percent thoracic contribution to Vt were below normal limits, especially in patients with SMA. However, we observed a large interindividual variability in both groups. For example, only 13 of the 34 patients with SMA and 3 of the 16 patients with myopathy displayed negative percent thoracic contributions, i.e., full paradoxical breathing (this difference was significant). By contrast, TAPB was nearly normal during IPPV. This was in line with previous observations on a group of five patients with neuromuscular disorders: three subjects with spinal transection, one with congenital myopathy, one with SMA. As noted in this previous study, it may be posited that the decrease in thoracoabdominal asynchrony during IPPV was linked to the passivity of the ventilatory muscles during mechanical ventilation. During spontaneous breathing, the thoracoabdominal asynchrony was mainly the result of the imbalance of muscular forces which normally expand the thoracic and the AB compartments, and accordingly, the thoracoabdominal asynchrony should disappear when all ventilatory muscles are inactive. During IPPV, the thoracic and the AB compartments move in phase under the effect of the ventilator pressure.

The present data support the idea previously expressed that the indices of thoracoabdominal asynchrony during mechanical ventilation provide an indirect assessment of the respiratory muscles activity. This may be of practical utility when direct electromyogram is not available in the clinical setting. In particular, the assessment of thoracoabdominal asynchrony may reflect the degree of familiarity with IPPV, which is achieved through a learned partial inhibition of ventilatory activity, i.e., a small inspiratory depression (to trigger the ventilator) followed by the relaxation of the respiratory muscles. However, the hypothesis that thoracoabdominal asynchrony closely reflects the pattern of muscular activity should be directly tested by studying the actual correlation between the electromyogram of respiratory muscles and TAPB variables. Although the main trend was that the abnormal TAPB tended to resume after IPPV, we observed small prepost improvements. These were not expected to occur if muscle passivity was the only cause of the normalization of TAPB. These short-term effects were possibly the result of the expansion of atelectatic zones during IPPV. Long-lasting expansion of atelectatic zones was obtained by inflating the lungs up to an airway pressure of 30 cm H2O in anesthetized patients. Three inflations held for 15 s caused an expansion of atelectatic zones which lasted for at least 40 min later. A similar process may explain the short-term effects of IPPV in the present study.

It is of interest that post-IPPV effects concerned %RC and thoracic volume, but not Vt. Similarly, it has been reported that a 15-min period of IPPV had no short-term effect on lung volumes in subjects with generalized neuromuscular disorders. Similarly, no immediate significant improvements in lung volumes or pulmonary compliance after IPPV were found in tetraplegics. Our results showed that the same levels of Vt before and after IPPV were achieved with different TAPB. Whether or not these changes were clinically significant is difficult to decide, especially if we take into account the fact that all the subjects currently underwent daily 30- to 60-min sessions of IPPV. We may posit that this daily practice may have prevented the development of atelectasis in the poorly ventilated zones of the upper thorax, as previously noted. If so, the effects of one single session could hardly be observed.

In general terms, the two groups displayed comparable abnormalities in TAPB, despite the different nature of the neurologic disorders. In SMA, the diaphragm is active, whereas intercostal and AB muscles are paralyzed. In Duchenne's and congenital myopathies, the diaphragm may be also partly paralyzed. As noted above, under the hypothesis that abnormal TAPB is caused by the imbalance of muscular forces which normally expand the thoracic and abdominal compartments, patients with myopathy would exhibit larger %RC than patients with SMA. This may also explain that abnormal SMA, as reflected by LBI and the phase angle, were more frequent in subjects with SMA.

Although positive pressure breathing has been questioned in many applications, it should be stressed that the validity of this technique has been acknowledged in patients at risk of respiratory failure because of decreased respiratory function secondary to kyphoscoliosis or neuromuscular disorders and in patients with atelectasis that had not improved with simpler therapy. (Contrary to Handelsman's contention, DeTroyer and Deisser did not state that patients with neuromuscular disorders “did not benefit from IPPB as generally administered.” These authors reported that lung volumes and static pulmonary compliance were not acutely modified by IPPB, but they added, “It is possible that continuous use of IPPB [for example 3-15 min sessions every day] in such patients could help to prevent further alteration in pulmonary function. More importantly, it is possible that periodic hyperinflation of the lungs in the early stages of the disease prevents the development of what we believe to be diffuse microatelectasis and then alters the natural history of the condition.”). Negative outcomes of
positive pressure breathing therapy in other respiratory diseases do not extend to neuromuscular disorders, because the crucial peculiarity of IPPV in neuromuscular disorders is to be basically a prevention tool. Because the respiratory disorder is a consequence of muscle weakness or paralysis, achieving the expansion of the lungs through IPPV arguably counters the respiratory pathophysiologic effects of muscular deficiency. This approach is fundamentally different from the therapeutic purposes in COPD, asthma, cardiovascular disorders, and postoperative atelectasis, which are the main categories of patients treated by this technique.24-26

Despite long-term clinical experience with patients with neuromuscular disorders,27 only a large-scale clinical trial may provide convincing evidence that IPPV is a valid method. This clinical trial would necessarily rely on an objective assessment of the patients' ventilatory dysfunction and the effects of IPPV in given conditions (ventilator pressure, binding, duration of sessions, etc.). The present study provided quantitative assessment of the thoracoabdominal asynchrony during spontaneous breathing and also for various levels of IPPV pressure. In addition to previous results,22 these results enhance the ability to estimate pulmonary function in neuromuscular disorders and the efficiency of mechanical ventilation.

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