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Reduced Inspiratory Muscle Endurance Following Successful Weaning From Prolonged Mechanical Ventilation*

Angela T. Chang, PhD; Robert J. Boots, MBBS; Michael G. Brown, MAppSc; Jennifer Paratz, PhD; and Paul W. Hodges, PhD

Study objectives: Respiratory muscle weakness and decreased endurance have been demonstrated following mechanical ventilation. However, its relationship to the duration of mechanical ventilation is not known. The aim of this study was to assess respiratory muscle endurance and its relationship to the duration of mechanical ventilation.

Design: Prospective study.

Setting: Tertiary teaching hospital ICU.

Patients: Twenty subjects were recruited for the study who had received mechanical ventilation for ≥48 h and had been discharged from the ICU.

Measurements: FEV₁, FVC, and maximal inspiratory pressure (PImax) at functional residual capacity were recorded. The PImax attained following resisted inspiration at 30% of the initial PImax for 2 min was recorded, and the fatigue resistance index (FRI) [PImax final/PImax initial] was calculated. The duration of ICU length of stay (ICULOS), duration of mechanical ventilation (MVD), duration of weaning (WD), and Charlson comorbidities score (CCS) were also recorded.

Relationships between fatigue and other parameters were analyzed using the Spearman correlations (p).

Results: Subjects were admitted to the ICU for a mean duration of 7.7 days (SD, 3.7 days) and required mechanical ventilation for a mean duration of 4.6 days (SD, 2.5 days). The mean FRI was 0.88 (SD, 0.13), indicating a 12% fall in PImax, and was negatively correlated with MVD (r = −0.65; p = 0.007). No correlations were found between the FRI and FEV₁, FVC, ICULOS, WD, or CCS.

Conclusions: Patients who had received mechanical ventilation for >48 h have reduced inspiratory muscle endurance that worsens with the duration of mechanical ventilation and is present following successful weaning. These data suggest that patients needing prolonged mechanical ventilation are at risk of respiratory muscle fatigue and may benefit from respiratory muscle training.

Key words: fatigue; inspiratory muscle; mechanical ventilation

Abbreviations: APACHE = acute physiology and chronic health evaluation; CCS = Charlson comorbidity score; CMV = controlled mandatory ventilation; FRC = functional residual capacity; FRI = fatigue resistance index; ICULOS = ICU length of stay; MVD = duration of mechanical ventilation; NMBA = neuromuscular blocking agent; PImax = maximal inspiratory pressure; RPE = rate of perceived exertion

Thirty-nine percent of all patients admitted to the ICU receive mechanical ventilation. Mechanical ventilation is most commonly used in the management of patients with acute respiratory failure as it reduces the work of breathing and diaphragm activity. However, prolonged mechanical ventilation promotes respiratory muscle weakness in the absence of critical illness and is localized to the respiratory muscles. The degree of weakness has been related to the duration of mechanical ventilation and has...
been suggested as a possible cause of delayed weaning from mechanical ventilation.\(^8\,^9\)

Prolonged mechanical ventilation has been defined as mechanical ventilation for > 48 h.\(^10\) As mechanical ventilation during this period is most likely due to the primary condition of the patient or to early complications of underlying illness, it is unlikely that patients will recover quickly following > 48 h of mechanical ventilation.\(^11\) Although controlled mandatory ventilation (CMV) for longer than this period of time has reduced diaphragm strength in animal models,\(^3\,^4\) the effect on muscle fatigue appears to be more complex.

There have been inconsistent reports of the effect of up to 5 days of CMV on diaphragm endurance, and studies have reported no change,\(^4\,^5\) decreased endurance,\(^12\) or an improvement in diaphragm endurance.\(^13\) However, this variability may be due, at least in part, to the shorter period of mechanical ventilation, with larger changes seen in patients receiving mechanical ventilation for > 7 days.\(^13\) Preliminary work by Anzueto et al\(^3\) has indicated that periods of CMV for 11 days reduced diaphragm endurance time by 45%. However, the relationship between the endurance of the diaphragm and other inspiratory muscles and the duration of mechanical ventilation was not examined. In addition, the above studies were all undertaken in animals that were not critically ill and ventilated with CMV only. Although these models provide insight into the effects of mechanical ventilation on the respiratory muscles, further investigation into respiratory muscle fatigue in critically ill patients is needed to design preventative interventions. The aims of this study were as follows: (1) to assess the inspiratory muscle endurance following successful weaning and discharge from the ICU; and (2) to study the relationship between respiratory muscle endurance and mechanical ventilation duration.

**Materials and Methods**

**Subjects**

Twenty subjects (9 women) with a mean age of 62 years (SD, 16 years) who had received mechanical ventilation (using synchronized intermittent mandatory ventilation and/or biphasic positive airway pressure ventilation, where the latter is a subtype of pressure-controlled ventilation\(^14\)) for > 48 h were recruited for the study from the institutional ICU (Table 1). Subjects were enrolled as consecutive patients admitted to the ICU who met the selection criteria. All study subjects were alert and cooperative, and had been discharged from the ICU for 24 h. Subjects were excluded if they had experienced a cerebrovascular accident, had a head or spinal cord injury, had preexisting diaphragm palsy, neuropathy, or myopathy, had unstable ischemic heart disease, ischemic changes, or arrhythmias on an echocardiogram, had a temperature of > 37.8°C except for systemic inflammatory response syndrome, had experienced thoracic trauma or burns to the thorax, required a fraction of inspired oxygen of > 30%, or were unable to be transported by wheelchair to the laboratory.

Written informed consent was obtained from the subject, with ethical clearance from the institutional human medical research ethics committee. All procedures were conducted in accordance with the Declaration of Helsinki.

**Measurements**

**Spirometry:** Measurements of the subject's FVC and FEV\(_1\) were measured using a spirometer (Compact; Vitalograph; Lenexa, KS) in accordance with American Thoracic Society guidelines.\(^15\)

**Inspiratory Muscle Strength:** The maximal inspiratory pressure (P\(_{\text{Imax}}\)) was recorded at functional residual capacity (FRC) using a two-way valve with two inflatable balloon valves (model 5600; Hans Rudolph; Kansas City, MO) attached to a flanged mouthpiece (n = 19) via a bacterial/viral filter (B&V Bird Pty Ltd; Middlepark, VIC, Australia). Two inductance plethysmographs (Respiritrace; Non-Invasive Measuring Systems; North Bay Village, FL) bands were placed at the level of the nipple and umbilicus to measure chest expansion and to monitor the repeatability of FRC to within 150 mL or 10% of chest wall motion observed during an isovolume maneuver.

**Demographic Details:** Measurements of the duration of mechanical ventilation (MVD) [in days] and weaning duration (WD) [in hours], which was defined as the time from the initiation of the first spontaneous breathing trial using either continuous positive airway pressure and pressure support or T-piece trials until extubation, and the ICU length of stay (ICULOS) were recorded from the subject's medical records. In addition, demographic information, including primary ICU admission diagnosis, medical history, the degree of comorbidity quantified via the Charlson comorbidity score (CCS),\(^16\) and the use of corticosteroids and neuromuscular blocking agents (NMBAs), were recorded. The subject's severity of acute injury was assessed with the acute physiology and chronic health evaluation (APACHE) II score within 24 h of ICU admission.

**Procedure:** Data were recorded in two sessions. Spirometry (FEV\(_1\), FVC) was measured in the first session. P\(_{\text{Imax}}\) and endurance measurements were made during a second session to minimize fatigue. Spirometry was performed with subjects in a high sitting position. For one subject with a tracheostomy, an attachment was used. Prior to inspiratory muscle testing, pressure transducers and inductance plethysmographs were calibrated. Inductance plethysmography bands were placed around the subject's torso with the subject seated. The subject was instructed to breathe through the inspiratory valve via the mouthpiece/attachment, and the inspired volume was measured by a respiratory mechanics monitor (Ventrak; Novametrix; Wallingford, CT) attached at the inspiratory port. An isovolume maneuver was used to calibrate the inductance plethysmography bands.\(^17\) During testing, the subject's heart rate and pulse oximetric saturation were monitored via a pulse oximeter.

Subjects exhaled to FRC. The balloon valves were then inflated, and the subject inhaled maximally for 4 s as the P\(_{\text{Imax}}\) was recorded (solid-state differential pressure transducer, model LX06015D; Sensym; Milpitas, CA). The balloons were then deflated. Data were collected via a data acquisition system (model ML735 PowerLab; ADInstruments; Castle Hill, NSW, Australia) and were analyzed using specific software (Chart; ADInstruments).
<table>
<thead>
<tr>
<th>Age/Sex</th>
<th>Diagnosis</th>
<th>ICULOS, d</th>
<th>MVD, d</th>
<th>WD, h</th>
<th>Highest Fio₂, cm H₂O</th>
<th>Airway</th>
<th>Days Post CCS</th>
<th>APACHE II Score</th>
<th>FEV₁, % predicted</th>
<th>FVC, % predicted</th>
<th>Pmax Initial, cm H₂O</th>
<th>Pmax Final, cm H₂O</th>
<th>FRI</th>
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<td>(L) Pneumothorax</td>
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<td>3</td>
<td>0</td>
<td>23</td>
<td>97</td>
<td>99</td>
<td>−42</td>
</tr>
<tr>
<td>52/M</td>
<td>Hematemesis, PR bleed</td>
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<td>4</td>
<td>20</td>
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<td>−33</td>
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<td>None</td>
<td>7</td>
<td>5</td>
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<td>43</td>
<td>53</td>
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<td>7</td>
<td>36</td>
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<td>None</td>
<td>7</td>
<td>0</td>
<td>11</td>
<td>41</td>
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<tr>
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<td>25% burns</td>
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<td>None</td>
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<td>Abdominoperineal resection</td>
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<td>3</td>
<td>4</td>
<td>0.5</td>
<td>5</td>
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<td>6</td>
<td>3</td>
<td>14</td>
<td>97</td>
<td>97</td>
<td>−33</td>
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<td>12</td>
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<td>2</td>
<td>0</td>
<td>14</td>
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<tr>
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<td>10</td>
<td>2</td>
<td>24</td>
<td>42</td>
<td>47</td>
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<td>5</td>
<td>98</td>
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<td>11</td>
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<tr>
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<td>4</td>
<td>0.6</td>
<td>10</td>
<td>Tracheostomy</td>
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<td>39</td>
<td>39</td>
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<td>7</td>
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<td>22</td>
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<td>13</td>
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<td>N</td>
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<td>4</td>
<td>17</td>
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<tr>
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<tr>
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<td>97</td>
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<td>10</td>
<td>None</td>
<td>4</td>
<td>3</td>
<td>19</td>
<td>58</td>
<td>72</td>
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<tr>
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<td>5</td>
<td>41</td>
<td>0.5</td>
<td>5</td>
<td>None</td>
<td>13</td>
<td>3</td>
<td>21</td>
<td>87</td>
<td>73</td>
<td>−28</td>
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<tr>
<td>73/F</td>
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<td>3</td>
<td>50</td>
<td>0.5</td>
<td>5</td>
<td>None</td>
<td>16</td>
<td>1</td>
<td>26</td>
<td>21</td>
<td>31</td>
<td>−24</td>
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<tr>
<td>58/F</td>
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<td>46</td>
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<td>1</td>
<td>16</td>
<td>72</td>
<td>74</td>
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<tr>
<td>Mean</td>
<td></td>
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<td>8</td>
<td>5</td>
<td>42</td>
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<td>7</td>
<td>7</td>
<td>2</td>
<td>21</td>
<td>61</td>
<td>66</td>
<td>−33</td>
</tr>
</tbody>
</table>

†A Pmax of 0% was used during the endurance trial.
Subjects then breathed through an inspiratory resistance that was equivalent to 30% of the initial P\textsubscript{max} (Threshold IMT trainer; Respirationics; Murrysville, PA), with further P\textsubscript{max} measurements made at 30-s intervals. This level of resistance was selected as preliminary trials indicated that resistance equivalent to 50% of P\textsubscript{max} resulted in severe dyspnea. If the subject was not able to inspire at 30% of their P\textsubscript{max}, no resistance was applied and P\textsubscript{max} measurements were repeated at 30-s intervals for 2 min (n = 3). During the loading task, the rate of perceived exertion (RPE) was recorded using a modified Borg scale (scale, 0 to 10),\textsuperscript{11} with testing ceasing if the RPE was ≥ 7. If, at any time, pulse oximetric saturation fell > 10% from initial values or to < 90%, the heart rate increased > 30 beats per minute, or there was any other clinical deterioration, the testing ceased and supplemental oxygen administered. Four subjects were unable to complete the testing protocol. These subjects reported an RPE score of ≥ 7 before completion of the loading trial.

Data Analysis: The fatigue resistance index (FRI) is defined as the change in P\textsubscript{max} following a fatiguing task divided by the initial P\textsubscript{max} strength\textsuperscript{19} and was calculated as the P\textsubscript{max} difference in FRI between a fatigue load of 0% to 50% applied and P\textsubscript{max} measured after a 2-min inspiratory loading protocol divided by the initial P\textsubscript{max}.

Statistical Analysis: The change in P\textsubscript{max} following the fatigue protocol was analyzed with a repeated-measures analysis of variance. All relationships with FRI were analyzed using Spearman \( \rho \) correlations. Kruskal-Wallis analysis was used for nonparametric variables. The relationship between long-term ventilation and FRI, age, comorbidities, APACHE II score, and ICULOS was assessed by division into subjects with MVD of ≥ 7 days and subjects with MVD of < 7 days. Subjects who were unable to complete the inspiratory loading component (n = 4) were also compared with subjects who completed the protocol (n = 16). Finally, the effect of inspiratory load during the fatigue task (P\textsubscript{max}, 0% vs 30%, respectively) on FRI was analyzed. A total of 16 subjects were required to detect a correlation coefficient of > 0.6 and a 10% fall in P\textsubscript{max} with a power of 0.8 and a significance of 0.05.

**Results**

Spirometry, P\textsubscript{max}, and demographic data are shown in Table 1. The P\textsubscript{max} fell following the 2-min inspiratory loading protocol from −34.7 cm H\textsubscript{2}O (SD, 19.6 cm H\textsubscript{2}O) to −30.4 cm H\textsubscript{2}O (SD, 17.06 cm H\textsubscript{2}O; \( p = 0.002 \)). The mean FRI was 0.88 (SD, 0.13), representing a 12% fall in P\textsubscript{max} after the loading protocol. There was no difference in any parameter between subjects who completed the inspiratory loading protocol (n = 16) and those who did not complete it (n = 4; \( p > 0.05 \)). There was no difference in FRI between a fatigue load of 0% P\textsubscript{max} (n = 3) and 30% P\textsubscript{max} (n = 13; \( p = 0.332 \)).

Relationship Between FRI and Other Parameters

Greater fatigue (lower FRI) was related to longer MVD (\( \rho = −0.65; p = 0.007 \)) [Fig 1]. There was no relationship between FRI and FEV\textsubscript{1} (\( \rho = 0.193; p = 0.49 \)), FVC (\( \rho = −0.004; p = 0.99 \)), age (\( \rho = 0.344; p = 0.19 \)), CCS \( \rho = 0.262; p = 0.37 \), APACHE II score (\( \rho = −0.192; p = 0.49 \)), ICULOS (\( \rho = −0.471; p = 0.07 \)), time since ICU discharge (\( \rho = 0.121; p = 0.66 \)), inspiratory muscle strength (\( \rho = 0.022; p = 0.94 \)), or the use of NMBAs (\( \rho = 0.392; p = 0.13 \)).

**Effect of Long-term Ventilation on FRI**

Subjects who received mechanical ventilation for ≥ 7 days (n = 4) had a lower FRI compared to those who received mechanical ventilation for < 7 days (0.77 vs 0.92, respectively; \( p = 0.047; n = 16 \)) [Fig 2]. Patients who received mechanical ventilation for

![Figure 1](https://example.com/figure1.png)

**Figure 1.** Relationship between FRI and mechanical ventilation. The horizontal line denotes FRI = 1. All points below the line denote decreased force-generating capacity (ie, fatigue), and all points above the line denote increased force-generating capacity.

![Figure 2](https://example.com/figure2.png)

**Figure 2.** Change in P\textsubscript{max} after the loading trial. Top, A: subject ventilated for < 7 days. Note that there is no change in P\textsubscript{max} post-loading task at 30% of P\textsubscript{max}. Bottom, B: subject received mechanical ventilation for ≥ 7 days. Note the fall in P\textsubscript{max} compared to initial levels.
a longer duration were younger (48.0 vs 64.9 years, respectively; p = 0.023), had been admitted to the ICU for a longer period of time (12.8 vs 6.5 days, respectively; p = 0.007), and required weaning for longer periods (16.3 vs 9.1 h, respectively; p = 0.030). There was no difference in CCS (p = 0.567) or APACHE II scores (p = 0.273) between groups.

**Discussion**

This is the first study to investigate the relationship between the duration of mechanical ventilation and inspiratory muscle endurance in patients who had received mechanical ventilation in the ICU. The results show that endurance is reduced with longer periods of mechanical ventilation in patients who were successfully weaned following mechanical ventilation for ≥ 48 h. The effect on strength cannot be determined, however, as absolute lung volume was not recorded and FRC was standardized between trials only. There was no correlation between inspiratory muscle endurance and other parameters, including APACHE II score, comorbidities, age, inspiratory muscle strength, administration of NMBAs, or duration of ICU admission.

**Decreased Inspiratory Muscle Endurance Following Prolonged Ventilation**

Although we have demonstrated that prolonged mechanical ventilation is associated with reduced inspiratory muscle endurance in patients who received mechanical ventilation for ≥ 48 h, conflicting findings have been reported in animal studies. No change in diaphragm FRI was demonstrated by Sassoon et al in rabbits following 72 h of CMV, and by Yang et al in rats following CMV for 57.6 h. In contrast, Capdevila et al reported a fall in diaphragm and intercostal FRI after 51 h of CMV in rabbits, and Shanely et al found an increase in FRI after CMV for 18 h in rats. However, the increased endurance in the latter study may be due to the shorter period of CMV and the resultant increased oxidative capacity of the diaphragm. Conversely, atrophy of the oxidative fibers may occur with prolonged periods of mechanical ventilation, leading to decreased endurance. This was supported by the negative relationship between MVD and FRI in the present study.

The inspiratory muscle fatigue demonstrated in the current study may be due to the longer period of mechanical ventilation (mean duration, 110.4 h) compared to those in the animal studies described above. Interestingly, one study that investigated long-term mechanical ventilation (11 days) in baboons found decreased endurance of the diaphragm. However, those data were preliminary findings as only three animals were studied and no statistical analysis was undertaken.

Reduced endurance of the inspiratory muscles may be the cause rather than the outcome of longer periods of mechanical ventilation. It is possible that patients who required longer periods of mechanical ventilation may have had a preexisting decrease in diaphragm endurance, which may have contributed to the increased period of ventilation. Whether the increased fatigability is the cause or the effect of prolonged mechanical ventilation does not compromise the importance of the findings and the implications of these findings for rehabilitation.

The lower FRI in patients receiving mechanical ventilation for ≥ 7 days suggests that these patients may not be able to meet respiratory demands with the tasks associated with increased inspiratory load, such as rehabilitation and the activities of daily living. This may restrict a patient’s performance, as ventilatory limitations have been shown to limit function.

**Possible Causes of Reduced Endurance**

There are two main types of fatigue, central and peripheral. Central fatigue is defined as a reduction in the voluntary contraction force due to reduced CNS output, and peripheral fatigue occurs due to the failure of the neuromuscular junction or the contractile component of muscle. Peripheral fatigue has been demonstrated to occur following mechanical ventilation in animals, with both transmission fatigue (high-frequency) and contractile fatigue (low-frequency) present following CMV. However, the role of central fatigue is not yet known. Although the present study demonstrates inspiratory fatigue following mechanical ventilation in critically ill patients, the separate central and peripheral contribution in the development of fatigue requires further investigation.

Weakness and fatigue following mechanical ventilation may be a manifestation of conditions such as critical illness polyneuropathy, critical illness myopathy, and disuse atrophy. In addition to these conditions, the sustained use of medications such as NMBAs or corticosteroids contributes to the development of weakness and fatigue, however, their effect on the contractile properties of respiratory muscles in patients receiving mechanical ventilation is not yet known. Regardless of the type of fatigue and the possible mechanisms behind them, the current study demonstrates that inspiratory muscle fatigue is present in patients following prolonged mechanical ventilation (ie, MVD for ≥ 48 h) and was not related to the use of NMBA in the current study.
Limitations

Due to the nature of the clinical population, it was not possible to assess the patient’s initial FRI. It is difficult to determine which patient will require prolonged mechanical ventilation, and thus it is not feasible to assess all patients prior to their ICU admission. However, studies in healthy individuals found no central or peripheral inspiratory muscle fatigue following resisted breathing. Similarly, stable patients with COPD have been shown to inspire against 30% of Pmax for up to 30 min without complication.

The follow-up period after discharge from the ICU was variable in the current study, as subjects were recruited when deemed clinically suitable to undergo the spirometry and muscle strength tests by their medical team. As the initial cause of ICU admission and clinical course was variable, so too was the follow-up period, with a mean duration of 7.2 days (SD, 3.5 days) post-ICU discharge, and this reflects the heterogeneous patient population. Despite this variability, there was no effect of the length of time since ICU discharge on FRI. This suggests that the effect of mechanical ventilation on FRI is not resolved following disconnection from the ventilator and may imply the need for specific intervention to improve the endurance capacity of the respiratory muscles.

It was not possible to use the same resistance for the fatigue protocol across subjects. Three subjects were not able to tolerate resistance equal to 30% of their Pmax, and the fatigue protocol was performed with no additional resistance. In these subjects, Pmax measurements were repeated at 30-s intervals, and fatigue developed as a result of the repeated Pmax efforts. Despite the use of two different loads, the change in FRI was not related to the applied load, and thus it appears that the minimal load of the equipment itself was sufficient in these subjects to demonstrate the reduction in inspiratory muscle endurance.

Several patients (n = 4) were unable to complete the 2-min inspiratory loading trial due to severe dyspnea. These subjects were generally comparable to those who completed the trial. Their inability to tolerate the inspiratory loading trial may have been related to severe inspiratory muscle weakness as two subjects had a Pmax of < 13 cm H2O. One subject was recruited following her third ICU admission, and increased morbidity associated with readmission to the ICU may have predisposed her to dyspnea. An RPE of > 7 was reported in one subject following resisted breathing at 50% of Pmax. This subject was the first to participate in the study, and the results led to the modification of the inspiratory loading to 30% of the initial Pmax.

A limitation of the analysis of long-term ventilation (≥ 7 days) is that few subjects required ventilation for this time (n = 4). This is representative of the proportion of patients who experienced prolonged mechanical ventilation in the ICU. These preliminary findings warrant further research in controlled groups of subjects with short-term and long-term ventilation to further investigate the effect of mechanical ventilation on FRI.

Clinical Implications

Patients who require prolonged mechanical ventilation (ie, MVD for ≥ 48 h) may have reduced endurance of the respiratory muscles following successful weaning, and this is more severe with increased periods of mechanical ventilation. Although it is not known whether prolonged mechanical ventilation is a cause of inspiratory muscle fatigue or an outcome, the presence of inspiratory muscle fatigue in this population may increase the risk of respiratory pump failure. Therefore, specific rehabilitation such as inspiratory muscle training may be indicated to improve inspiratory muscle endurance in this patient population.

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