Abdominal muscle fatigue after maximal ventilation in humans

DIMITRIS KYROUSSIS, GARY H. MILLS, MICHAEL I. POLKEY, CARL-HUGO HAMNEGARD, NICHOLAOS KOULOURIS, MALCOLM GREEN, AND JOHN MOXHAM
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Kyroussis, Dimitris, Gary H. Mills, Michael I. Polkey, Carl-Hugo Hamnegard, Nicholaos Koulouris, Malcolm Green, and John Moxham. Abdominal muscle fatigue after maximal ventilation in humans. J. Appl. Physiol. 81(4): 1477–1483, 1996.—Abdominal muscles are the principal muscles of active expiration. To investigate the possibility of abdominal muscle low-frequency fatigue after maximal ventilation in humans, we stimulated the nerve roots supplying the abdominal muscles. We used a magnetic stimulator (Magstim 200) powering a 90-mm circular coil and studied six normal subjects. To assess the optimum level of stimulation and posture, we stimulated at each intervertebral level between T7 and L1 in the prone, supine, and seated positions. At T10, we used increasing power outputs to assess the pressure-power relationship. Care was taken to avoid muscle potentiation. Twitch gastric pressure (Pga) was recorded with a balloon-tipped catheter. Mean (±SD) baseline twitch Pga measured with the subjects in the prone position at T10 was 23.5 ± 5.4 cmH2O. Within-occasion mean twitch Pga coefficient of variation was 4.6 ± 1.1%. Twitch Pga was measured with the subjects in the prone position with stimulation over T10 before and after 2 min of maximal isocapnic ventilation (MIV). Twenty minutes after MIV, mean twitch Pga fell by 17 ± 9.1% (P = 0.03) and remained low 90 min after MIV. We conclude that after maximal ventilation in humans there is a reduction of twitch Pga and, therefore, of low-frequency fatigue in abdominal muscles.

METHODS

Six well-trained members of our laboratory were recruited for the study. All were in good health and without respiratory disease. The study had Ethics Committee approval.

Pga and esophageal pressure (Pes) were measured with balloon-tipped catheters 110 cm in length (Morgan, Rainham, Kent, UK), positioned and tested in the standard manner (11, 15). Both catheters were connected to Validyne MP45-1 differential pressure transducers (range ± 200 cmH2O, Validyne, Northridge, CA). Transdiaphragmatic pressure (Pdi) was defined as the difference between Pga and Pes.

Maximal isocapnic ventilation (MIV) was performed with an apparatus previously described (16). Briefly, the subject inhaled from a 6-liter anesthetic bag supplied with an air-O2-CO2 mixture. Inhaled gas composition and end-tidal PCO2 were monitored with a Hewlett-Packard 78356A gas monitor (Hewlett-Packard, Waltham, MA). End-tidal PCO2 was maintained at 5.5 ± 0.5 kPa by adjustment of the inhaled CO2 concentration. The inhaled O2 concentration was kept at 22 ± 2%. Expiratory flow was measured with a Fleisch no. 4 pneumotachograph head (Fleisch, Lausanne, Switzerland) connected to a Mercury CS6 electrospirometer (Mercury Electronics, Glasgow, UK).

All signals were digitized via a 12-bit NB-M10-16 analog-to-digital converter (National Instruments, Austin, TX) and acquired onto a Macintosh Quadra 700 computer (Apple Computer, Cupertino, CA) running LabVIEW software (National Instruments, Austin, TX).

A Magstim 200 magnetic stimulator was used to power a 90-mm-diam magnetic coil (Magstim, Whitland, Dyfed, Wales, UK).

To assess whether the action potential evoked during magnetic stimulation of the abdominal muscles changes after MIV, we measured electromyographic (EMG) signals of the abdominal muscles in three subjects. We compared the peak-to-peak amplitude and the area of the action potentials of the three subjects before and after MIV.

To assess whether twitches at the T10 level stimulated the diaphragm, we simultaneously recorded abdominal muscle and diaphragm EMGs in one subject. One pair of electrodes was placed over the external oblique at the level of the umbilicus or over the rectus muscle at the same level. Ground electrodes were placed close to the recording electrodes. The resulting signals were passed via long leads to a Magstim Neurosign 100 preamplifier and amplifier and displayed via a combined pressure and EMG recording program based on LabView software with a recording frequency of 2 kHz. To
measure electrical activity of the diaphragm, we used an esophageal electrode catheter. The catheter was positioned at the point of maximal EMG activity during sniffing and was fixed at the nose by tape.

Protocol

In five subjects during a first session, we assessed 1) whether stimulation was supramaximal, 2) the effect of coil position at different levels between T7 and L1 on twitch Pga, 3) the effect of different postures (seated, prone, and supine) on twitch Pga, 4) the reproducibility of twitch Pga at each posture, and 5) the effect of twitch potentiation on twitch Pga.

In a second session on a different day, we studied the effect of maximal ventilation on twitch Pga in six subjects.

First session. Measurements were performed at relaxed end-expiratory pressures with the mouth closed. The subjects were a noseclip during stimulation and loosened their belts. Pes was marked on the screen to ensure that all stimulations were performed at constant end-expiratory Pes and, by implication, lung volume. The vertebral levels between T7 and L1 were marked on the skin with a pen. The coil was positioned at each level, and three stimulations were made. Then the coil was positioned at the T10 level, and the stimulator was charged to a range of predetermined percentages of maximal power output (10, 20, 30, 40, 50, 60, 70, 80, 90, 95, and 100%). Three stimulations were made at each power setting. The order of the power settings was randomized. At T10, 10 more stimulations were performed at 100% output. The above procedures were performed for the seated, prone, and supine postures. In the supine posture, the subjects were studied only at the T10 level because positioning of the coil at different levels was impossible without inducing abdominal muscle potentiation. At each posture before measurements were made, the subjects rested for 20 min to avoid the effect of abdominal muscle potentiation on twitch Pga. To assess the effect of potentiation on twitch Pga, the subjects performed maximal expiratory efforts against a closed airway for 5 s, followed by a twitch at the T10 level.

Second session. Stimulations were performed with the subjects lying prone on a bed. The coil center was positioned at the point where the T10 level was marked and secured with tape, and its contour was marked. Abdominal muscles serve to flex and rotate the trunk, and the subjects therefore rested for 20 min, during which time body movements were minimized to avoid twitch potentiation. After the rest period, 10 stimulations were performed that served as baseline data. The six subjects then performed MIV for 2 min. All of them were familiar with the MIV technique. They sat in a chair breathing through the MIV circuit trying to maintain the highest possible ventilation for as long as the run lasted. Throughout the run, the subjects were vigorously verbally encouraged. No specific instructions were given about tidal volume, duty cycle, or respiratory frequency. Pes, Pga, and expiratory flow were recorded throughout the run. After the run, the subjects lay prone, carefully avoiding any further movements; the coil was positioned in place and secured with tape; and 10 stimulations were performed 10, 20, 30, 60, and 90 min after MIV.

The Pga-time product of the abdominal muscles during expiration (PgaTP) was calculated for each breath as the product of mean pressure, expiratory time, and respiratory rate expressed over 1 min in centimeters of water times seconds per minute. The beginning and end of expiration were determined from the flow signal. Pga during expiration was integrated from a baseline level taken as the resting end-expiratory Pga before MIV.

Within-session twitch Pga reproducibility for each posture was assessed by the coefficient of variation (CV) of 10 stimulations at 100% power output over T10 for the prone, supine, and seated postures. Within-day reproducibility was assessed by the CV of 10 stimulations at 100% power output over T10 and the 3 stimulations over T10 when measuring twitch Pga at the levels between T7 and L1, 30 min later. We also measured the CV for twitch Pga for all the measurements of session 2 (100% power output) to assess the reproducibility of twitch Pga before and at each time interval after the MIV run.

Three subjects performed a sham run during which twitch Pga baseline measurements were followed by breathing quietly through the MIV circuit for 2 min, and then measurements were repeated after 20 min in the prone position.

In one subject, we followed the time course of twitch Pga recovery for 24 h.

Statistics were calculated with a paired (Wilcoxon signed rank test) nonparametric test (Statview 4.0, Abacus Concepts, Berkeley, CA). A level of P < 0.05 was taken as significant.

RESULTS

Coil Positioning

Small differences in twitch Pga were found when stimulations were performed at different levels between T7 and L1 for both the prone and seated postures (Fig. 1).

Effect of Posture and Potentiation

The mean twitch Pga was similar for the three postures for both the unpotentiated and potentiated twitches (Table 1). Maximal expiratory maneuvers against a closed airway greatly increased twitch Pga (Table 1).

Reproducibility

The reproducibility of twitch Pga for the prone and supine postures was similar, whereas in the seated posture reproducibility was less good. Within-occasion mean (±SD) CVs for the five subjects for the prone, supine, and seated postures were 5.7 ± 3.08, 6.8 ± 2.35, and 9.4 ± 5.04%, respectively. Same-day between-
occasion mean CVs for the prone and seated postures were 3.5 ± 1.81 and 12.2 ± 11.2%, respectively. Table 2 shows twitch Pga CVs for measurements before and 10, 20, 30, 60, and 90 min after MIV. Each CV value is derived from 10 twitch Pga measurements.

No difference in twitch Pga was detected before and after the sham run in three subjects. Mean baseline twitch Pga was 20 ± 1.2 cmH₂O, and 20 min after the sham run, it was 20.6 ± 1.7 cmH₂O.

A representative Pga trace during magnetic stimulation of the abdominal muscles is shown in Fig. 2. An EMG action potential of the abdominal muscles sampled with surface electrodes sited over the external oblique is shown. There was no action potential detected from the esophageal electrode, indicating that the diaphragm was not activated.

Supramaximality

Stimulation of the abdominal muscles was not supramaximal in any of the three postures. As the power output of the magnetic stimulator increased, a proportional increase in twitch Pga was observed. Figure 3 shows the mean twitch Pga in the five subjects during stimulations over T₁₀ in the prone posture plotted against the magnet's power output. Similar results were obtained for the seated and supine postures.

Effect of MIV on Twitch Pga

Twenty minutes after MIV, a fall in twitch Pga was observed in all six subjects; the mean reduction was 17 ± 9% (range 7–33%; P = 0.03; Fig. 4). Twitch Pga remained low for the 90-min period during which stimulations were performed (Fig. 4). Throughout these studies (session 2), the magnetic power output was 100%.

The time course of twitch Pga recovery over 24 h in one subject is shown in Fig. 5.

Table 1. Twitch Pga with stimulation at T₁₀

<table>
<thead>
<tr>
<th></th>
<th>Twitch Pga, cmH₂O</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Unpotentiated</td>
</tr>
<tr>
<td>Prone</td>
<td>19.7 ± 4.5</td>
</tr>
<tr>
<td>Supine</td>
<td>20.0 ± 5.7</td>
</tr>
<tr>
<td>Seated</td>
<td>19.7 ± 3.3</td>
</tr>
</tbody>
</table>

Values are means ± SD. Twitch Pga, gastric pressure during magnetic stimulation of abdominal muscles at 100% magnetic power output.

Table 2. Twitch Pga coefficient of variation before and after MIV

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>Baseline</th>
<th>10 Min</th>
<th>20 Min</th>
<th>30 Min</th>
<th>60 Min</th>
<th>90 Min</th>
<th>All Data</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>4.7</td>
<td>2.7</td>
<td>3.3</td>
<td>4.3</td>
<td>4.2</td>
<td>2.6</td>
<td>3.6</td>
</tr>
<tr>
<td>2</td>
<td>6.3</td>
<td>3.0</td>
<td>6.6</td>
<td>3.9</td>
<td>4.6</td>
<td>6.6</td>
<td>5.1</td>
</tr>
<tr>
<td>3</td>
<td>6.0</td>
<td>9.2</td>
<td>3.6</td>
<td>4.4</td>
<td>5.2</td>
<td>7.7</td>
<td>6.0</td>
</tr>
<tr>
<td>4</td>
<td>4.1</td>
<td>3.7</td>
<td>3.7</td>
<td>4.0</td>
<td>2.8</td>
<td>6.1</td>
<td>4.1</td>
</tr>
<tr>
<td>5</td>
<td>5.6</td>
<td>6.5</td>
<td>3.2</td>
<td>3.2</td>
<td>8.1</td>
<td>4.8</td>
<td>5.2</td>
</tr>
<tr>
<td>6</td>
<td>3.3</td>
<td>2.2</td>
<td>2.4</td>
<td>2.1</td>
<td>2.1</td>
<td>7.7</td>
<td>3.3</td>
</tr>
</tbody>
</table>

Means ± SD (n = 60) 5.0 ± 1.2 4.6 ± 2.8 3.8 ± 1.4 3.6 ± 0.9 4.5 ± 2.1 5.9 ± 2.0 4.6 ± 1.1 (n = 360)

n, No. of measurements. MIV, maximal isocapnic ventilation.

Fig. 2. Pga during magnetic stimulation of abdominal muscles at T₁₀ intervertebral level. An EMG action potential sampled from surface electrodes sited over external oblique is shown. A simultaneous recording from an esophageal electrode failed to show any electrical activity from diaphragm.

Effect of MIV on Ventilation and Pga<sub>TP</sub>

Mean ventilation for the six subjects at the beginning of MIV was 198 ± 12 l/min (range 218–183 l/min). Ventilation rapidly declined to a mean of 133 ± 11 l/min 1 min after the start of MIV. Therafter, ventilation showed little change and at the end of the run was 121 ± 71 l/min.

A marked decrease in Pga<sub>TP</sub> was observed in all subjects during the course of MIV (Fig. 6). Maximal Pga<sub>TP</sub> was assessed from the first 5 breaths and was compared with the Pga<sub>TP</sub> of the last 10 breaths. The fall
in mean $PGA_{TP}$ for the six subjects was $66 \pm 22\%$ ($P = 0.03$). $PGA_{TP}$ tended to plateau after the first minute of MIV (Fig. 7).

The action potential evoked from the external oblique during magnetic stimulation was very similar in amplitude and configuration before and after MIV in the three subjects. Mean EMG amplitude increased by 7%, and mean EMG area increased by 1%. A representative EMG trace from one subject is shown in Fig. 8.

The twitch $PGA$ fall 20 min after MIV correlated with the percent fall in $PGA_{TP}$ in each subject during the course of MIV ($r^2 = 0.69; P = 0.04$) and maximal $PGA_{TP}$ ($r^2 = 0.79; P = 0.02$; Table 3).

**DISCUSSION**

We have magnetically stimulated the nerve roots supplying the abdominal muscles and have shown that twitch $PGA$ decreases after MIV in normal subjects. This finding suggests that low-frequency fatigue occurs in the abdominal muscles under these circumstances.

Supramaximal stimulation of a muscle before and after a fatiguing protocol ensures that all axons of the nerves supplying the muscle are constantly stimulated, and, therefore, any changes of muscle contractility observed can be attributed to muscle fatigue. The stimulation of the abdominal muscles was submaximal as judged by the increase in pressure at increasing levels of magnetic power output. Nevertheless, we believe that stimulation of abdominal muscles was kept constant throughout the study. Action potentials evoked before and after MIV were similar in amplitude and configuration in the three subjects in whom EMGs were measured, indicating that neither transmission failure nor derecruitment of muscle fibers was the cause for the fall in twitch $PGA$. Thus the decrease in twitch $PGA$ was the result of contractile fatigue. Furthermore, measurements were reproducible, indicating that the same proportion of the abdominal muscles was acti-
vated at each stimulation. The mean twitch Pga CV of the six subjects, measured from all measurements performed before and after MIV, was \(<5\%\) and was similar for baseline measurements and for measurements after MIV. The stimulation power output was constantly at 100% throughout the study. The coil was taped to the subject’s back for as long as the study lasted with the exception of the MIV run. At this point, the subject was seated in a chair for a few minutes and then returned to the prone posture on the bed. The coil position had been marked, thereby allowing exact repositioning. The observation that the positioning of the coil at different levels between T7 and L1 did not result in any important changes in twitch Pga suggests that small changes in the coil positioning could not affect results. When three of the subjects underwent the same study procedures, but instead of breathing maximally through the MIV circuit, they breathed quietly for 2 min, we could not detect any twitch Pga difference before and after the sham run. We are therefore confident that muscle activation, although not maximal, remained constant throughout our studies.

Abdominal muscle length could affect twitch Pga because force generation is determined by the resting length of muscle. To avoid volume changes, stimulations were always performed against a closed airway and at the same end-expiratory lung volume as judged by Pes traces.

It is unlikely that during our study there were significant changes in abdominal muscle configuration. It has been previously shown that changes in lung volume have a very small effect on electromechanical coupling of the abdominal muscles or the linear dimensions of the abdominal surface (9). Furthermore, the volume change during MIV is small (17). Changes in posture could affect abdominal muscle length. Our subjects lay prone on a bed and remained still for as long as the study lasted.

When abdominal muscles contract, they produce a positive pressure acting on the abdominal wall, diaphragm, and lower rib cage. Change of diaphragm and

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>Maximal Pga Tp</th>
<th>%Pga Tp Fall</th>
<th>%Twitch Pga Fall</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>2,252</td>
<td>30</td>
<td>7</td>
</tr>
<tr>
<td>6</td>
<td>2,328</td>
<td>47</td>
<td>10</td>
</tr>
<tr>
<td>4</td>
<td>3,075</td>
<td>70</td>
<td>17</td>
</tr>
<tr>
<td>1</td>
<td>3,418</td>
<td>78</td>
<td>17</td>
</tr>
<tr>
<td>5</td>
<td>3,980</td>
<td>84</td>
<td>19</td>
</tr>
<tr>
<td>2</td>
<td>4,153</td>
<td>86</td>
<td>33</td>
</tr>
<tr>
<td>Mean±SD</td>
<td>3,201±85</td>
<td>66±22</td>
<td>17±9</td>
</tr>
</tbody>
</table>

Pga Tp, Pga-time product in cmH₂O x s x min⁻¹.

Fig. 7. A typical pattern of decline in Pga Tp during MIV in one subject. Note that a steep Pga Tp decline is followed by a plateau 60 s after start of maximal ventilation.

Fig. 8. Pga and action potential elicited by magnetic stimulation of abdominal muscles before (A) and 20 min after (B) MIV.

Table 3. Pga Tp and twitch Pga relationship
rib cage compliance with fatigue could affect twitch Pga. However, previous studies have demonstrated that there is no change in skeletal muscle (19) and chest wall (27) compliance with loading up to the point of fatigue.

We studied the possibility of direct diaphragm activation by the magnetic field during stimulation of the nerve roots of the abdominal muscles by simultaneously recording the electrical activity of the external oblique via surface electrodes and of the diaphragm via an esophageal electrode. A typical compound action potential was detected by the surface electrodes, whereas no action potential could be detected by the esophageal electrode. This indicates that the diaphragm is electrically silent during magnetic stimulation of abdominal muscles in the way described in this study.

The force developed by a muscle in response to stimulation is influenced by the recent contractile history. An increase in twitch tension after tetanic stimulation or voluntary contractions is termed twitch potentiation (24). This phenomenon has been observed in a wide variety of muscles including the diaphragm in humans (14, 26). We have previously shown that after a maximal voluntary contraction twitch Pdi elicited by magnetic stimulation of the phrenic nerves can increase by up to 72% (26). Recovery is usually complete 20 min after the voluntary contraction (26). This suggests that potentiation must be considered in any protocol that involves measurement of twitch pressure. Repeated muscle contractions during a fatigue protocol can induce twitch potentiation, and, therefore, twitch potentiation and fatigue can coexist (13, 20). This suggests that to accurately quantify low-frequency fatigue a period of ~20 min should elapse after a fatiguing protocol before using the twitch-stimulation technique. In the present study, we showed that abdominal muscles also exhibit the phenomenon of twitch potentiation. We observed a substantial increase in twitch Pga after maximal expiratory efforts. During the studies, care was taken to avoid twitch potentiation of the abdominal muscles, and our subjects relaxed for 20 min before baseline stimulations. Abdominal muscles are used to flex, rotate, and support the trunk. When subjects are seated, contraction of abdominal muscles of variable intensity takes place to support the body (22). Variability of twitch Pga was greater in the sitting posture, and this may have reflected variable twitch potentiation. We therefore chose the prone position when studying the effect of MIV on twitch Pga. The subjects lay still on a bed, thus avoiding any contraction of abdominal muscles.

The prone posture was preferred to the supine position for the MIV study because it was easier for the operator to position, secure, and check the coil position. Although there was a slight, but insignificant, increase in twitch Pga as the coil was positioned over the lower levels, we arbitrarily decided to use T10 for the study because it lies midway between T8 and L1. If a 90-mm magnetic coil were positioned with its center over T10, we considered that it could perhaps stimulate nerve roots between T8 and T12.

Measurement of MIV has been used to test ventilatory muscle function, with the duration varying between 12 s and 10 min. It has been shown that ventilation declines rapidly within the first 2 min of starting MIV, with little change between 2 and 10 min (5). The decline in ventilation is paralleled by a fall in pressure generation (17). Inspiratory muscle fatigue has been documented after MIV in terms of slowing the maximal relaxation rate of inspiratory muscles (12, 17) and a reduction in twitch Pdi (8). The documentation of abdominal muscle fatigue, both by slowing the abdominal muscle maximal relaxation rate (11) and, in the present study, by a fall in twitch Pga after MIV, indicates that abdominal muscle fatigue could contribute to the decline in performance observed during maximal ventilation. During maximal ventilation, both muscle groups are driven to their limits, each sharing a proportion of the work of breathing and eventually developing fatigue, which could then limit pressure generation and ventilation.

We detected a mean twitch Pga reduction of 17% 20 min after MIV. This is less than the mean fall detected in twitch Pdi under the same circumstances in a previous study in which Hamnegard et al. (8) observed a 25% reduction in diaphragm twitch pressure. This difference might reflect the differences in strength in relation to load of the two muscle groups during maximal ventilation. There was a wide range in the fall of twitch Pga between subjects (7–33% of baseline twitch Pga) after MIV. This difference between subjects could reflect different loads during MIV. Subjects who created higher PgaTP values during MIV had greater falls in PgaTP at the conclusion of MIV and substantial reductions in twitch Pga. Thus abdominal muscle low-frequency fatigue, detected by twitch Pga, was related to the magnitude of expiratory pressure generation and decline.

The twitch Pga reduction persisted for the 90 min over which measurements were made, and in one subject, recovery was substantial but not complete 24 h after the conclusion of MIV. A persistent impairment of muscle contractility is characteristic of low-frequency fatigue and has been described in previous studies of limb and respiratory muscles (4, 8, 16). The observation of incomplete twitch Pga recovery 24 h after the conclusion of MIV is in agreement with a study that followed the recovery of twitch Pdi in eight subjects over 24 h after a fatiguing protocol (13). The authors observed that 24 h after the exercise the recovery was not complete. Polkey et al. (18) have made a similar observation after the recovery of quadriceps twitches over a period of 36 h.

Inspiratory muscle fatigue has been extensively investigated in normal subjects. Because the work of breathing is mainly performed by the inspiratory muscles, this muscle group is likely to be exposed to severe loading and fatigue. Reduction in inspiratory muscle strength can be of substantial clinical importance and may play a role in respiratory failure. However, there is evidence that abdominal muscles, through a variety of mechanisms, are also functionally important when ventilation increases. These muscles vigorously contract to achieve expiration, reducing expiratory time
and thus contributing to increased breathing frequency. Previous studies (2) have demonstrated that expiratory muscles can act as accessory muscles of inspiration. Abdominal muscle contraction increases expiratory flow and reduces functional residual capacity, allowing the respiratory system to work at a lower volume (3). The elastic and gravitational energy stored during expiration by the action of abdominal muscles is subsequently released during inspiration, and, therefore, inspiratory muscle work is shared by the expiratory muscles (3). Furthermore, contraction of the abdominal muscles displaces the diaphragm into the thorax, lengthening its fibers and placing them on a more advantageous portion of their length-tension curve, thereby increasing their capacity to generate force (6, 7). In circumstances where the diaphragm and other inspiratory muscles are subjected to an excessive load or are fatigued, the role of expiratory muscles in contributing to ventilation and supporting inspiratory muscle function could be important.

The appreciation of the coordinated function of inspiratory and expiratory muscles when the demand placed on the respiratory system is increased and the finding that maximal ventilation results in fatigue of both muscle groups raise the question of whether abdominal muscle fatigue could be clinically relevant. After heavy exercise, diaphragmatic fatigue may occur (10). In such circumstances, abdominal muscle fatigue could also be a reasonable hypothesis because these muscles vigorously contract both to facilitate respiratory pump function and also to support, flex, and rotate the body. Moreover, abdominal muscle loading has been shown to contribute to an increased effort sensation (23). Intensification of dyspnea in circumstances where the abdominal muscles are heavily recruited could also be of clinical significance because it may contribute to exercise intolerance.

We conclude that MIV induces low-frequency fatigue of the abdominal muscles in normal subjects and that this may be a factor in limiting breathing capacity.

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