Effect of inspiratory muscle strength training on inspiratory motor drive and RREP early peak components

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Huang, Chien Hui, A. Daniel Martin, and Paul W. Davenport. Effect of inspiratory muscle strength training on inspiratory motor drive and RREP early peak components. J Appl Physiol 94: 462–468, 2003. First published September 20, 2002; 10.1152/japplphysiol.00364.2002.—This study investigated the effect of inspiratory muscle strength training (IMST) on inspiratory motor drive [mouth occlusion pressure at 0.1 s (P0.1)] and respiratory-related evoked potentials (RREP). It was hypothesized that, if IMST increased inspiratory muscle strength, inspiratory motor drive would decrease. If motor drive were related to the RREP, it was further hypothesized that an IMST-related decrease in drive would change RREP latency and/or amplitude. Twenty-three subjects received IMST at 75% of their maximal inspiratory pressure (PImax) with the use of a pressure threshold valve. IMST consisted of four sets of six breaths daily for 4 wk. P0.1 and the RREP were recorded before and after IMST. Post-training, PImax increased significantly by 36.0 ± 2.7%. P0.1 decreased significantly by 21.9 ± 5.2%. The increase in PImax was significantly correlated to the decrease in P0.1. RREP peaks P1a, Nc, P1, and N1 were identified pre- and post-IMST, and there was no difference in either amplitude or latency for those peaks. These results demonstrate that high-intensity IMST significantly increased PImax, decreased P0.1, but did not change the RREP.

maximal inspiratory pressure; mouth occlusion pressure at 0.1 s; mouth occlusion pressure; evoked potential; pressure threshold training; respiratory-related evoked potentials

INSPIRATORY MOTOR DRIVE, MEASURED by the pressure generated after the first 0.1 s of inspiration when the airway is briefly occluded [mouth occlusion pressure at 0.1 s (P0.1)], is significantly increased when inspiratory muscles are weakened by curarization in healthy subjects (4, 20). In patients with diseases that exhibit weak inspiratory muscle strength, P0.1 increases significantly (1, 13, 18, 37). It was further reported that P0.1 was lower for certain chronic obstructive pulmonary disease patients requiring mechanical ventilation who were successfully weaned, compared with those who were not weaned (31). These findings point to a relationship between decreased inspiratory muscle strength and increased inspiratory motor drive.

The combination of inspiratory motor drive generating the forces necessary for ventilation and the afferent feedback providing higher brain centers with information about the effect of the motor drive on ventilation is essential for the perception of respiratory events. Respiratory muscle afferents have been suggested to be one of the components of load perception (8). Increased P0.1 is often associated with increased respiratory sensitivity in patients (5, 13, 15, 33). In normal subjects, increased respiratory sensitivity was associated with increased motor drive induced by partial neuromuscular blockade of the respiratory muscles (4). Therefore, it appears that reduced respiratory muscle strength increases inspiratory motor drive, which in turn results in the increase of respiratory sensitivity. This change in respiratory sensitivity may be mediated by a change in the magnitude of the motor drive and/or the respiratory mechanoreceptor information related to the change in drive.

Respiratory muscles have the capacity to respond to both endurance and strength-training stimuli (26). Inspiratory muscle strength training (IMST) has been applied to both healthy subjects and patients to increase inspiratory muscle strength, often measured as the maximal inspiratory pressure (PImax). It has been reported that IMST increases inspiratory muscle strength and decreases inspiratory load sensation in normal subjects (21, 34) and patients with chronic obstructive pulmonary disease (19, 28, 29, 32). This suggests that changing inspiratory motor drive can influence respiratory sensation. However, none of these studies investigated the relationship between increased inspiratory muscle strength and inspiratory motor drive. The first hypothesis of the present study is that inspiratory pressure threshold training will increase IMST and decrease inspiratory motor drive as measured by P0.1.

Evoked potentials have been used in sensory systems to record the cortical neural activity elicited by a synchronous stimulation of afferents that project to specific cerebral cortical neurons (9). The respiratory-related evoked potential (RREP) is a cortical-evoked neural response elicited by respiratory mechanoreceptors (9). Early components of the RREP (P1a, N6, P1, and N1) refer to those peaks that occur within 100 ms

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poststimulus, which are thought to represent the initial sensory processing of inspiratory occlusion-activated mechanoreceptors (30, 41). A relationship between inspiratory motor drive and the RREP was reported in the initial RREP study by Davenport et al. (9). They found a correlation between P1 (the first positive peak) latency and P0.1. Davenport et al. (10) increased P0.1 with steady-state hypercapnia. The P0.1 increased 250%, the P1 latency decreased 15–20%, and the P1 amplitude did not change. Their results suggested a relationship between somatosensory RREP latency and the inspiratory motor drive as measured by P0.1. The relationship between ventilatory mechanics and the RREP was further supported by reports that both P1 amplitude and magnitude estimation of inspiratory load were correlated to the magnitude of the load (22, 40) and transdiaphragmatic pressure (23).

Based on these results, it is possible that a change in inspiratory motor drive in steady-state normocapnia elicited by an increase in inspiratory muscle strength with IMST may lead to a change in P1 latency and/or amplitude. The effect of decreasing inspiratory motor drive with IMST on the RREP has not been investigated. Therefore, the second purpose of the present research was to investigate the effect of the hypothesized decrease in inspiratory drive with IMST on the amplitude and latency of the early components of the RREP. It was hypothesized that IMST would increase the latency and decrease P1 amplitude.

METHODS

This study was performed on 23 normal subjects. Subjects were classified as normal with no history of cardiorespiratory disease, no history of smoking, and no evidence of present major or minor illness. All of the subjects were naive to inspiratory strength training. This study was reviewed and approved by the University of Florida Institutional Review Board. The study was explained to the subjects, and written informed consent was obtained before enrollment into the study. All of the subjects completed 4 wk of IMST. Demographic characteristics of the subjects are presented in Table 1.

Pulmonary function test. The subjects were seated upright in a comfortable chair. Spirometry testing conformed to American Thoracic Society standard, and data were collected by a computerized spirometer (Jaeger Toennies, Medizintechnikmit System, version 4.5). Pulmonary function tests of forced vital capacity and forced expiratory volume in 1 s were recorded. A forced expiratory volume in 1 s >70% predicted was required to continue in the experiment.

Inspiratory muscle strength measurement. Inspiratory muscle strength was measured at the mouth by an electronic pressure manometer (Micro MPM, Micro Medical). Pimax indirectly reflects inspiratory muscle strength, defined as the greatest negative pressure obtained at the mouth and sustained for at least 1 s while performing a maximal inspiratory effort (3). Subjects were seated with nose clips on. After exhaling to residual volume, subjects placed their lips around a mouthpiece and inspired as forcefully as possible. Repeated measurements were taken at least five times, with a 60- to 120-s rest between trials, until three measurements within 10% variation were attained. The average of these three measurements was recorded as subjects’ Pimax.

P0.1. The P0.1 after the initiation of inspiration was measured as described by Whitelaw and coworkers (42, 43). The occlusion valve was closed during expiration, resulting in occlusion of the following inspiration. The occlusion was presented every two to six respiratory cycles. P0.1 was measured at least five times, and the average of these measurements was calculated.

RREP. All RREP experiments were done in a sound-insulated room, separating the subject from experimenter. Subjects were studied seated, with the back, neck, and head comfortably supported. The subject’s chin was stabilized, and an electrode cap with integral tin electrodes was placed (Quik-Cap, Neuromedical Supplies, Sterling, VA) on their head to record scalp EEG activity. The electrode positions were based on the International 10/20 system.

A 12-channel EEG electrode montage was recorded in the following scalp positions: F3, Fz, F4, C3, Cz, C4, P3, Pz, and P4 referenced to the joined ear lobes. Sculp and electrode contact was made by the application of electroconducting paste. Tin ear-clip electrodes were places on both ears. Vertical electrooculogram activity was monitored with bipolar electrodes placed on the upper and outer canthus of the right eye. The impedance level for each electrode was checked and maintained <5 kΩ. The electrode cap was connected to an EEG system (model 12 neurodata acquisition system, Grass Instruments, West Warwick, RI). The EEG activity was band-pass filtered (0.3 Hz–1 kHz), amplified, and led into an on-line signal-averaging computer system (Cambridge Electronic Design, Cambridge, UK). The EEG activity was recorded and monitored with an oscilloscope on-line during data collection (511A; Tektronix, Beaverton, OR).

Subjects were studied semireclined, breathing through a mouthpiece and non-rebreathing valve, with a nose clip in place. Care was taken to suspend the valve to minimize facial muscle activity. The mouth pressure (Pm) signal was recorded by a differential pressure transducer (model MP-45, Validyne Engineering), digitized, and led into the on-line signal-averaging computer system. The inspiratory port of the non-rebreathing valve was connected to a pneumotachograph and occlusion valve. Throughout the RREP experiment, subjects watched a videotape. A transistor-transistor logic pulse generated by the inspiratory occlusion valve controller triggered the collection of 50 ms of pretrigger and 950 ms of posttrigger EEG and Pm data. The duration of the occlusion was ~350 ms. Individual 1,000-ms epochs of EEG, electrooculogram, and Pm data were collected and stored in computer memory. Each occlusion was separated by two to six unoccluded breaths. There were two RREP trials: 1) an inspiratory occlusion trial that consisted of 100 occlusion presentations and 2) a control trial in which a side port was open to allow unoccluded inspiration when the occlusion valve was activated.

Each of the recorded epochs of the 100 presentations were stored on computer disk for off-line averaging and analysis. Individual presentations were recalled from the computer-

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**Table 1. Demographic characteristics of subjects**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td>10 women, 13 men</td>
</tr>
<tr>
<td>Age, yr</td>
<td>28.8 ± 2.3</td>
</tr>
<tr>
<td>Height, cm</td>
<td>170.3 ± 1.5</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>72.7 ± 3.2</td>
</tr>
<tr>
<td>FEV1, % predicted value</td>
<td>95.8 ± 2.5</td>
</tr>
<tr>
<td>FVC, % predicted value</td>
<td>94.3 ± 2.5</td>
</tr>
</tbody>
</table>

Values are means ± SE. FEV1, forced expiration volume in 1 s. FVC, forced vital capacity.
stored file of the experimental trial and displayed. The averaging method was previously reported from this laboratory (6, 7). The nomenclature for the peaks was based on previous reports (9).

The presence, latency, and amplitude of the four early peak components, P1a, Nf, P1, and N1, in the averaged occlusion trials were determined for each scalp electrode site. Peak latencies were measured as the time from onset of the occlusion to the RREP peak. The zero-peak amplitude is defined as the voltage at the peak. The latencies and amplitudes of P1a, Nf, P1, and N1 peaks were determined for each individual subject. P1a was defined as a positive peak occurring 20–40 ms after the onset of the occlusion. Nf was defined as the negative peak occurring between 40 and 60 ms after the occlusion. P1 was defined as the positive peak occurring 50–70 ms after the onset of occlusion. N1 was defined as the negative peak occurring between 90 and 130 ms after the onset of occlusion.

**IMST protocol.** The subjects received 4 wk of IMST. The training was conducted 5 days/wk, Monday through Friday, in the laboratory. The training was performed individually under supervision by the same experimenter through the whole training period. Subjects underwent a 2-day introductory period, training for the first day at 30% of Pimax followed by the second day at 60% of Pimax. On the third day of training onward, training intensity was set at 75% of Pimax. Each subject used a custom-built inspiratory muscle trainer, a pressure-threshold device, which provided a pressure threshold load to inspiration between 14 and 160 cmH2O. Each daily IMST session consisted of four sets of six training breaths per set. An inspiratory training breath lasted for 1–3 s of sustained inspiratory effort through the inspiratory training device, and each set was separated by 1–2 min of quiet breathing. It required ~10 min to complete the daily IMST. Every Monday, the experimenter measured the subjects’ Pmax and adjusted the trainer to 75% of the new Pmax.

**Statistical analysis.** One-way repeated-measures ANOVA was used to analyze the difference in Pmax to detect the effect of training periods. Two-way repeated-measures ANOVA was used to analyze the effect of IMST and effect of scalp position on early components of RREP. Pearson correlation was used to compare the relationship between Pmax and P0.1. A paired t-test was used to compare the difference in P0.1. A paired t-test was also used to compare the difference in RREP peak latency pre- and post-IMST. Significance level was set at P < 0.05.

**RESULTS**

IMST resulted in a progressive increase in Pmax in all subjects from a mean value of 97.8 ± 3.81 (SE) to 132.2 ± 4.5 cmH2O, which represented a 36 ± 2.71% increase (Fig. 1). Pmax displayed a significant training period effect with each weekly Pmax measurement significantly greater than pretraining Pmax [F(4,88) = 103.2, P < 0.001]. Tukey’s multiple comparison also showed that every weekly Pmax pairwise comparison was significantly different, except for the pair comparison between the 3rd and 4th wk.

Inspiratory drive, measured as P0.1, was significantly decreased in response to IMST from a mean value of 1.53 ± 0.14 cmH2O before training to 1.19 ± 0.11 cmH2O after training, which represented a 21.9 ± 5.2% decrease (P < 0.01). Pearson product-moment coefficient of correlation showed that a negative correlation existed between Pmax and P0.1 (correlation coefficient = –0.44, P < 0.01).

The relationship between increased Pmax and decreased P0.1 after IMST is shown in Fig. 2. The change in Pmax (post-IMST Pmax − pre-IMST Pmax) was significantly correlated with the change in P0.1. The correlation coefficient was −0.46 (P < 0.01).

A constant pattern of four RREP peaks, P1a, Nf, P1, and N1, was identified. The mean peak latencies of early components of the RREP are shown in Table 2. The mean peak latencies of the four RREP components pre-IMST were as follows: P1a = 25.88 ± 1.33, Nf = 43.32 ± 1.38, P1 = 58.96 ± 1.64, and N1 = 102.37 ± 2.41 ms. The mean peak latencies of the four RREP components post-IMST were as follows: P1a = 26.43 ± 1.20, Nf = 42.86 ± 1.36, P1 = 57.37 ± 2.39, and N1 = 96.52 ± 2.48 ms. No significant difference between pre-IMST and post-IMST mean peak latency in any early component was found.

**Fig. 1.** Maximal inspiratory pressure (Pmax) increases over the training period. Values are means ± SE. *P max significantly increased compared with the pretraining Pmax (P < 0.001).

**Fig. 2.** The difference in mouth occlusion pressure at 0.1 s (P0.1; cmH2O) and the difference in Pmax (cmH2O). Values are shown as the absolute value from post-inspiratory muscle strength training (IMST) – pre-IMST. An inverse relationship between the difference in P0.1 and the difference in Pmax was found after IMST (P < 0.05).
The mean peak amplitudes of the RREP for scalp positions are shown in Table 3. P1a and Nf peaks were found in scalp position F3, F4, C3, C4, C6, and C7. P1 and N1 were found in scalp position C3, C4, C6, C7, P3, and P4. No differences in amplitudes were found between pre-IMST and post-IMST.

P1 mean peak amplitudes displayed neither scalp position effect nor IMST effect. P1 and N1 were found to be greatest in the parietal area, which was in scalp positions F3 and F4, P1 was found to have the greatest amplitude in the somatosensory area, which was in scalp positions F3 and F4, P1a was found to have the greatest amplitude in the parietal area, which was in scalp positions F3 and F4. P1a was found to have the greatest amplitude in the frontal area, which was in scalp positions F3 and F4. Tukey's multiple comparisons showed that Nf was found to be greatest in the frontal area, which was in scalp positions F3 and F4, P1 was found to be greatest in the parietal area, which was in scalp positions P3 and P4, N1 was found to be greatest in the frontal area, which was in scalp positions F3 and F4, P1 was found to be greatest in the parietal area, which was in scalp positions F3 and F4, P1a was found to have the greatest amplitude in the somatosensory area, which was in scalp positions C3, C4, C6, and C7; however, the difference was not significant.

DISCUSSION

The result of this study showed that 4 wk of high-intensity and low-repetition IMST increased Pimax significantly and was accompanied by decreased P0.1. IMST improved the subjects' maximal pressure generation ability, and there was a corresponding decrease in inspiratory motor drive.

The present study used 75% Pmax, 24 breaths/day for 4 wk, and resulted in a 36% increase in Pmax, which was greater than that in other training protocols (14, 19, 25–28, 39). Most previous studies (14, 19, 25–28, 39) used 30% Pmax as training intensity for inspiratory strength training and had subjects train for 6–18 wk.

According to Faulkner (12), overload, specificity, and reversibility are three principles in designing training regimes to obtain a desired training response. Leith and Bradely (26) concluded that these principles are important in respiratory muscle training. A more recent study by Tzelepis and colleagues (39) that compared the effect of high-pressure (force) and high-flow (velocity) type IMST confirmed these principles. The authors suggested that training protocols characterized by generating pressure or flow will specifically increase maximal pressure or maximal flow. The pressure threshold valve utilized in the present study requires the subjects to generate a pressure to open the valve before airflow occurred. The training protocol in the present study provided an effective and efficient stimulus for increasing maximal pressure generation ability.

Several mechanisms could account for the increases in Pmax. Changes in muscle length can alter strength. In the present study, Pmax was measured after a maximal exhalation. It is not likely that diaphragm muscle length shortened in response to IMST in these normal subjects. To minimize a learning effect, Pmax was measured multiple times until the subjects consistently performed three reliable tests within 10% variation. It is also possible that IMST induced inspiratory muscle hypertrophy. However, a training period of 4 wk is probably insufficient to elicit significant change of muscular hypertrophy or distribution of fiber type. An animal study showed that 8 wk of inspiratory resistive loading induced a hypertrophy of type II fibers in the

Table 2. Pre-IMST and post-IMST latency of early respiratory-related evoked potential components

<table>
<thead>
<tr>
<th>Latency</th>
<th>Pre-IMST</th>
<th>Post-IMST</th>
</tr>
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<tbody>
<tr>
<td>P1a</td>
<td>25.88 ± 1.33</td>
<td>26.43 ± 1.20</td>
</tr>
<tr>
<td>Nf</td>
<td>43.32 ± 1.38</td>
<td>42.87 ± 1.36</td>
</tr>
<tr>
<td>P1</td>
<td>58.96 ± 1.64</td>
<td>57.37 ± 2.39</td>
</tr>
<tr>
<td>N1</td>
<td>102.37 ± 2.41</td>
<td>96.52 ± 2.48</td>
</tr>
</tbody>
</table>

Values are means ± SE in ms. IMST, inspiratory muscle strength training.

Table 3. Pre-IMST and post-IMST peak amplitudes of early components of respiratory-related evoked potential in scalp positions

<table>
<thead>
<tr>
<th>Electrode Position</th>
<th>Pre-IMST</th>
<th>Post-IMST</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>P1a</td>
<td>Nf</td>
</tr>
<tr>
<td>F3</td>
<td>0.912 ± 0.34</td>
<td>−2.946 ± 0.61</td>
</tr>
<tr>
<td>F4</td>
<td>1.034 ± 0.39</td>
<td>−2.470 ± 0.64</td>
</tr>
<tr>
<td>C3</td>
<td>1.638 ± 0.35</td>
<td>−0.556 ± 0.49</td>
</tr>
<tr>
<td>C4</td>
<td>1.377 ± 0.38</td>
<td>−0.615 ± 0.51</td>
</tr>
<tr>
<td>C6</td>
<td>1.641 ± 0.34</td>
<td>−0.890 ± 0.54</td>
</tr>
<tr>
<td>C7</td>
<td>0.691 ± 0.73</td>
<td>−0.721 ± 0.71</td>
</tr>
<tr>
<td>P1</td>
<td>1.006 ± 0.71</td>
<td>−2.609 ± 0.74</td>
</tr>
<tr>
<td>P2</td>
<td>1.900 ± 1.13</td>
<td>−3.072 ± 0.78</td>
</tr>
<tr>
<td>P3</td>
<td>2.609 ± 0.88</td>
<td>−2.146 ± 0.57</td>
</tr>
<tr>
<td>P4</td>
<td>2.701 ± 1.22</td>
<td>−1.492 ± 0.75</td>
</tr>
<tr>
<td>P5</td>
<td>3.143 ± 1.18</td>
<td>−1.158 ± 0.54</td>
</tr>
<tr>
<td>P6</td>
<td>3.654 ± 1.14</td>
<td>−1.212 ± 0.50</td>
</tr>
</tbody>
</table>

Values are means ± SE in µV.
diaphragm (2). This is in line with data obtained from quadriplegic humans, in whom diaphragm thickness was increased after 2.5–4.5 mo of IMST (24). Thus muscle hypertrophy after 4 wk of IMST is unlikely to completely account for the increase in P_{max}.

Neural adaptations can occur in the initial phases of strength training and cause a measurable difference in muscle strength (36). Increases in strength can be achieved without morphological changes in muscle but not without neural adaptation (11). Significant increase in P_{max} was found in the 2nd wk of training and continued to increase throughout the remaining 2 wk. It is believed that neural adaptation occurred within the first 2–3 wk of training, which is the main reason for the weekly significant increase in P_{max}. Muscle hypertrophy-related increases change more slowly and may have become evident during the 3rd and 4th wk of training, as evidenced by the slowing of the rate of increase in P_{max}.

The increase in P_{max} resulted in a decreased P_{0.1}. Post-IMST, P_{0.1} was significantly decreased, and the decrease in P_{0.1} was correlated with the increase in P_{max}. Most previous studies demonstrated a close relationship between weak muscle strength and increased P_{0.1} (1, 17, 18, 31). The present study extends and supports the relationship between inspiratory muscle strength and inspiratory motor drive by demonstrating a significant decrease in P_{0.1} with IMST. It is unlikely that the decrease in P_{0.1} is due to physiological variability or a learning effect over time. Mean values from at least five measurements were calculated before and after IMST. Garcia-Rio and colleagues (16) showed that baseline P_{0.1} did not change between two visits of 2 mo apart in normal subjects. Thus the results support the first hypothesis of this study. The decreased P_{0.1} observed in the present study is best explained by an increase in inspiratory muscle strength measures due to an initial neural adaptation and subsequent slow-increase inspiratory muscle development.

The present study is the first to record RREP in response to IMST. In the present results, P_{1a}, N_{f}, P_{1}, and N_{1} have been recognized in a similar pattern, as previous reported in the literature (6, 7, 10, 22, 23, 38). The early components of the RREP were similar to previous reports (6, 30), which found that P_{1} was greatest at scalp sites overlying the somatosensory cortex, whereas N_{f} was found to be greatest in the frontal cortex. However, no IMST effect was found in either amplitude or latency in any peak of the RREP. This showed that 4-wk IMST, which increased P_{max} and decreased P_{0.1}, had no significant effect on the early peaks of the RREP. The result suggests that motor drive may not be the dominant factor mediating the RREP.

Previous topographical analysis suggested a cortical generator located possibly in the motor or premotor cortex precentrally for N_{f} and in the somatosensory cortex postcentrally for P_{1} (30). Both N_{f} and P_{1a} in the present study were found in similar locations. Webster and Colrain (41) found no effect of either attention or occlusion duration on either P_{1a} or N_{f}. They concluded that P_{1a} was unlikely to be of cortical origin. Therefore, P_{1a} and N_{f} may be unrelated to drive with midinspiration occlusion, because of the fact that the inspiratory drive is executed when occlusion is presented and is not stimulus magnitude dependent.

N_{1} has been consistently produced by respiratory stimuli (6, 7, 10, 22, 23, 38). In the present results, N_{1} was found greatest in the temporal regions, similar to the previous finding (6). With a latency of ~100 ms, N_{1} is on the border between early (<100 ms) and late (>100 ms) components. N_{1} has been reported to be affected by cognitive factors (41), which were found in the late components but not in the early components. Webster and Colrain (41) found attention resulted in augmentation of the N_{1} component, which was not found in early components N_{f} and P_{1}. In the present study, N_{1} was found 102 ms pre-IMST and 97 ms post-IMST and showed no significant difference in response to IMST. The attention conditions were the same pre- and post-IMST, so there appears to be no effect of IMST-related changes in inspiratory motor drive on the N_{1} peak.

There was a trend for the P_{1} peak amplitude to decrease after IMST, which, however, did not reach statistical significance. The nonsignificant change in the amplitude of the early RREP components can probably be explained by the occlusion stimulation. Occlusion is a “maximal stimulation” in terms of load magnitude. A small load activates fewer receptors than a large load, and occlusion activates the maximal number of receptors. The P_{1} peak amplitude increases with increases in the resistive load magnitude (22, 23, 40). One possible relationship between P_{1} amplitude and a decreased drive may be a reduced stimulation of receptors when a reduced motor “effort” occurs with lower motor drive, thus producing the trend toward reduced P_{1} amplitude. However, the nonsignificant effect of IMST found in P_{1} amplitude indicates that the post-training occlusions produced a sufficiently similar P_{1} amplitude effect, a similar magnitude of cortical activation in response to this maximal inspiratory load stimulation, for the decrease to fail to reach statistical significance.

RREP P_{1a} latency has been reported to correlate with P_{0.1} (35). P_{1} latency decreased with increased resting P_{0.1} with occlusions presented at the onset of inspiration. Revelette and Davenport (35) showed that increased inspiratory drive with steady-state hypercapnia resulted in a 250% increase in P_{0.1}, a decreased P_{1} latency, and no change in amplitude with midinspiratory occlusions. They did not report results for the other early peaks (N_{f} or N_{1}) of the RREP. These reports suggest that the RREP P_{1} peak latency may be affected by a large transient increase in inspiratory motor drive. In the present study, drive decreased, and the change in P_{0.1} was much smaller than the transient increase reported for hypercapnia (35). The decreased P_{0.1} in the present study did not result in a change in P_{1} latency. Thus the results of this study do not support the second hypothesis: a decrease in resting in-
spiratory motor drive did not result in a change in RREP early peak latency or amplitude.

In conclusion, the present results demonstrated that 4 wk of high-intensity and low-repetition IMST significantly increases PImax, resulting in a corresponding decrease in P0.1. A negative correlation was found between PImax and P0.1, suggesting an inverse relationship between inspiratory motor drive and PImax. This study also showed that the decrease in P0.1 was correlated with the increase in IMST as measured by the RREP. However, with increased PImax and decreased P0.1, neither peak amplitude nor latency of the early peaks of the RREP changed in response to IMST. This suggests that peripheral respiratory muscle strength level can modify the respiratory central motor drive, without a change in the RREP.

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