Putative protective effect of inspiratory threshold loading against exercise-induced supraspinal diaphragm fatigue

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J Appl Physiol 98: 991–998, 2005. First published October 15, 2004; doi:10.1152/japplphysiol.00528.2004.—The present investigation was intended to assess the consequences of an inspiratory load on the diaphragm central component of fatigue during exercise. We recorded the motor potential evoked (MEP) by transcranial magnetic stimulation of the motor cortex in 10 subjects. The diaphragm and rectus femoris were studied before and 10, 20, and 40 min after two 16-min cycling exercise (E) trials requiring 55% of maximal oxygen uptake: 1) one with an inspiratory threshold load (E + ITL), corresponding to 10% of maximal inspiratory pressure; and 2) the other without the load (E). Dyspnea, heart rate, electromyographic activity of the sternocleidomastoid, and diaphragm work were significantly higher in E + ITL than in E. Neither trial affected the response to phrenic motor stimulation, which was performed 15 and 25 min postexercise, or the maximal inspiratory pressure (116 and 120 cmH2O before E and E + ITL, respectively, and 110 and 114 cmH2O at 30 min postexercise). Whereas the amplitude of the diaphragm MEP was unaffected by E + ITL (+2.1 ± 29.4%), a significant decrease was observed 10 min after E compared with baseline (−37.1 ± 22.3%) and compared with E + ITL. The MEP amplitude of rectus femoris remained unchanged with E and E + ITL. The recruitment of synergistic agonists during E + ITL may have normalized the major ventilatory stress and reset up the excitability of the diaphragm pathway.

respiratory muscles; transcranial magnetic stimulation; motor cortex

FATIGUE DEVELOPS WHEN STRIATED skeletal muscles are confronted with loads that challenge their strength and endurance, involving both peripheral (downstream of the neuromuscular junction) and central mechanisms (see Ref. 13 for a review). Central fatigue is common in humans (7, 9, 33) and is viewed by some as a mechanism to protect against exercise-induced muscle damage or exercise-induced homeostatic failure (13).

As with other skeletal muscles, inspiratory muscles are susceptible to fatigue, both in response to specific loading (16) and after exhaustive endurance exercise. For example, highly fit normal volunteers exhibit decreased static inspiratory pressures after prolonged or extreme whole body exercise [e.g., a triathlon (17) or marathon (20)]. Because of the volitional nature of static inspiratory pressure maneuvers, the question of the central or peripheral nature of the mechanisms involved has been left open. Johnson et al. (19) found convincing evidence of contractile alterations (peripheral fatigue) when they compared the results of bilateral supramaximal phrenic nerve stimulation before and after exercise-induced diaphragm fatigue in healthy humans. This does not, however, exclude a central component. Indeed, Vérin et al. (34) demonstrated supraspinal diaphragm fatigue following incremental treadmill exercise. In this study, the depression of the diaphragmatic motor potentials evoked by transcranial magnetic stimulation was more pronounced and slower to recover than that of the quadriceps.

During CO2-stimulated ventilation (38) or exercise, plastic changes in respiratory muscle recruitment occur. The contribution of the diaphragm tends to decrease, with the muscle acting increasingly as a flow generator rather than a pressure generator (2). The contribution of extradiaphragm inspiratory and expiratory muscles to ventilation augments so as to maintain the global efficacy of the ventilatory pump. This is true when ventilatory loading starts with fresh muscles (19) and is even more evident when the diaphragm has been previously fatigued (30). These adaptive strategies are central in nature and may have a protective function for the diaphragm. The present study was designed to address a part of this issue in healthy volunteers. The specific aims were to determine 1) whether moderate cycling exercise leads to a depression in motor-evoked potentials (MEP) and 2) how an imposed inspiratory threshold load (ITL) [known to provoke a strong recruitment of extradiaphragm inspiratory muscles (39)] would influence this phenomenon. We hypothesized that the diaphragm responses to transcranial magnetic stimulation after exercise would be dependent on whether the exercise was performed with or without ITL.

METHODS

Participants. Ten healthy volunteers (5 men, 5 women; age 21–33 yr; body mass index 18.7 to 25.8 kg/m2; all but one righthanded), free of any disease or significant medical history, were studied. Their maximal oxygen uptake (VO2max) ranged from 32.0 to 56.9 ml·min−1·kg−1 (see Table 1). They were instructed to maintain their usual level of physical activity during the study period and not to modify their caffeine or nicotine consumption. They refrained from alcohol consumption, sedative intake, and heavy exercise during the experimental period.
adjusted to open at an inspiratory pressure of \(\sim 10\%\) of \(P_{\text{Imax}}\).

The inspiratory load was added 1 min after the beginning of exercise. Expiration remained free throughout testing. Mouth pressure and flow were continuously recorded during both trials from the setup of the inspiratory load.

While exercising, the subjects were asked to rate their dyspnea every 90 s on a visual scale.

No instructions were given to the subjects regarding breathing pattern.

**Muscle activity during exercise.** The electromyographic (EMG) activity of the sternocleidomastoid muscles (SCM) and transdiaphragmatic pressures were monitored continuously during the two trials (E and E + ITL) on the six subjects numbered 5 to 10 in Table 1.

Spontaneous EMG activity of the SCM was obtained by surface recording using pairs of disposable silver cup electrodes. These were placed over the midpoint of the muscle belly (4). The raw EMG signals were full-wave rectified and integrated with time of burst to provide an integrated EMG activity index. During the 16-min cycling bout, the subjects were asked to maintain their posture on the ergometer and any part of the signal containing interference caused by head movement was removed.

The transdiaphragmatic pressure was determined as the difference between gastric and esophageal pressures continuously recorded over the two exercise trials. The catheter tip pressure transducer (CTO-2, Gaeltec, Scotland, UK) was gently positioned in the esophagus and stomach after local anesthesia of the nasal passage with xylocaine spray (xylocaine 5% nebulator, Astra). Transdiaphragmatic pressure deflections were integrated over time and served to reflect the work of the diaphragm.

**Transcranial magnetic stimulation.** The responses to transcranial magnetic stimulation were studied in all subjects, who were seated comfortably in a quiet room, in a relaxed state.

Surface recordings of the diaphragm and rectus femoris EMGs were obtained using pairs of disposable silver cup electrodes. The right costal diaphragmatic activity was recorded with active electrodes sprayed (xylocaine 5% nebulizer, Astra). Transdiaphragmatic pressure deflections were integrated over time and served to reflect the work of the diaphragm.

**Exercise protocol.** Each subject performed two exercise trials: one with an ITL added (E + ITL) and one without (E). The runs were performed in random order, at a minimal interval of 2 days and at the same time of the day. Each trial consisted of a 5-min warm-up at 50% of the exercising load, immediately followed by a 16-min cycling bout. The exercising load required 55% of \(V_{\text{O}2\text{max}}\) and was below the ventilatory anaerobic threshold determined according to Wasserman et al. (37).

Subjects were instructed to cycle between 60 and 70 rpm. Throughout the tests, gases were analyzed by breath by using the automated system described above.

Inspiratory loading was achieved during the E + ITL runs by connecting an inspiratory threshold valve (threshold IMT, Respirationics, Germany) to the inspiratory side of a two-way breathing valve (type 2700, Hans Rudolph) connected to the face mask. The valve was

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**Table 1. Individual characteristics of the subjects**

| Subject No. | Gender (M/F) | Age, yr | Height, cm | Weight, kg | MAF, W | \(V_{\text{O}2\text{max}}\), ml/min \(\cdot k\text{g}^{-1}\) | \(P_{\text{Imax}}, \text{cmH}_{2}\text{O}\) |
|-------------|-------------|--------|------------|-----------|-------|-----------------|------------------|
| 1           | F           | 30     | 168        | 65        | 225   | 38.0            | 135              |
| 2           | M           | 30     | 175        | 79        | 350   | 56.9            | 146              |
| 3           | F           | 33     | 169        | 59        | 175   | 36.0            | 105              |
| 4           | M           | 24     | 184        | 81        | 300   | 43.5            | 202              |
| 5           | M           | 25     | 182        | 62        | 290   | 56.3            | 175              |
| 6           | M           | 27     | 180        | 77        | 350   | 52.0            | 159              |
| 7           | F           | 22     | 170        | 55        | 200   | 36.9            | 100              |
| 8           | F           | 22     | 176        | 66        | 275   | 35.4            | 135              |
| 9           | F           | 26     | 163        | 60        | 200   | 32.0            | 112              |
| 10          | M           | 21     | 185        | 79        | 350   | 46.6            | 132              |
| Mean        |             | 26     | 175        | 68        | 276   | 43.4            | 140              |
| SD          |             | 4      | 7          | 9         | 66    | 9.1             | 32               |

M, male; F, female; MAF, maximal aerobic power, defined as the highest power the subject was able to maintain for 1 min during the incremental test; \(V_{\text{O}2\text{max}}\), maximal oxygen uptake; \(P_{\text{Imax}},\) maximal inspiratory pressure.

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24 h preceding the tests. Of note, an 11th subject who had been recruited dropped out of the study because he could not tolerate transcranial magnetic stimulation.

The protocol had been approved by the appropriate ethics committee, and all subjects gave their informed, written consent for the study.

**Preliminary evaluation.** At the first visit, each subject underwent a physical examination and a 12-lead ECG. \(V_{\text{O}2\text{max}}\) was measured by using an electronically braked cycle ergometer (Ergometrics 800, Ergoline, Germany) as follows. After an 8-min warm-up at a heart rate of \(\sim 110\) beats/min, the load was increased every minute by 25 W until the subject was unable to continue, despite vigorous coaching. During this procedure, the subjects breathed through a face mask (Hans Rudolph, Kansas City, MO) connected to a pneumotachograph (type 2700, Hans Rudolph, Kansas City, MO) connected to a pneumotachograph (type 2700, Hans Rudolph) continuously and averaged over every 15-s period. Heart rate was recorded against an occlusion. Mouth pressure was measured by a differential pressure transducer (\(\pm 250\) cmH\(_2\)O, Validyne, Northridge, CA), analyzed, and then saved to a desktop computer. The MEP latency was measured as the time elapsed from the stimulus to the first muscle activity. The protocol had been approved by the appropriate ethics committee, and all subjects gave their informed, written consent for the study.

The following criteria had to be met to accept the highest \(V_{\text{O}2\text{max}}\) mean over 15 s at the time of task failure as maximal: 1) stabilization of \(V_{\text{O}2}\), 2) a respiratory exchange ratio > 1.1, and 3) attainment of the age-predicted maximal heart rate (220 – age).

The capacity of the subjects to produce static inspiratory pressures was measured in terms of the volitional maximal inspiratory pressure (PMAX) developed from residual volume against an occluded airway. PMAX was determined as the highest pressure maintained over 1 s against an occlusion. Mouth pressure was measured by a differential pressure transducer (\(\pm 250\) cmH\(_2\)O, Validyne, Northridge, CA), digitized at 500 Hz (MP100 Manager V3.2.6, Biopac Systems, Santa Barbara, CA), and stored on a computer.

**Exercise protocol.** Each subject performed two exercise trials: one with an ITL added (E + ITL) and one without (E). The runs were performed in random order, at a minimal interval of 2 days and at the same time of the day. Each trial consisted of a 5-min warm-up at 50% of the exercising load, immediately followed by a 16-min cycling bout. The exercising load required 55% of \(V_{\text{O}2\text{max}}\) and was below the ventilatory anaerobic threshold determined according to Wasserman et al. (37). Subjects were instructed to cycle between 60 and 70 rpm. Throughout the tests, gases were analyzed by breath by using the automated system described above.

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The six subjects in whom muscle activity was studied had a slightly modified exercise protocol: diaphragm responses to phrenic nerve stimulation were also recorded before exercise (after transcranial stimulations) and 15 and 25 min
after exercise. The purpose of these measurements was to assess any peripheral fatigue that may have developed. These subjects also performed the \( P_{\text{max}} \) maneuvers described above (see Preliminary evaluation above) at rest and 30 min after the end of each exercise trial.

The phrenic nerves were bilaterally stimulated with cervical magnetic stimulation (CMS) using 100% of the maximal output of a Magstim 200 system and a circular 90-mm coil (S/N, maximum output, 2 T; pulse duration, 0.05 ms; Magstim). Subjects were seated with the neck slightly flexed; they wore a nose clip and breathed through a mouthpiece with a small leak (to avoid glottis closure). Five stimulations were delivered at the functional residual capacity determined by visual control of chest movements, with at least 30-s intervals to avoid a twitch-on-twitch potentiation.

Mouth pressure measurement in response to phrenic nerve stimulation was performed as described in the Preliminary evaluation section. The amplitude of the twitch mouth pressure (\( P_{\text{mtw}} \)) signal was measured as the difference between baseline and the peak pressure during phrenic stimulation. The EMG (M-wave) and mechanical responses to CMS were acquired similarly to the responses to transcranial magnetic stimulation (see above). We took particular care in verifying the absence of difference in the M-wave amplitude of the diaphragm.

Statistical procedures. The distributions of the amplitudes and latencies of the MEP were checked for normality using the \( D \)-test of Kolmogorov-Smirnov. Because none of the MEP distributions was normal, a log-transformation was applied. The normality of the distributions after transformation was confirmed by \( D \) values of 0.077 for the diaphragm in the E and E/H1100 ITL trials, and \( D \) values of 0.087 and 0.077 for the quadriceps in the E and E/H1101 ITL trials, respectively (all \( P > 0.2 \)). The latencies of the diaphragm MEP were normally distributed during free and loaded exercise (all \( D < 0.1 \) and \( P > 0.2 \)).

Two-way analysis of variance with repeated measures was used to determine differences in the mean values of MEP amplitudes and latencies over the duration of exercise and recovery (Statistica Kernel version 5.5 Statsoft). The analysis for MEP amplitudes was performed on log-transformed data.

When a main statistical effect was found by ANOVA, means were compared by using a Newman-Keuls post hoc test.

Paired \( t \)-tests were used to assess differences between trials (with and without inspiratory load) when the effect of time was not analyzed.

Data are expressed as means \( \pm \) SD, unless otherwise stated. Differences were considered statistically significant when the probability \( P \) of a type I error was \( \leq 0.05 \).

RESULTS

Cardioventilatory data. Two subjects had to interrupt the loaded exercise trial before its scheduled end because of excessive hyperventilation, inducing acute dizziness in one case and intolerable dyspnea in the other. As they stopped only 1 and 2 min before the end of exercise, respectively, they were maintained in the analyses.

The mean minute ventilation, breathing frequency, \( V_t \), and duty cycle recorded over 15 min of the exercise were not different between the free and loaded runs (Fig. 1), as reflected by \( P \) values of 0.94, 0.69, 0.13, and 0.27, respectively. However, the individual breathing patterns in response to the threshold load varied among the subjects. Overall, the mean inspiratory flow rate (\( V_t/\text{inspiratory time} \)), which is assumed to reflect the ventilatory command, did not differ between the two runs.

All of the subjects but one scored a significantly higher dyspnea during the trial with inspiratory loading than during the free trial of whole body exercise (Fig. 2A).

![Fig. 1](http://jap.physiology.org/) Individual and mean variations between the 2 sessions, exercise (E) and inspiratory-loaded exercise (E + ITL), for the following ventilatory parameters: ventilatory output (A), breathing frequency (B), tidal volume (C), and respiratory duty cycle (D), i.e., ratio between inspiratory time and total time of a cycle.

NS, nonsignificant.

J Appl Physiol • VOL 98 • MARCH 2005 • www.jap.org
The mean heart rate was significantly higher during the loaded run than the free run (147 ± 15 vs. 144 ± 18 beats/min, \( P < 0.05 \)) (Fig. 2).

Muscle activity. The sternocleidomastoid activity was significantly higher (5.3 ± 2.5 vs. 15.4 ± 10.4) when the inspiratory load was added (Figs. 3A and 4). Indeed, the clinical observation of rib cage dynamics supported these results.

In the only three subjects whose diaphragm work could be measured during the two exercise runs, integrated transdiaphragmatic pressure increased from 18.3 ± 6.1 cmH2O/ms in the free trial to 56.7 ± 9.0 cmH2O/ms in the loaded one, and mean Pdi followed the same increment pattern (Figs. 3B and 4).

CMS and Pmax. The variance analysis showed that the Pmax was not affected by the trial (exercise vs. loaded exercise), the effort (after vs. before, whether including an inspiratory load or not), or a combination of these factors (Fig. 5A). Similarly, Pmtw did not change across conditions of time or exercise (Fig. 5B). The M-wave amplitude also remained unaffected by these factors (Fig. 6).

Responses to transcranial magnetic stimulation. ANOVA revealed an overall interaction effect between time of measurement and trial condition on the amplitudes of the diaphragm MEP (\( P = 0.038 \)). The amplitudes were not significantly different between the two trials at rest (\( P = 0.209 \)). Ten minutes after the end of E, however, they were significantly lower than at baseline and at the same point in time after E + ITL (Fig. 7A); 20 and 40 min after E, the amplitudes remained lower than at baseline, although not significantly so (\( P = 0.061 \) and 0.075, respectively). Conversely, E + ITL had no effect on the amplitudes of the diaphragm MEP (after vs. before, all \( P \) values > 0.88).

The average diaphragm MEP latencies were 15.5 ± 1.1 ms at baseline in the free condition and 16.0 ± 1.1 ms in the loaded condition. The latencies were unchanged over time during a given session and unaffected by ITL (\( P = 0.77 \)).

Figure 7B describes the evolution in the amplitudes of the rectus femoris MEP with time. The MEP amplitudes were unchanged over time after E and unaffected by ITL. The MEP latencies also remained unchanged between and during the trials (\( P = 0.98 \)). The average latency at rest was 22.1 ± 2.5 ms for E and 22.4 ± 2.1 ms for E + ITL.

DISCUSSION

This study shows that nonexhaustive whole body exercise in normal individuals can induce neurophysiological changes compatible with supraspinal diaphragm fatigue and that this effect is not present when the exercise is combined with a moderate level of ITL.

Methodological issues. A decrease in the amplitude of the motor potentials evoked by transcranial magnetic stimulation can be interpreted in terms of supraspinal fatigue when no significant metrological changes occur. In the present study, we took particular care to maintain a constant body posture and a constant relationship between the stimulating coil and the scalp throughout the experiments (see METHODS). In addition, because changes in the interelectrode impedance or the spatial relationship between the electrodes and the recorded motor units could theoretically have played a role in the variations that we observed, we compared the amplitude of the responses to peripheral stimulation before and after exercise. We observed no variation in the EMG response to CMS, which was expected, given that this signal is generally unaffected by fatigue protocols involving nonisotonic efforts. We are thus confident
that changes in the stimulus or changes in the recording conditions did not play a significant role in the phenomena that we observed.

The measurement of the pressure generated in response to CMS allowed for the control of peripheral muscle fatigue in this study. In terms of exercise intensity, ventilatory demand, and degree of loading, the experimental conditions were such that contractile fatigue was extremely unlikely to occur and particularly so in the diaphragm (19). Indeed, the absence of variation in the Pmtw suggests that peripheral fatigue did not develop.

Finally, nonfatiguing efforts tend to potentiate the EMG response to transcranial magnetic stimulation (3). This “post-exercise facilitation” phenomenon is the result of a transient increase in the excitability of the α-motoneurons induced by muscular activity (28), a feature that we did not observe after either type of exercise run. The MEP latencies in our subjects, which were in the normal range for unfacilitated responses (29), did not differ from their baseline values after exercise. The maximal inspiratory maneuver has potential facilitatory effects, but it was followed by 10 min of rest. The subjects remained quiet, i.e., motionless and silent, during the recovery. Postexercise facilitation, therefore, does not seem likely to have been a confounding factor.

Central diaphragm fatigue in response to unloaded exercise. The twitch interpolation technique has been used to assess the degree of voluntary activation of a given muscle during a voluntary contraction (21, 24). It consists of superimposing supramaximal stimulations on the nerve commanding an already contracting muscle. When task failure is associated with an additional mechanical output in response to this stimulation, fatigue is assigned to a central component. Central diaphragmatic fatigue has been evidenced using this technique in various contexts (5, 21) characterized by diaphragm-oriented loading protocols. Véris et al. (34) seem to have been the first to provide arguments for central diaphragm fatigue after whole body exercise. They found that both the diaphragm and the quadriceps exhibited postexercise decrements in their responses to transcranial magnetic stimulation after an incremental walking exercise, likely to require high ventilatory outputs. Compared with their study, we did not observe signs of supraspinal fatigue in the recti femori. The reason for this difference may lie in the nature of the exercise paradigm used: our protocol was specifically designed not to provoke exhaustion. The two studies are, however, consistent in that they suggest a greater susceptibility to supraspinal fatigue in the diaphragm than in the quadriceps. This result is compatible with the view that central fatigue has a protective function (13). It is also tempting to draw a parallel between the results of
these two studies and the fact that normal subjects achieve greater activation of the quadriceps than of the diaphragm during similar loading protocols (14). This feature has been called on by Guleria et al. (14) to explain the relative difficulty of experimentally producing diaphragm contractile fatigue compared with other muscles.

Of note, most work on supraspinal fatigue has involved intense muscular efforts, generally monoarticular and leading to task failure. The muscles under study have generally been small and distal (8, 28), with some exceptions (27, 33). This was not the case for the diaphragm in our experiments, which makes comparison with other results difficult. However, supraspinal fatigue has been observed in several muscles as a result of whole body exercise (18) (as opposed to specific limb solicitation).

**Effects of ITL on the postexercise diaphragm response.** Increased ventilatory demand is often associated with changes in the activity and recruitment pattern of the respiratory muscle groups (19, 26). These changes generally tend to put the burden of the ventilatory load on extradiaphragm muscles. For example, exercise is normally associated with a precocious and strong recruitment of abdominal muscles that directly contribute to the ventilatory work by their expiratory action. They also indirectly help inspiration by bringing the rib cage at end expiration below its equilibrium position, therefore making rib cage elastic recoil available for inspiration (10). Rib cage and neck muscles are also recruited during exercise. They contribute directly to the expansion of the rib cage and decrease rib cage distortability (21). As a result, it has been shown that the nature of the diaphragm contraction changes during exercise (2), and, therefore, its energy expenditure is not increased disproportionately. Studies, particularly on limb muscles, have clearly demonstrated that a given task can be accomplished.

![Fig. 6. Representative M-wave trace recorded in 1 subject at the measurement time points of baseline and 15 and 25 min post-exercise (post) of the E trials (A) and the E + ITL trial (B).](image1)

![Fig. 7. Means and SE for motor-evoked potential amplitudes of the diaphragm (A) and rectus femoris (B) at rest and 10 (P10), 20 (P20), and 40 min after (P40) the end of exercise. Open bars, E trial; solid bars, E + ITL trial. *P < 0.05 compared with rest in the same condition; #P < 0.05 compared with the other condition at the same time. NS, interaction effect between the time and condition effects.](image2)
EXERCISE-INDUCED DIAPHRAGM SUPRASPINAL FATIGUE

with different recruitment strategies. For example, fatigue of the vastus lateralis has been shown to modify the recruitment of synergistic muscles, thereby preserving the success of a knee extension task (1). Given the compensatory mechanisms that are developed in patients with diaphragm paralysis (15) and in healthy subjects exercising with fatigued inspiratory muscles (30), it also appears reasonable to assume that changes in the pattern of extradiaphragm respiratory muscle activity would allow the ventilatory demand to be met, despite reduced diaphragm activity. To support this, Johnson et al. (19) showed that twitch transdiaphragmatic pressure was reduced at all lung volumes after task failure in 12 healthy volunteers, with a variety of fitness levels exercising to exhaustion at 85 and 95% of their VO2max. In this study, the contribution of the diaphragm to the respiratory motor output tended to decrease with the duration of the effort, with a shift of the load toward other muscles.

Inspiratory loading favors the recruitment of inspiratory neck and rib cage muscles (30), and the amplitude partly determines endurance. For this study, the choice of 10% of the Pmax was based on preliminary studies that showed the load to be demanding and capable of generating strong neck and rib cage muscle contractions. The literature (6, 34) reveals that this load is associated with a normal respiratory duty cycle and is unlikely to induce low-frequency diaphragm fatigue, which had to be avoided in this study. The recruitment of additional muscles with loading is observed in patients with chronic obstructive pulmonary disease and in healthy subjects. Both resistive (16) and threshold (11) loading lead to strong solicitation and sometimes fatigue (16, 39) of extradiaphragmatic muscles. The ITL added to the burden of the exercise-related increase in ventilatory demand consistently resulted in recruitment of extradiaphragmatic muscles, as shown by the EMG recording of the SCMs. Moreover, our subjects reported dyspnea during the loaded protocol, whereas this was not the case during the “free” protocol. Yet Fitting et al. (12) and Ward et al. (36) both clearly showed that recruiting the SCM and parasternal intercostal muscles rather than the diaphragm to perform a given respiratory task is strongly associated with dyspneic sensations. Therefore, we suggest that the redistribution in the respective roles of the inspiratory muscles is related to the disappearance of the postexercise depression in diaphragm MEPs in the E + ITL condition.

This is not a simple assertion that the decrease in MEP is due to lower recruitment of the diaphragm. There is probably no correlation between the amount of work produced by a muscle group at a given time and the fatigue of its command. Instead, we propose that the decreased excitability after exercise is linked to the reorganization of the diaphragm’s motor command. When moderate exercise is not prolonged, this reorganization could optimize the use of resources. Changing the breathing has already been presented as an optimization strategy that is implemented during the development of fatigue (11). Consequently, it could occur without any change in the mechanical output or alteration in the maximal performance (Pmax, in this case), but it may still be a limiting factor when exercise is prolonged, probably through the development of dyspnea (22, 31). Sufficient data are lacking to venture a hypothesis about whether the adaptation depresses the excitability of the motor command, as reflected by a decrease in MEP amplitude, or whether decreased excitability results in reorganized recruitment. We, nevertheless, suspect that the second position will be borne out and propose that, early in the E + ITL trial, mechanical affinences led to a relative inhibition of the diaphragm motor command that later normalized. Even though it results in dyspnea, which is another constraint, the recruitment of neck and rib cage muscles controls the major stress to the respiratory system, i.e., the inspiratory load. It is possible that, in this condition, the excitability of the respiratory pathways is reset up during the exercise itself and is not influenced by slighter changes in the homeostasis. Longer inspiratory loaded exercise would be interesting to study to assess whether dyspnea continues to act as a safety feature by leading to the end of exercise before the respiratory system adapts to metabolic changes through variation in the excitability of its command. In contrast, a finer regulation of the diaphragm command, based on both mechanical and chemical affinences, could be sought during exercise. This would result in a prolonged relative domination of inhibitory over excitatory mechanisms.

In conclusion and despite its limitations, this study seems to contribute to the current body of knowledge suggesting that several mechanisms act in concert to protect the diaphragm against overload-induced damage. These mechanisms include a reduced central activation in loaded conditions (5), the early occurrence of supraspinal fatigue or inhibition (21), and agonist recruitment to overcome an increased demand.

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EXERCISE-INDUCED DIAPHRAGM SUPRASPINAL FATIGUE

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