

Diaphragm and intercostal surface EMG and muscle performance after acute inspiratory muscle loading

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Abstract

We examined the effect of an acute bout of submaximal non-fatiguing inspiratory loading (IL) on maximal inspiratory pressure (MIP), and on the activation of the diaphragm (DI) and intercostals (IC) using surface electromyography (sEMG). After baseline measurements, 12 healthy subjects performed two sets of 30 inspiratory efforts at a load equivalent to 40% of their initial MIP. MIP and maximal DI and IC sEMG activity were recorded after the first and second set of IL, and 15 min after task cessation. After IL, MIP reached (\pm S.E.M.) $111 \pm 4\%$ ($P=0.032$) of baseline values, and during MIP, DI and IC root mean square (RMS) sEMG amplitude increased significantly above baseline ($143 \pm 21\%$, $P=0.039$ and $137 \pm 33\%$, $P=0.016$, respectively). The significant increase in MIP and RMS amplitude after IL suggests that MIP efforts were initially submaximal, and that prior loading enabled full activation. The changes in DI and IC RMS amplitude may also reflect an improvement in the synergy between them during these maximal efforts.

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1. Introduction

Acute prior activity of the inspiratory muscles has been shown to enhance inspiratory strength (Volianitis et al., 2001b), athletic performance (Volianitis et al., 1999, 2001a) and reduce the effort perception (Volianitis et al., 2001a) and magnitude estimation of inspiratory loads (Revelette and Wiley, 1987). Such isolated exercise sessions elicit acute, transient responses (da Nobrega, 2005), which if repeated frequently cause more permanent adaptations, referred to as an exercise training response (Thompson et al., 2001). Whilst the concept of exercise training has been applied to the inspiratory muscles over the past decade (for reviews, see Lotters et al., 2002; McConnell and Romer, 2004), there is a paucity of research describing the neurophysiological events that underlie the acute response to inspiratory muscle loading.

Evidence suggests that during evaluation of maximal isometric strength of the inspiratory muscle, a task learning effect occurs (as with other maximum voluntary isometric contrac-

tions), which influences baseline and subsequent measures (Meldrum et al., 2003; Volianitis et al., 2001a; Wen et al., 1997). Indeed, the variability relating to the number of manoeuvres performed can result in an underestimation of maximal inspiratory pressure (MIP) by as much as 20 cm H₂O (Wen et al., 1997). If a task learning effect does indeed exist, changes in MIP seen after an acute, or even chronic, intervention may simply reflect the subject becoming more accustomed to the manoeuvre, rather than specific improvements in the properties of the inspiratory muscles.

During deliberate inspiratory efforts the inspiratory intercostal muscles of the lateral chest wall tend to be recruited more than during quiet breathing (Whitelaw and Feroah, 1989). During such efforts a synergistic coordination of inspiratory muscle activity is required to maintain thoracoabdominal configuration and to generate maximal pressures (Roussos et al., 1979). Enhanced synergy between several muscles other than the diaphragm (i.e. external intercostals and sternocleidomastoid) has been postulated as a mechanism responsible for the increased MIP that is elicited by a brief period of submaximal inspiratory loading (Volianitis et al., 2001a). Enhanced intermuscular coordination represents the ability of the nervous system to activate involved muscles in synergy, which in turn may increase force

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output (Almasbakk and Hoff, 1996). Alterations may occur in the complex interactions between muscles when a movement is repeated many times, with the result that performance of that movement is enhanced (Kottke et al., 1978).

Thus, the purpose of the present study was to examine the effect of an acute bout of non-fatiguing, submaximal inspiratory muscle loading upon diaphragm and external intercostal sEMG and muscle performance in healthy human beings. We hypothesise that the pressure generating capacity of the inspiratory muscles will be enhanced by an acute bout of prior activity. We believe that this will be characterised by an increase in the sEMG amplitude in both the diaphragm and intercostals, reflecting increased activation during maximal effort. Furthermore, sEMG data will be used to assess whether there is an altered inspiratory muscle activation pattern after acute inspiratory loading that serves to increase force output.

2. Methods

We studied 12 healthy subjects (6 male), with no history of pulmonary or neuromuscular disease (mean \pm S.D.; age: 25 ± 9 years; stature: 177.1 ± 9.2 cm; body mass: 74.5 ± 15.2 kg; MIP: 125.6 ± 30.8 cm H₂O). Each subject gave written, informed consent to participate in the study, which was approved by Brunel University Ethics Committee.

2.1. Maximal inspiratory mouth pressure

Inspiratory muscle strength was estimated using the surrogate measure of maximal inspiratory mouth pressure during a quasi-static effort commencing at residual volume (RV). Initial maximal inspiratory pressure was measured using a mouth pressure meter (Precision Medical Ltd., Pickering, North Yorks, UK), which incorporated a flanged mouthpiece with a 1 mm leak (to prevent the production of artificially high pressures with the buccal muscles when the glottis is closed), a differential pressure transducer and a microprocessor.

Measurements of MIP were made from RV, which was verified at every measurement by asking subjects to exhale from total lung capacity via a Microloop spirometer (Micro Medical Ltd., Kent, UK). This ensured consistency of achieving RV over the course of the whole experiment. The subject was then encouraged verbally to make a maximal inspiratory effort for 2–3 s. Since all future data were collected from the subjects in a seated upright position, this initial MIP measurement was also performed in this position. A minimum of 5 and a maximum of 10 technically satisfactory measurements were conducted, and the highest of three measurements with <5% variability or within 5 cm H₂O difference was defined as maximum (Wen et al., 1997). This initial MIP value was then used to calculate the magnitude of the inspiratory load to be used in the acute inspiratory loading bout.

Throughout the subsequent experimental protocol, MIP was measured using a system that incorporated a manually operated three-way stopcock with a flanged mouthpiece (Hans Rudolph Inc., Kansas City, MO, USA). The mouthpiece could be occluded by turning an internal shutter, so that quasi-static

mouth pressure could be recorded. This mouthpiece also contained a small 1 mm leak to avoid glottis closure and was attached via 1 m of 2 mm diameter polyethylene tubing to a Validyne DP15 differential pressure transducer (± 230 cm H₂O) (Validyne Co., Northridge, CA, USA) and a Validyne CD15 carrier demodulator (Validyne Co.). This signal was digitized using an analogue to digital converter (Micro1401, Cambridge Electric Design, Cambridge, UK) and acquired using commercially available software (Spike 2, Cambridge Electronic Design) on a personal computer (Packard Bell, NEC Computers, Angers, France). Real-time mouth pressure traces were displayed to the subject.

2.2. Diaphragm and external intercostal EMG

Diaphragm and external intercostal EMG were recorded transcutaneously from pairs of bipolar differential electrodes (Goldy Karaya Gel electrodes, 28 mm diameter, silver/silver chloride, Arbo[®], Henley Medical, Stevenage, UK) placed on the cleaned, abraded skin. For the diaphragm the electrodes were placed in the lowest intercostal spaces on the right side of the body, at the mid-clavicular line, and for the external intercostal muscles, in the fifth intercostal space at the posterior axillary line. A ground electrode was placed on the sternum. The distance between a pair of electrodes was kept to a minimum and was never more than 2 cm, and care was taken to place the electrodes in the same orientation as the muscle fibres. Once the electrodes were positioned and a clear EMG signal was confirmed (by a deep inspiration), the electrodes were fixed in place using adhesive surgical tape. It has been established that with appropriate placement of electrodes, quality EMG recordings, minimally disturbed by unwanted external factors, can be obtained from the diaphragm and intercostals (Duiverman et al., 2004).

The electromyographic responses were amplified (gain $\times 3000$) (1902, Cambridge Electronic Design, Cambridge, UK), band-pass filtered between 20 Hz and 5 kHz and digitised at a sampling rate of 4 kHz using an analogue to digital converter (Micro1401, Cambridge Electronic Design), and finally acquired and later analysed using commercially available software (Spike 2 v4.11, Cambridge Electronic Design).

The influence of the ECG on the EMG signal was minimised by recording from the right side of the body. To further minimise any signal contamination by ECG on the diaphragm EMG during the 2–3 s MIP effort, RMS was measured from the segments between QRS complexes.

2.3. Inspiratory muscle loading

A pressure threshold inspiratory loading device (POWERbreathe[®], Gaiam Ltd., Warwickshire, UK) was used to provide the acute bout of inspiratory muscle loading. The intensity and duration of the acute bout was based on the protocol used by Volianitis et al. (2001a,b), where two sets of 30 breaths at an intensity of 40% of the MIP measured prior to the experimental procedure are performed. Forty percent of maximum capacity has been suggested to approximate the upper loading limit before fatigue of the diaphragm occurs

(Roussos and Macklem, 1982). This inspiratory load was obtained by measuring the pressure at the mouth as subjects breathed through the loading device, and a needle tipped, fine bore tube was passed from the manifold of the mouthpiece to the mouth pressure meter (described previously to evaluate MIP). The desired pressure (40% of subjects pre-recorded MIP) was achieved by adjusting the degree of spring compression within the loading device.

The subjects were instructed to perform dynamic manoeuvres starting near RV and terminating towards total lung capacity for each breath. A nose clip was worn throughout the inspiratory loading protocol. Because of the increased tidal volume, a decreased, but spontaneous breathing frequency was adopted by the subjects in order to prevent, or minimise, the effects of hypocapnia. To this end, a low duty cycle resulted which also helped to ensure the acute bout was non-fatiguing.

2.4. Experimental procedure

At least 24 h (range 24–33 h) prior to the main experimental procedure subjects performed initial tests of MIP. Subjects also attended at least one further familiarisation session in order to individually calibrate the pressure threshold device according to their MIP, and to become familiarised with breathing through the pressure threshold device.

Before the loaded breathing sets (baseline), immediately after set 1, immediately after set 2 and 15 min post-cessation of the breathing task, experimental data was collected. Data collection immediately after the breathing sets was initiated within 20 s of completion of the set, and during the 15 min recovery, subjects sat relaxed and refrained from talking. At each of the data collection points, maximal inspiratory efforts of 2–3 s duration were performed three times, with a 30 s break between each.

2.4.1. 'Sham' trial

In six of the subjects the experimental protocol was repeated within 2 weeks of the original experiment (range 7–12 days), but instead of performing two sets of loaded inspirations, subjects sat relaxed and breathed through a flanged mouthpiece with no resistance. The time between baseline measurements and subsequent data collection time points was calculated from the time taken to perform 30 breaths using the pressure threshold device for each individual subject in experiment one. The average time between data collection points was 2.5 min. The subjects then sat relaxed for a further 15 min when a final set of data collection was made. In this way, the entire data collection procedure was replicated, but with the loaded breathing substituted by quiet breathing.

2.5. Data analyses

Data were analysed off-line. The EMG signal was analysed in the time domain, as root mean square (RMS) amplitude with a time constant of 25 ms. Computer aided analysis was performed over a 0.5 s window initiated at the point of peak pressure during the maximal inspiratory effort for both the diaphragm and intercostals. Peak inspiratory pressures were recorded from

each maximal effort, and the highest value was recorded as the MIP.

Statistical tests (z -scores and Mahalanobis distances) were performed on the data set to identify univariate and/or multivariate outliers. These tests revealed that there were no statistical outliers, and therefore all data was included in subsequent statistical analyses. MIP and RMS sEMG results were analysed using a one-way repeated measures analysis of variance (ANOVA) and Bonferonni post hoc test to assess the difference between the baseline and post-set 1, post-set 2 and post-15 min values. A one-way ANOVA with a within group comparison was used to compare the percentage change from baseline in the six subjects who participated in both the experimental and sham trials. A Pearson's correlation coefficient was used to assess the association between variables. Values of $P < 0.05$ were considered statistically significant. Data points are expressed as both percentage of baseline, with baseline being 100%, and raw scores, and are mean \pm standard errors of the mean (S.E.M.). Statistical analyses were performed using SPSS v11.5 for Windows (SPSS Inc., Chicago, IL, USA).

3. Results

3.1. Maximal inspiratory pressure

MIP was $111 \pm 4\%$ ($P = 0.032$) of baseline immediately after the loaded breathing protocol, returning to near baseline levels after 15 min (Fig. 1). However, when testing was performed after quiet, relaxed breathing in the sham trial, no significant changes in MIP were observed (Fig. 1 and Table 1).

3.2. Diaphragm and external intercostal sEMG

During maximal inspiratory efforts both diaphragm and intercostal RMS amplitude increased above baseline after the two sets of loaded inspirations ($143 \pm 21\%$, $P = 0.039$ and $137 \pm 33\%$, $P = 0.016$; Table 1). After 15 min RMS amplitude

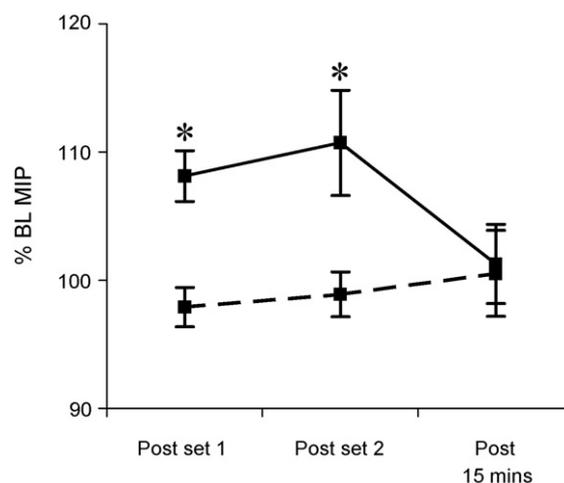


Fig. 1. Change in MIP after two sets of loaded inspirations at 40% MIP (solid line) and after the sham protocol (broken line). All data is normalised to baseline (BL) values. Asterisk (*) denotes significantly different from baseline ($P \leq 0.05$).

Table 1
Mean (\pm S.E.M.) data for MIP and diaphragm (DI) and intercostal (IC) RMS EMG amplitudes before and after two sets of loaded inspirations (IL) and relaxed breathing (sham)

	Baseline		Post-set 1		Post-set 2		Post-15 min	
	IL	Sham	IL	Sham	IL	Sham	IL	Sham
MIP (cm H ₂ O)	121 \pm 10	110 \pm 10	130 \pm 10*	109 \pm 10	132 \pm 9*	109 \pm 10	120 \pm 11	111 \pm 11
DI EMG (μ V)	43 \pm 9	24 \pm 6	70 \pm 21	22 \pm 5	50 \pm 10* Ω	21 \pm 5	52 \pm 4	24 \pm 6
IC EMG (μ V)	73 \pm 18	49 \pm 15	79 \pm 17	50 \pm 14	91 \pm 20* Ω	48 \pm 15	93 \pm 20	48 \pm 12

Asterisk (*) denotes significantly different from baseline ($P < 0.05$) and (Ω) denotes a significant correlation between change from baseline ($P = 0.003$).

was still elevated from baseline in both the diaphragm and intercostals (126 ± 14 and $131 \pm 12\%$, respectively) (Fig. 2). Fig. 3 depicts representative mouth pressure and sEMG data from one subject during a maximal inspiratory effort.

3.3. The sham trial

In the sham trial, no significant changes were observed in MIP, or in the RMS amplitude of diaphragm and intercostal EMG at any of the data collection points, during the maximal inspiratory efforts (Table 1). Furthermore, diaphragm and intercostal RMS sEMG percentage change from baseline differed significantly from that of the sham trial after the breathing sets ($P = 0.037$ and 0.038 , respectively). Percentage change in MIP after the loaded inspirations was also significantly different from changes occurring after the sham trial ($P = 0.016$). After 15 min recovery, change in diaphragm RMS sEMG and MIP from baseline did not differ significantly from the sham trial ($P = 0.145$ and 0.780 , respectively), however, change in intercostal RMS sEMG still showed a significant difference from the sham trial ($P = 0.03$) at this time point.

3.4. Relationship between mouth pressure generation and inspiratory muscle activation

Statistical analyses revealed no significant correlation between the percentage changes from baseline in MIP and sEMG amplitude of either the diaphragm or intercostals. However, a significant correlation ($r = 0.772$, $P = 0.003$) existed between the change in sEMG amplitude of the diaphragm and the change in sEMG amplitude of the intercostals after the second set of loaded inspirations (Fig. 4).

4. Discussion

An acute bout of non-fatiguing submaximal inspiratory loading increased global inspiratory strength, as measured using MIP, and increased RMS amplitude of diaphragm and intercostal sEMG during subsequent maximal inspiratory efforts. The increase in inspiratory strength was transient, decreasing to baseline values after 15 min; however, the increase in RMS sEMG was more long-lasting, with values remaining above baseline after 15 min of recovery.

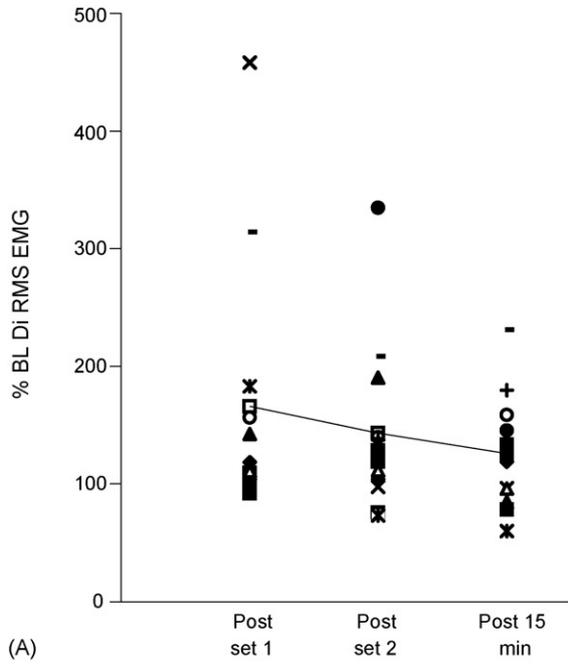
Surface EMG recordings provide a popular, routine tool with which to investigate chest wall muscle function. However, analysis and interpretation of the surface EMG can easily be

confounded if the recorded EMG signal of interest is contaminated by non-physiological signals, or by signals originating from muscles located adjacent to the muscle under investigation (Sinderby et al., 1996). The use of sEMG is a potential limitation of our study, but one we do not believe affects the interpretation of our results. Duiverman et al. (2004) have confirmed that, with appropriate placement of electrodes, high quality EMG recordings can be made, and that these are minimally disturbed by unwanted external factors. To limit signal contamination we placed electrodes in a way that has been shown to minimise recording muscle cross-talk, i.e. diaphragmatic surface electrodes were placed in the lowest accessible intercostal space close to the costochondral junction and maintaining the interelectrode distance at less than 2 cm (Demoule et al., 2003; Glerant et al., 2006; Maarsingh et al., 2000). By following the 'best practice' suggested by previous studies we are confident that cross-talk from accessory respiratory muscle activation did not play a significant role in the changes that we observed. This is substantiated by data from the sham trial.

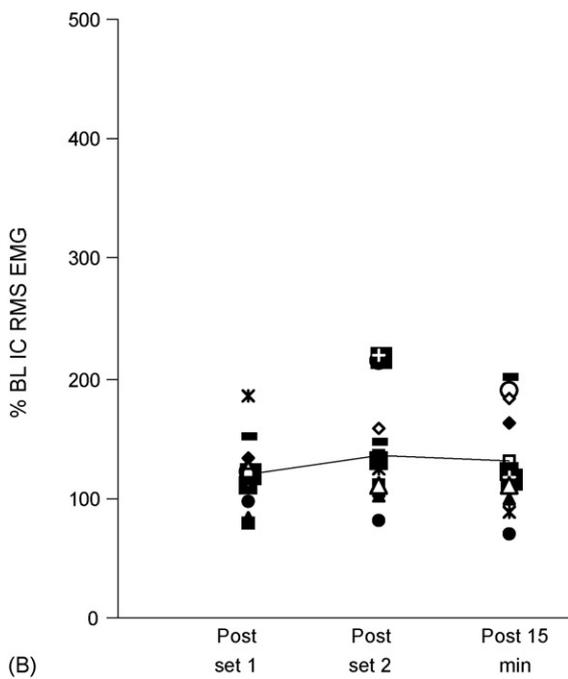
The magnitude of the improvement in MIP that we observed is consistent with findings of earlier studies employing a similar intervention (ranging from 7 to 11.2%; Volianitis et al., 2001a,b, respectively). However, the present study is the first to examine sEMG data during these augmented MIP manoeuvres following prior inspiratory activity.

RMS amplitude has been shown to have a positive relationship with force production (De Luca, 1997). Whilst our data are consistent with this, we were surprised not to find a correlation between the increases in sEMG amplitude and the increases in the force generating capacity of the inspiratory muscles (MIP). However, the requirement of considerable accessory muscle (e.g. scalenes and sternocleidomastoids) recruitment with increasing inspiratory pressure generation, may serve to confound the EMG–force relationships of this system due to the heterogeneous muscle recruitment during deliberate inspiratory effort (Yokoba et al., 2003). Whilst it is evident from our data that fuller activation of both the diaphragm and intercostals was achieved after prior loaded activity, it is possible that changes in the strategy of motor unit recruitment employed (Zhou et al., 1987), brought about by prior loading may be more strongly correlated with pressure generation. This is an area that warrants further investigation.

A number of neural factors may be involved in an increased force output and sEMG amplitude of a muscle or muscle groups. Our observed increase in both diaphragm and intercostal sEMG may reflect increased motor unit recruitment, and



(A)



(B)

Fig. 2. (A) Diaphragm (DI) and (B) intercostal (IC) RMS sEMG expressed as a percentage of baseline (BL) values, after two sets of loaded inspirations. Data points represent individual subjects and the dashed line connects the data points representing the mean. Changes were significantly different from baseline at the post-set 2 time point ($P \leq 0.05$).

thus an increased activation of the target muscles (Hunter and Enoka, 2003). Using phrenic nerve stimulation, McKenzie et al. (1997) found, that voluntary activation of the diaphragm during MIP efforts was initially submaximal (94.7%) during a resistive breathing protocol. In the context of the present study, it is also possible that the baseline MIP efforts were submaximal, and that prior loading enabled full activation. Future work in this area should utilise twitch interpolation techniques to investigate

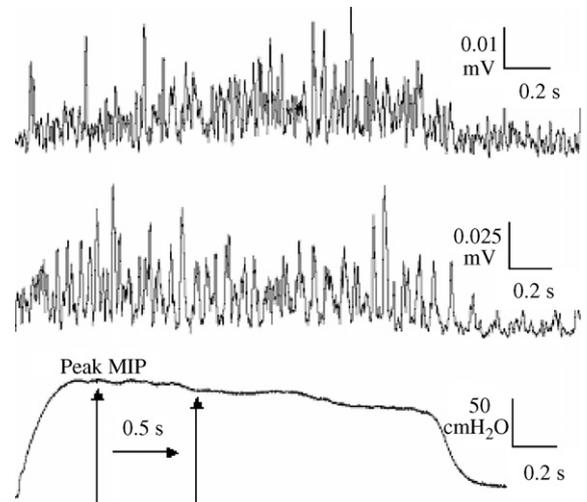


Fig. 3. Diaphragm (top) and intercostal (middle) RMS sEMG and mouth pressure (bottom) during a maximal inspiratory effort in one subject. The arrows indicate identification of peak inspiratory mouth pressure and subsequent 0.5 s window of EMG analysis.

the evidence of initial submaximal activation provided by our observations.

Alternatively, the increase in sEMG amplitude of the diaphragm and intercostals may reflect a shift from random to synchronised motor unit firing activity (Lloyd, 1971). The effect of motor unit synchronisation on EMG amplitude can be explained by a reduction in the cancellation of the EMG signal that occurs when positive and negative phases of action potentials overlap. With increased synchrony there is little cancellation, and the EMG amplitude becomes larger (Yao et al., 2000). The bout of prior loading may have served to produce a significant synchronisation of motor units during subsequent maximal voluntary contraction (Milner-Brown et al., 1975; Suzuki et al., 2002).

From previous research we know that activation of inspiratory accessory muscles increases during deliberate inspiratory

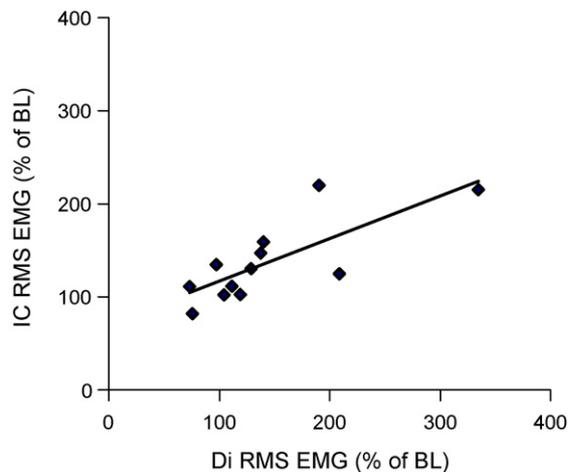


Fig. 4. Correlation between the change from baseline (BL) of sEMG RMS amplitude for diaphragm (DI) and intercostals (IC) after the second set of loaded inspirations.

efforts (Hershenson et al., 1989; Roussos et al., 1979; Whitelaw and Feroah, 1989). The observed increase in intercostal sEMG amplitude after the two sets of loaded inspirations is consistent with these previous findings. However, after the first set of loaded inspirations, intercostal sEMG amplitude increased to only 121% of baseline, whilst diaphragm sEMG amplitude increased to 166% of baseline. This suggests that when initially faced with a submaximal inspiratory load the diaphragm is the principal pressure generator. However, after the second set of 30 loaded inspirations diaphragm sEMG activity was slightly lower than after the first set (143% of baseline), whilst intercostal sEMG activity showed a further significant increase to 137% of baseline. This may indicate a 'shift' in the recruitment pattern of the inspiratory muscles that reflects an improvement in the synergy between them during maximal efforts. This would be consistent with the notion that during loaded breathing the contribution of diaphragm and intercostal muscle activity to the work of breathing changes to 'optimise the use of resources' and prolong endurance time (Jonville et al., 2005; Roussos et al., 1979). This adjustment in intramuscular coordination could reflect altered central drive allowing increased co-contraction of accessory muscles involved in the maximal effort (Anderson and Behm, 2005). Thus, the augmentation of force (characterised by the increased MIP) subsequent to the bout of submaximal inspiratory muscle activity in the present study could be attributed to changes in synergistic behaviour.

4.1. Time course of effects

The increase in MIP seen after the acute loading bout is transitory, since MIP returned to around baseline levels after 15 min of recovery. Interestingly, the increases seen in sEMG did not return to baseline after this time. This suggests that although activation of the inspiratory muscles remained elevated above baseline after 15 min, the contribution to MIP made by factors such as synergistic coordination may have diminished, such that MIP decreased to its baseline level. The pattern of these temporal changes imply that the change in MIP cannot be explained solely by enhanced activation of the muscle concerned, which is also consistent with the lack of correlation between changes in sEMG and MIP.

It is conceivable that fatigue (induced by the acute loading protocol) may have been responsible for the dissociation between global inspiratory strength (MIP) and muscle activation (sEMG). It is recognised that following an acute bout of muscle and motor unit activation, potentiation and fatigue of the neuromuscular apparatus may simultaneously occur (Chiu et al., 2004; Rassier and Macintosh, 2000; de Ruiter et al., 2005). Thus, *in vivo* performance is a measurement of the interaction between these two variables at any one time (Chiu et al., 2004). It is possible that immediately after the acute bout of inspiratory loading the augmented MIP and sEMG amplitude reflected a predominance of force potentiation, whilst after 15 min recovery, potentiation was no longer sufficient to compensate for any low frequency fatigue (LFF) that may have been produced by the loading protocol. Although the submaximal load and volume of the acute bout used in the current study was selected

to avoid fatigue, LFF has been reported to occur after submaximal voluntary contractions (Ratkevicius et al., 1995) and can persist in the absence of gross metabolic or electrical disturbance to the muscle (Jones, 1996). The return to baseline values of maximum inspiratory pressure generation after 15 min could reflect LFF (which is characterised by a loss of force), and the sustained increase in sEMG amplitude may no longer reflect increased activation, but an increase in motor unit discharge rates and additional motor unit recruitment to compensate for the loss of pressure generating capacity.

4.2. Reliability of MIP and sEMG measures

It has previously been suggested that performing a 'long series' ($n=20$) of MIP manoeuvres results in a task learning effect, and results in an increased peak performance (Wen et al., 1997). In the present study, the improvements in MIP and changes in sEMG were due to the acute inspiratory loading intervention, and not to the task learning associated with repeated maximal efforts. We are confident of this because subjects who performed the sham trial showed no significant changes in MIP or sEMG amplitude. Although measurements demanding cooperation, volitional motivation and technique from subjects may indeed show improvements of results over time due to a task learning effect, our results clearly indicate that with a thorough familiarisation procedure, no task learning effect is observed.

4.3. Practical consequences

4.3.1. Reliable MIP measurement

The findings of the present study have implications for the reliable measurement of MIP. In studies requiring stable and accurate baseline measures of inspiratory muscle strength prior to an intervention, a reliable measure of MIP is essential. Previously, it has been suggested that at least 10 maximal inspiratory efforts are required to obtain a peak MIP measurement (Wen et al., 1997), and others have reported significant increases throughout 18 MIP measurements (Volianitis et al., 2001b). Performing such a large number of manoeuvres at each testing session may be extremely time consuming, especially since each maximal effort has to be followed by an adequate recovery period to avoid fatigue. However, our results are in agreement with those of Volianitis et al. (2001b), who found that if MIP measurements were made after prior respiratory activity, consisting of 2 sets of 30 loaded inspirations (at 40% MIP), a reliable baseline could be measured with the first subsequent MIP effort. Our data suggest a similar pattern, where the substantial increases in MIP seen after the first set of inspiratory efforts plateau (<2% change) after the second set, with no further significant increases in MIP being attained. This emphasises that an acute bout of prior submaximal inspiratory activity can, in a relatively short time, with limited specialised equipment, provide a stable baseline measurement of MIP.

In conclusion, the findings of the present study demonstrate that prior acute inspiratory muscle activity transiently enhances maximum pressure generation of the inspiratory muscles, and increases EMG activity of the diaphragm and external inter-

costals. This suggests that the submaximal loading protocol conditioned subjects to more fully activate the inspiratory muscles during maximal inspiratory efforts. The increased sEMG amplitude was longer lasting than the MIP enhancement. Future studies will address the degree of change in voluntary activation after the prior inspiratory activity, and whether the ‘uncoupling’ of MIP and sEMG after 15 min recovery represents the development of low frequency fatigue.

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