

Activity of *latissimus dorsi* muscle during inspiratory threshold loads

M. Orozco-Levi, J. Gea, J. Monells, X. Aran, M.C. Aguar, J.M. Broquetas

Activity of latissimus dorsi muscle during inspiratory threshold loads. M. Orozco-Levi, J. Gea, J. Monells, X. Aran, M.C. Aguar, J.M. Broquetas. ©ERS Journals Ltd, 1995.

ABSTRACT: The ability of the *latissimus dorsi* muscle (LD) to participate as an accessory inspiratory muscle has been the subject of controversy. Electromyographic (EGM) activity of LD was evaluated in 11 healthy subjects (aged 30 ± 2 yrs; forced expiratory volume in one second (FEV_1) $106 \pm 5\%$ predicted; maximal inspiratory pressure (P_{imax}), 120 ± 6 cmH₂O) under different breathing conditions. The ipsilateral *biceps brachii* was chosen as the control muscle. The EMG was recorded from surface electrodes, but needle electrodes were also used for LD evaluation in a subset of three subjects. The EMG signal from both muscles was recorded simultaneously, rectified and integrated, with subtraction of the electrocardiographic signal. Situations evaluated were: 1) maximal voluntary contraction (MVC); 2) apnoea; and 3) breathing under progressive inspiratory threshold loads (20–100% P_{imax} , at 20% intervals).

A close relationship was evident between LD recordings from surface and needle electrodes ($r=0.975$). Activity of LD at baseline was $1.8 \pm 0.4\%$ MVC, and showed a phasic increase during inspiration under loads. This change had a linear tendency and was significant for loads corresponding to 40, 60, 80 and 100% of P_{imax} when compared to the control muscle. At this latter level, LD activity was equivalent to $32 \pm 5\%$ MVC (range 11–61%), whereas mean activity of the control muscle was less than 7.5% MVC.

These results demonstrate that LD is progressively recruited in healthy subjects during inspiratory loading, and suggest that LD could participate as an accessory muscle for the breathing effort under specific conditions. For these reasons, LD does not appear to be an appropriate control for studies of the respiratory muscles.

Eur Respir J., 1995, 8, 441-445.

Inspiratory loading induces an increase in the activity of the main inspiratory muscles, such as the diaphragm and parasternal intercostals [1–3], and may induce muscle fatigue. Activity of other muscles from the chest wall, or even the trunk, can also increase under these circumstances [4, 5]. Nevertheless, both the specificity and physiological significance of this recruitment remain controversial. *Latissimus dorsi* (LD) is a trunk muscle. Its origins are in the dorsal and lumbar vertebrae, the sacrae midline, the posterior iliac crest and the lower ribs. All of its fibres converge together to insert into the humerus by a common tendon. The LD participates in different functions, such as maintaining body posture and collaborating in the adduction and internal rotation of the arm [6, 7]. Although some anatomists and physiologists have argued that LD can also be implicated in respiratory movements under specific circumstances [4–7], other authors have considered, in contrast, that it does not participate in breathing at all [8, 10]. For this last reason, LD has been included as control muscle in several structural [9, 10], as well as electrophysiological [11], studies of the respiratory muscles.

However, we [12] have recently described a direct relationship between the size of LD fibres and the severity of airways obstruction in chronic obstructive pulmonary disease (COPD) patients. Although the cause of these structural change still remains unclear, we hypothesized that it could be related to the recruitment of LD under certain circumstances involving ventilatory load. This study was carried out in order to investigate the activity of LD when progressive inspiratory loads are applied in healthy subjects.

Material and methods

Study subjects

Eleven healthy volunteers (all male, aged 30 ± 2 yrs; forced expiratory volume in one second (FEV_1) $106 \pm 5\%$ predicted; maximal inspiratory pressure (P_{imax}) 120 ± 6 cmH₂O) were included. The study was approved by the local Research Committee of Human Investigation. Subjects were requested not to perform important

Servei de Pneumologia and Servei de Neurofisiologia, Hospital del Mar, Unitat de Recerca de Pneumologia, I.M.I.M. Universitat Autònoma de Barcelona, Barcelona, Catalonia, Spain.

Correspondence: M. Orozco-Levi
Servei de Pneumologia
Hospital del Mar
Passeig Marítim 25-29
08003
Barcelona
Spain

Keywords: Accessory muscles
electromyography
inspiratory loading
latissimus dorsi

Received: August 8 1994
Accepted after revision January 4 1995

Preliminary results presented at the European Respiratory Society Annual Meeting, Florence, Italy, 1993.

Supported by grants FIS 92/0314 and IMIM 1642380.

physical activity in the 72 h preceding the study. A specific questionnaire did not reveal any relevant interpersonal difference in their day-to-day activities. None of the volunteers were familiarized with either the protocol, the aim of the study, or the recording systems for the electromyographic (EMG) activity.

Study design

Pulmonary function tests. The day preceding the study, a forced spirometry was performed (Datospir 92, Sibel, Barcelona, Spain). Reference values were those from a Mediterranean population [13]. P_{max} was obtained from a manometer with an occludible mouth piece (Sibelmed 63, Sibel, Barcelona, Spain). P_{max} was performed from residual volume, and the maximal value of six consecutive manoeuvres was chosen.

Electromyographic studies. Subjects were studied whilst sitting comfortably in a chair, maintaining the head, neck and trunk fixed against a rigid backrest. The arms and hands rested on two lateral supports fixed to the chair. Volunteers were instructed to minimize the possibility of carrying out voluntary contractions or movements of the arms throughout the study. EMG activity was recorded from surface electrodes (Silver/silver chloride, Surface Electrodes 17637, Medelec, UK) using a bipolar technique. Interelectrode impedance was always less than 10 kOhm. For LD activity recording, two electrodes were placed caudally and laterally to the lower angle of the scapula on the nondominant side and separated by 2–3 cm. In a subset of three subjects, concentric needle electrodes were simultaneously used to validate the specificity of the surface electrodes for LD recordings. The ipsilateral *biceps brachii* was included as the control muscle, because it participates in arm movements but not in ventilation [6, 7]. Two surface electrodes were attached to the arm on the middle of the biceps muscle 2–3 cm apart. Electrodes were connected to a multichannel recorder (Mystro, Medelec MS 25, UK). The EMG activity of both muscles was bandpass-filtered (100–2,000 Hz) and simultaneously recorded in each situation throughout the study. EMG signals were processed (rectified and integrated with a time constant of 0.1 s) and printed on paper.

Measurement of EMG activity. The EMG signal was quantified using a morphometric semi-automatic system (Videoplan II, Kontron Electronics, Germany). Mean EMG activity ($\overline{\text{EMG}}$) was calculated from the area under the curve of the integrated signal. The intercept points between baseline and integrated EMG signal were considered as the limits. The same method was applied to quantify and remove the electrocardiographic (QRS complex) artefact. This was performed by arithmetic subtraction of the QRS complex areas from each EMG signal. Activity recorded from both muscles was normalized to that recorded during its maximal voluntary contraction (MVC), and expressed as percentage of MVC (%MVC). This method permitted intra- and interindividual comparisons.

Study protocol. The $\overline{\text{EMG}}$ activity from both muscles was recorded under three different conditions. Firstly, during the isometric MVC against a resistance. Whilst for LD subjects performed a forced adduction of the arm, for the *biceps brachii* they were asked to flex the arm from its physiological extended position. The maximum value obtained was chosen from three consecutive manoeuvres. Secondly, the muscles were evaluated during quiet breathing. Volunteers breathed through a low resistance two-way valve (Jaeger, Würzburg, Germany) with a pneumotachometer (Scenmate, Jaeger, Würzburg, Germany) inserted in the inspiratory circuit. Finally, activity of the muscles was registered while the subjects breathed under inspiratory overloads. Specifically, five levels of inspiratory threshold load (20, 40, 60, 80 and 100% of their P_{max}) were randomly tested. The first minute of breathing under each inspiratory load was considered as adaptation time without submission to muscle fatigue. EMG activity was quantified on the following five breaths for each level of inspiratory loading. Resting periods of 5 min were allowed between every step of load. Loads consisted of different weights hanging from a threshold valve placed on the inspiratory line. Opening pressure of the system for specific weights (1.9 cmH₂O per 10 g load) was previously determined.

Statistical analysis

Inspiratory loading and $\overline{\text{EMG}}$ activation of the muscles were analysed as independent and dependent variables, respectively. Results appear expressed as mean \pm SEM. Analysis of variance (ANOVA) for repeated measurements and slope analysis were used to compare activity recorded from LD with the activity recorded simultaneously from the *biceps brachii*. Pearson's coefficient (r) was used when appropriate. An alpha factor <0.05 was considered significant.

Results

All volunteers completed the entire protocol. During quiet breathing, tidal volume (V_T) was 490 \pm 110 ml, the inspiratory time (T_I) was 1.3 \pm 0.3 s, and the respiratory frequency (f_R) was 17 \pm 3 breaths \cdot min⁻¹. During loaded breathing, V_T and T_I decreased, whereas f_R increased. Thus, under 80% P_{max} loading, V_T was 320 \pm 160 ml, T_I 0.8 \pm 0.4 s and f_R 26 \pm 6 breaths \cdot min⁻¹.

Intraindividual variability coefficient of $\overline{\text{EMG}}$ was less than 7% for each situation. Analysis of the EMG, recorded from LD of three of the subjects carrying simultaneously surface and needle electrodes, indicated both no differences and a close correlation (r=0.975; p<0.001) between the two recording techniques (fig. 1).

Individual values of EMG activity at baseline and during the inspiratory loading are reflected in figure 2a (LD) and 2b (control muscle). During quiet breathing, activity of LD corresponded to 1.8 \pm 0.4% MVC, whereas brachial biceps activity was 2.6 \pm 0.3 %MVC. When inspiratory threshold loads were applied, $\overline{\text{EMG}}$ activity of LD increased linearly (tendencies analysis, p<0.001).

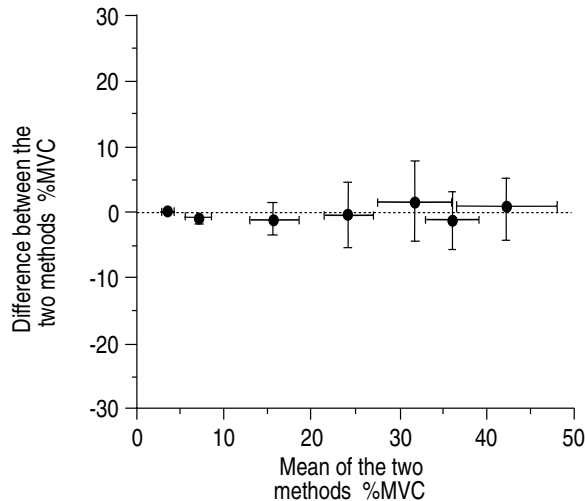


Fig. 1. – Concordance between surface and needle electrodes in the simultaneous recording of *latissimus dorsi* activity in three of the subjects breathing under different inspiratory loads. MVC: maximum voluntary contraction.

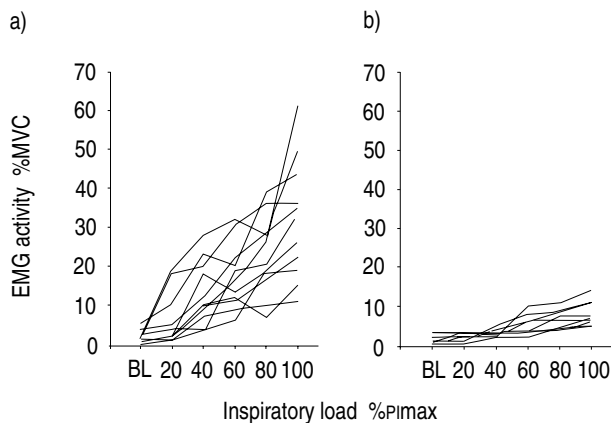


Fig. 2. – Individual activity of: a) *latissimus dorsi* muscle; and b) *biceps brachii* (control muscle) during inspiratory threshold loading. Pimax: maximal inspiratory pressure; MVC: maximal voluntary contraction; EMG: electromyographic; BL: baseline.

This change was evident ($p < 0.05$) from inspiratory loads corresponding to 40% Pimax and above when compared to the control muscle. The LD activity increased with a mean of $6.1 \pm 1.7\%$ MVC for every 20% Pimax increase in the inspiratory load. At 100% Pimax, LD showed a maximal activation, corresponding to $32 \pm 5\%$ MVC (range, 11–61%). At this level of load, mean activity of the control muscle (brachial biceps) was less than 7.5% MVC (fig. 2b). Maximal EMG activation was significantly higher for LD in terms both of absolute values ($p < 0.01$), and slope of the regression lines (slope analysis, $p < 0.05$). All subjects of our study were able to exceed EMG values recorded during the inspiratory manoeuvre when the MVC of LD was performed.

Discussion

The main finding of this study is the evidence that activity of *latissimus dorsi* muscle (LD) increases progressively

and linearly according to the inspiratory pressure generated.

In this study, participation of LD on the inspiratory movements was evaluated using an electrophysiological technique. A clear evidence of increase in LD activation was found during the inspiratory loading. Changes in LD activity were phasic (fig. 3) and directly associated with the magnitude of inspiratory loading (%Pimax). Both the visual analysis of the curves (fig. 2) and statistical analysis revealed a linear tendency in the EMG increase. The physiological significance of this activation in terms of respiratory work, oxygen up-take, or inspiratory pressure generated was not determined. Nevertheless, contribution of LD to breathing movements under inspiratory loads can be also analysed from an anatomical and mechanical point of view. Firstly, LD receives fibres from the lower three or four ribs. Thus, contraction of LD could result in elevation of the lower ribs during inspiration, particularly if the humerus is fixed [6]. Secondly, from a mechanical point of view, force developed for LD during the inspiratory effort could have been measured. However, this technique has some limitations to be applied in volunteers. Although an alternative method could have been to demonstrate a shortening of LD fibres, the absence of shortening in the muscle fibres does not rule out muscle work. In fact, an isometric contraction could be present to a best fixing of the rib cage and trunk [5].

It is worth noting that analysis of LD activity was focused on the inspiratory movements. Inspiratory loading was chosen because it might be related to the loads imposed by a chronic airway obstruction. In fact, in chronic obstructive pulmonary disease (COPD) patients dynamic compression of the airways during expiration is a limiting factor for the action of expiratory muscles while ventilatory loading. Compensatory mechanisms in these circumstances are mainly dependent on the activity of the inspiratory muscles [14]. If the inspiratory muscles, mainly the diaphragm, are functionally impaired (*i.e.* muscle fatigue, pulmonary hyperinflation), a progressive recruitment of additional muscles (termed accessory muscles) would be expected, in order to contribute to the inspiratory effort. We have recently found [12] indirect evidence of such a potential participation of LD under specific ventilatory circumstances. Therefore, structural changes were observed in the LD from COPD patients. Specifically, size of LD fibres directly correlated with the degree of airways obstruction. We hypothesize that these structural changes in LD from COPD patients could be associated with a chronic recruitment of the muscle. At this time, several studies have provided evidence to support this hypothesis. Firstly, GRONBAEK and SKOUBY [4] found that 40% of patients with airway obstruction *versus* 0% of controls exhibited inspiratory activity of LD muscle during quiet breathing. More recently, CALA *et al.* [5] demonstrated a recruitment of LD in healthy subjects when breathing both under resistive loading and at high lung volumes. Our results confirm that LD participates in the inspiratory efforts, and clearly demonstrate that recruitment of LD shows a phasic linear increase which is proportional to the inspiratory threshold load.

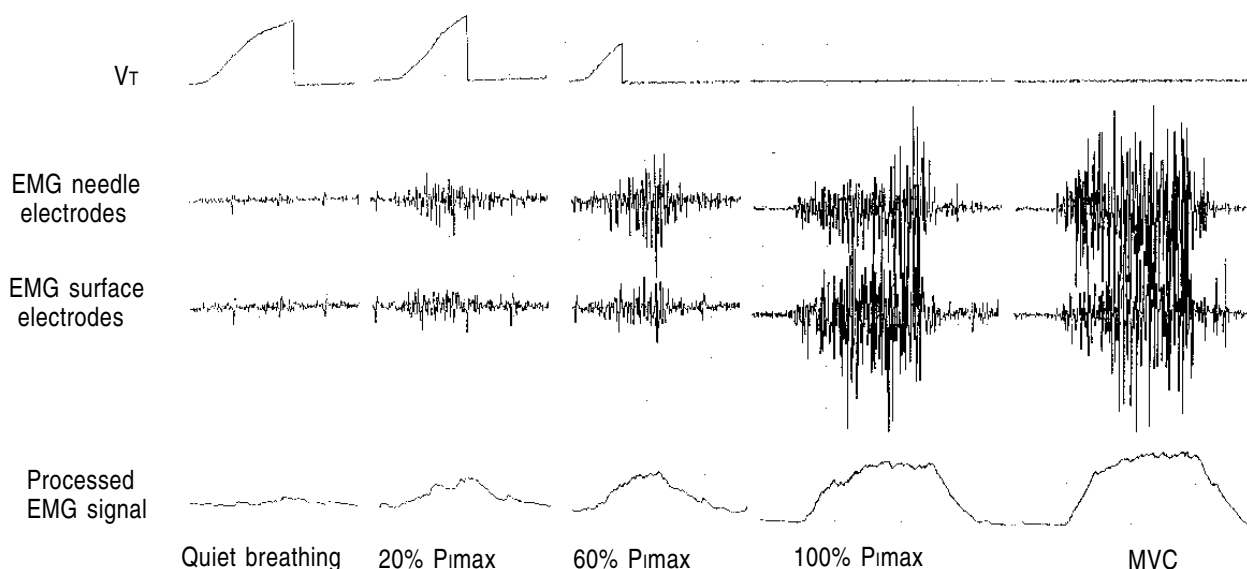


Fig. 3. – Inspiratory V_T and electromyographic signals of *latissimus dorsi* (LD) muscle from a subject simultaneously carrying needle and surface electrodes, during quiet breathing and during graded inspiratory loading. Processed (rectified and integrated) signal of the LD activity recorded via surface electrodes is also shown. For abbreviations see legend to figure 2.

LD muscle could participate in the inspiratory movements, both generating inspiratory pressure, with the shoulders as fixed points to raise the rib cage, and/or best-fixing the chest wall and trunk in order to optimize diaphragm contractions [5–7]. However, these findings do not necessarily exclude the possibility that LD could also participate in expiratory movements or cough. Depending on the sense of contraction, the costal fibres could potentially pull down the lower ribs, increasing the intra-thoracic pressure and contributing to a decrease in the cross-sectional area of the rib cage [5, 6]. In fact, a previous study found that 12% of patients with airway obstruction and 33% of controls actually recruited LD when expiring during forced breathing [4].

Several methodological points of this study should be discussed. Firstly, specificity of the surface electrodes or the potential contamination of EMG signals. Both LD and biceps belong to the most superficial anatomical plain and electrodes were placed directly above them. Thus, activity of adjacent deeper muscles, all of them smaller than LD (*i.e.* intercostals), do not appear as an important source of contamination. In addition, no differences were observed between recordings from surface and needle electrodes, when both techniques were simultaneously applied (fig. 1).

Secondly, the possibility of a variable inspiratory pressure during the same inspiratory load could be argued. This problem was partially avoided using a threshold valve, a system which implies a determined aperture pressure being always the same for a determined load. Despite the observed decrease of V_T during loaded breathing runs, we cannot exclude a potential increase in mouth pressure throughout the inspiration once the valve was opened.

Thirdly lung volumes were not normalized during the study. However, if functional residual capacity (FRC) changed this would not have invalidated the results, but

might rather be an additional explanation for them. In fact, CALA *et al.* [5] recently demonstrated that activity of LD increases in healthy subjects, particularly when breathing at high lung volumes under inspiratory resistive loading. As additional data, the inspiratory effort was standardized in the present study according to individual P_{max} , whereas EMG activity was normalized according to MVC. This normalization of EMG activity (*i.e.* %MVC) is statistically valid and likely to be more relevant to the study questions [15]. Thus, the present study makes it possible to establish the linear relationship between the magnitude of the inspiratory effort (according to P_{max}) and the increase in EMG activity of LD (referred to MVC). Moreover, differences found in this study between LD activity during the MVC manoeuvre and during 100% P_{max} loading (fig. 3) clearly demonstrate that a maximal inspiratory effort does not completely activate the LD motoneuron pool.

The muscle included as control (*biceps brachii*) showed a slight increase in EMG activity when submaximal inspiratory efforts were performed. The highest EMG of biceps was observed when breathing under loads of 100% P_{max} , although they were significantly lower than the values registered from LD (fig. 2b). We hypothesize that this activity recorded from the control muscle could represent: 1) a participation of the *biceps brachii* in fixing the humerus in order to optimize the effect of LD contraction; 2) a weak propagation of LD activity given that it is inserted in the humerus; and/or 3) a possible unspecific activation of some extrathoracic muscles when breathing at submaximum inspiratory loads.

In summary, this study clearly demonstrates that activity of *latissimus dorsi* muscle increases linearly during progressive inspiratory loads. Increase in LD activity was directly associated with the magnitude of the inspiratory pressure generated. These results and data from other previous studies [4, 5] strongly support the hypothesis

that LD can participate as an accessory muscle for inspiration. Therefore, we recommend that LD should not be considered an appropriate control for studies of the respiratory muscles. A trial comparing different training protocols for the respiratory muscles, including LD training, to fully evaluate its effects both on force and endurance of the respiratory muscles, seems to be warranted.

Acknowledgements: The authors are very grateful to M.A. Felez and J. Marrugat for helpful suggestions and statistical advice and to J.A. Salazar for his assistance in editing.

References

1. Cala SJ, Edyvean J, Rynn M, Engel LA. Oxygen cost of breathing: ventilatory vs pressure loads. *Aust NZ J Med* 1991; 21 (Suppl. 4): A666 (Abstract).
2. Campbell EJM. Accessory muscles. In: Campbell EJM, Agostoni E, Newson Davis J eds. *The Respiratory Muscles: Mechanics and Neural Control*. Philadelphia, P.A. Saunders, 1970; pp. 181–193.
3. Breslin EH, Garoutte BR, Kohlman-Carrieri V, Celli BR. Correlations between dyspnea, diaphragm and sternomastoid recruitment during inspiratory resistance breathing in normal subjects. *Chest* 1990; 98: 298–302.
4. Gronbaek P, Skouby AP. The activity of the diaphragm and some muscles of the neck and trunk in chronic asthmatics and normal controls. *Acta Med Scand* 1960; 168: 413–425.
5. Cala SJ, Edyvean J, Engel LA. Chest wall and trunk muscle activity during inspiratory loading. *J Appl Physiol* 1992; 73: 2373–2381.
6. Gardner MD, Gray DJ, O'Rahilly R. In: *Anatomy*. Volume II. Philadelphia; WB Saunders Co. 1963; pp. 142–143.
7. Testut L, Latarjet A. In: *Human Anatomy*. Volume I. Barcelona, Spain; Salvat Editores, 1979; pp. 861–864.
8. Delhez L. Contribution électromyographique a l'étude de la mécanique et du contrôle nerveux des mouvements respiratoires de l'homme. Liège, Vaillant-Carmanne, 1974; pp. 380.
9. Sanchez J, Brunet A, Medrano G, Debesse B, Derenne JPh. Metabolic enzymatic activities in the intercostal muscles and serratus muscles and *latissimus dorsi* of middle-aged normal men and patients with moderate obstructive pulmonary disease. *Eur Respir J* 1988; 1: 376–383.
10. Campbell JA, Hughes RL, Sahgal V, Frederiksen J, Shields TW. Alterations in intercostal muscle morphology and biochemistry in patients with obstructive lung disease. *Am Rev Respir Dis* 1980; 122: 679–686.
11. Field S, Sanci S, Grassino A. Respiratory muscle oxygen consumption estimated by the pressure-time index. *J Appl Physiol: Respirat Environ Exercise Physiol* 1984; 57: 44–51.
12. Orozco-Levi M, Gea J, Aran X, Minguella JM, Corominas JM, Broquetas JM. Structure of *latissimus dorsi* muscle and respiratory function. *J Appl Physiol* (in press).
13. Roca J, Sanchis J, Agustí Vidal J, Rodríguez-Roisin R. Spirometric reference values for a Mediterranean population. *Bull Eur Physiopathol Respir* 1986; 22: 217–224.
14. Pride NB. The role of respiratory mechanics in reducing ventilatory response to CO₂. *Bull Eur Physiopathol Respir* 1979; 15 (Suppl.): 75–84.
15. Matthews JNS, Altman DG, Campbell MJ, Royston P. Analysis of serial measurements in medical research. *Br Med J* 1990; 300: 230–235.