Scalene muscle activity during progressive inspiratory loading under pressure support ventilation in normal humans

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ABSTRACT

We hypothesized that (1) in healthy humans subjected to intermittent positive pressure non-invasive ventilation, changes in the ventilator trigger sensitivity would be associated with increased scalene activity, (2) if properly processed – through inspiratory phase-locked averaging – surface electromyograms (EMG) of the scalenes would reliably detect and quantify this, (3) there would be a correlation between dyspnea and scalene EMG. Surface and intramuscular EMG activity of scalene muscles were measured in 10 subjects. They breathed quietly through a face mask for 10 min and then were connected to a mechanical ventilator. Recordings were performed during three 15-min epochs where the subjects breathed against an increasingly negative pressure trigger (−5%, −10% and −15% of maximal inspiratory pressure). With increasing values of the inspiratory trigger, inspiratory efforts, dyspnea and the scalene activity increased significantly. The scalene EMG activity level was correlated with the esophageal pressure time product and with dyspnea intensity. Inspiration-adjusted surface EMG averaging could be useful to detect small increases of the scalene muscles activity during mechanical ventilation.

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1. Introduction

Quiet inspiration in healthy humans is mainly driven by the diaphragm, but it also involves the parasternal intercostals and the scalene muscles (Beau and Maissiat, 1843; Raper et al., 1966; De Troyer and Estenne, 1984; Gandevia et al., 1996; Hug et al., 2006; Saboisky et al., 2007). The scalene are recruited during inspiratory maneuvers, either static (Hudson et al., 2007) or dynamic (Raper et al., 1966; Katagiri et al., 2003; Hudson et al., 2007). Their tidal activity can increase in chronic diseases placing the diaphragm at mechanical disadvantage (chronic obstructive pulmonary disease (COPD) or kyphoscoliosis (De Troyer et al., 1994; Estenne et al., 1998)). Indeed patients with these conditions often exhibit palpable scalene inspiratory activity event when they are in stable condition. When breathing is acutely shifted to high lung volumes, the activity of the scalenes increases (Raper et al., 1966; Hudson et al., 2007). During spontaneous breathing trials in mechanically ventilated patients, irrespective of the underlying disease, palpable scalene muscle recruitment in inspiration can be a sign of respiratory distress (Pardee et al., 1984). In mechanically ventilated patients, the uncoupling of inspiratory neck muscle contractions from onset of machine breaths identifies certain forms of patient–ventilator asynchrony (Chao et al., 1997). Inspiratory neck muscle contractions can provide a useful clinical monitoring tool for the optimization of ventilator settings (Brochard et al., 1989). Inspiratory neck muscles are recruited progressively and intensely during incremental exercise in patients with chronic obstructive pulmonary disease (Yan et al., 1997). They are also recruited in critically ill patients failing a spontaneous breathing trial during mechanical ventilator weaning (Parthasarathy et al., 2007). Of note, there seems to be a relationship between the acute activation of inspiratory neck muscles and dyspnea, as pointed at as early as the beginning of the XIXth century (Magendie, 1816). The intensity of inspiratory neck muscle recruitment is associated with the intensity of dyspnea in healthy subjects submitted to experimental inspiratory loading, during either fatiguing protocols (Ward et al., 1988) or non-fatiguing ones (Bradley et al., 1986). The presence of a clinically visible inspiratory activation of inspiratory neck muscle is statistically associated with dyspnea in patients suffering from amyotrophic lateral sclerosis (Similowski et al., 2000).
The above elements are clear clues to the clinical relevance of studying the inspiratory recruitment of human neck muscles. Quantifying and monitoring their activity is however difficult. A compromise must be found between clinical examination, simple but hardly quantifiable, and intramuscular electromyographic (EMG) recordings, precise but too invasive and expertise-demanding for clinical applications. Within this frame, we have previously shown that phase-locking the averaging of surface scalene electromyograms to inspiration allowed an optimized detection of the scalene activity during quiet breathing and its quantification (Hug et al., 2006). The scalenes and sternomastoids are known to be recruited sequentially in humans. During progressive inspiratory effort, the activity of the sternomastoid typically starts well after the first half of the effort, whereas that of the scalene is noticeable as early as during the first tenth of the effort (Campbell, 1955; Raper et al., 1966; Hudson et al., 2007).

With these elements in mind, we hypothesized that (1) in healthy humans subjected to intermittent positive pressure non-invasive ventilation, modest increases in inspiratory loading induced by changes in the ventilator trigger sensitivity would be associated with increased scalene activity; (2) the inspiratory pressure-adjusted average surface electromyograms of the scalenes would reliably detect and quantify this increase in activity; (3) this would correlate with the intensity of respiratory discomfort.

2. Methods

2.1. Subjects

This study was part of an experimental program approved by the appropriate review board (Comité de Protection des Personnes se prêtant à des Recherches Biomédicales Pitié-Salpêtrière) and devoted to the study of patient–ventilator interactions from a model of normal volunteers receiving non-invasive mechanical ventilation. Ten healthy subjects participated in the study (six men, four women; aged 28.7 ± 2.0 years; height 176 ± 10 cm; weight 68.2 ± 13.8 kg). They were informed in detail of the purpose of the study and methods used, and gave written consent. During the experiments, they were seated in a comfortable chair with the arms and head supported, and had been instructed not to move or talk.

2.2. Measurements

The experimental setup is depicted by Fig. 1.

2.2.1. Pressure

Airway opening pressure was measured within an airtight facial mask (Pmask) (Comfort classic, Respironics, USA), using a linear differential pressure transducer (DP 15–34, range: ±200 cm H2O, Validyne, Northridge, CA, USA). Esophageal (Pes) and gastric (Pga) pressures were measured with two balloon-tipped catheters (thin-walled balloon sealed over a polyethylene catheter with distal side holes, 80 cm length, 1.4 mm internal diameter, Marquat, Boissy-St-Léger, France). The insertion of the catheters through the nose was carried out following topical anaesthesia (lidocain spray 10%). The gastric and esophageal balloons were inflated with respectively, 1 and 2 mL of air. The catheters were connected from the mechanical ventilator and asked to breath quietly for 10 min (i.e. Quiet Breathing condition; QB). At the end of this protocol, the subjects were disconnected from the ventilator. At the end of this protocol, the subjects were disconnected from the ventilator and asked to breath quietly for 15 additional minutes (washout condition, WO). Five of the 10 subjects were studied twice at a several weeks interval.

2.2.2. PetCO2

End-tidal CO2 was continuously measured through a dedicated port of the face mask using an infrared gas analyzer (IR505, Servomex SA, Saint-Denis La Plaine, France).

2.2.3. Surface EMG recordings

One pair of skin-taped silver cup electrodes 7 mm in diameter (Nihon Kohden, Tokyo, Japan) aimed at recording scalene EMG activity was placed in the posterior triangle of the neck (right side) at the level of the cricoid cartilage, so as to lie over the lower portion of the anterior scalene muscle. It was located during sniff maneuvers through palpation of the neck in the lower third of a line drawn between the middle of the mastoid process and the sternal notch. Another pair of electrodes was placed over the body of the right sternomastoid, 3 cm above the anterior head of this muscle. Within each electrode pair, the inter-electrode distance was 1 cm and impedance was kept below 2 kΩ by careful skin shaving and abrasion with an ether-saturated pad. The wires connected to the electrodes were carefully secured with tape to minimize movement artefacts. The common electrode was placed at the level of the manubrium sternum.

2.2.4. Intramuscular EMG recordings

In six subjects, a fine wire EMG electrodes (Inomed, Tullastrasse, Germany) was inserted into the left anterior scalene and sternomastoid muscles at the above-described locations, under real-time ultrasonographic guidance (Hewlett-Packard, Sonos 2000; probe = 5 MHz). The subjects were then asked to perform inspiratory maneuvers and opposed neck rotations to confirm the correct electrode placement.

2.2.5. Dyspnea

The intensity of dyspnea was rated using a visual analogue scale (VAS) constituted of a 100-mm horizontal scale over which the subjects had to place a cursor according to the intensity of their respiratory discomfort, between “none” (left) and “intolerable” (right) in response to the question: “How short of breath are you right now?”

2.3. Protocol

It is summarized by Fig. 1. In brief, the subjects first performed maximal inspiratory static maneuvers (of the Mueller type and from end-expiratory lung volume) in order to determine the maximal inspiratory pressure (Pimax) at the airway openings. Then, the balloon-tipped catheters and the EMG electrodes were placed and the subjects breathed quietly through the face mask for 10 min (i.e. Quiet Breathing condition; QB). After which they were connected to a mechanical ventilator (Servo i, Maquet SA, France) with an inspiratory support of +4 cm H2O to compensate for the resistance of the breathing circuit. Three 15’ epochs during which the subjects were confronted with an increasingly negative pressure trigger (approximately −5%, −10% and −15% of the maximal inspiratory pressure) were recorded. They were separated by 15’ epochs of quiet breathing after disconnection from the ventilator. At the end of this protocol, the subjects were disconnected from the mechanical ventilator and asked to breathe quietly for 15 additional minutes (washout condition, WO). Five of the 10 subjects were studied twice at a several weeks interval.
2.4. EMG processing

All EMG signals were fed to a Nihon Kohden Neuropack electromyograph (Nihon Kohden, Tokyo, Japan), with a 10 kHz sampling rate and were filtered (between 20 and 500 Hz and between 20 and 3 kHz for surface and intramuscular recordings, respectively). They were stored on an apple Macintosh computer for subsequent analysis (PowerLab, AD Instruments, Hastings, UK). Raw EMG data were root mean squared (RMS) with a time averaging period of 2 ms to quantify the activity level of the muscle.

The following procedure was then applied (Hug et al., 2006). For each condition in each subject, inspiratory efforts were identified from the Pmask signal (Chart 5.2, AD instruments, Hastings, UK). The continuous EMG RMS signal was then truncated in as many epochs as there were inspiratory efforts, each epoch starting 1 s before the beginning of the corresponding inspiratory effort and ceasing 2 s after its end and therefore containing the full inspiratory-related EMG activity. In the end, 40–50 consecutive such epochs of EMG, phase-locked to inspiration, were ensemble averaged. This produced a mean EMG RMS trace that was used for subsequent analysis (Fig. 1). Its mean value was used to quantify the intensity of the corresponding phasic inspiratory activity. All values were expressed in percentage of the activity so measured during quiet breathing (RMS%).

2.5. Statistical analysis

Data distributions consistently passed the Shapiro-Wilk normality test (Prism®4.01, Graphpad Software, San Diego, CA, USA). Values are therefore reported as mean ± SD. RMS% values were compared using an analysis of variance for repeated measures (subjects as the random factor, breathing condition as the inter-subject factor) with orthogonal contrasts as the post-hoc test (Statistix®, Tallahassee, FL, USA). The relationship between the scalene activity (RMS%) and the diaphragmatic contribution to inspiration (ΔPga/ΔPdi), the pressure time product of esophageal pressure (PTPes), ΔPes, ΔPga/ΔPes, the intensity of dyspnea were studied by calculating the Pearson product–moment correlation coefficient. This procedure was applied after correcting the data according to the normalization–renormalization procedure recommended by Poon (1988), to account for the possible distortions induced by pooling intra-subjects and inter-subjects measures (StatEL®, Paris, France). The degree of similarity of averaged RMS linear envelope patterns between intramuscular and surface recordings was assessed for each subject by using the cross-correlation technique. The cross-correlation coefficient (with lag time equal to zero, R0) was calculated according to the equation proposed by Li and Caldwell (1999) (custom written script, Origin 6.1, OriginLab Corporation, USA) from smoothed (triangular Barlett window width 3001 points) averaged signals. In the five subjects studied twice, the reproducibility of the
3. Results

3.1. Dyspnea

Dyspnea increased with the intensity of the inspiratory effort required to trigger the ventilator (Fig. 2).

3.2. Maximal inspiratory pressure and trigger values

$P_{i,max}$ amounted to $98 \pm 18 \text{ cm H}_2\text{O}$ on average for women and $118 \pm 6 \text{ cm H}_2\text{O}$ for men, within the normal range (ATS/ERS statement). In the four women, the trigger value was set to $-4.7 \pm 0.9 \text{ cm}$

trigger-related increases in the surface scalene RMS was assessed in terms of the $\eta^2$ coefficient of an intraclass correlation analysis (percentage of the total variability that reflects the tendency of two measures within any particular pair to have the same value) (Shrout and Fleiss, 1979). A $P$ value below 0.05 was considered indicative of statistical significance, namely of a less than 5% probability of erroneously rejecting the null hypothesis (type I error).
Fig. 4. Comparison of the scalene surface and intramuscular EMG patterns after inspiration-adjusted averaging (ventilator triggering set at 10% of maximal inspiratory pressure) in the six subjects where intramuscular recordings were performed. In all cases, the cross correlation coefficient ($R_0$) indicates a very high degree of similarity between intramuscular and surface recordings. For the sake of clarity the averaged EMG patterns are smoothed (triangular Barlett window width 3001 points). Of note, the surface EMGs are recorded on the right side, and the intramuscular ones are recorded on the left side.

$H_2O:\ -9.5 \pm 1.9 \text{ cm } H_2O$ and $-14.2 \pm 2.9 \text{ cm } H_2O$ for the 5%, 10% and 15% trigger condition, respectively. Because $P_{i, \text{max}}$ was very similar among the six men, the trigger values were the same for all of the $m$ (i.e. $-6, -12$ and $-18 \text{ cm } H_2O$ for, respectively, the 5%, 10% and 15% trigger condition).

3.3. $P_{\text{ETCO}_2$

No noticeable change in $P_{\text{ETCO}_2}$ was observed during the course of the study, both on average and on an individual basis.

3.4. Intramuscular EMG recordings

Two of the six subjects so studied reported a slight discomfort attributable to the insertion of the needle. In all the conditions, the scalene intramuscular recordings showed a phasic activity during tidal breathing with the exception of one subject (#6) during unloaded breathing (i.e. QB and WO conditions). Conversely, the sternomastoid intramuscular electrode was consistently silent, except in one subject (#6) when the trigger was set to 10% and 15% of $P_{i, \text{max}}$ (Fig. 3). Thus, the surface electrodes aimed at recording the sternomastoid picked up a cross-talk signal from the scalene. We, therefore, discarded this signal, and focused the analysis on the activity recorded by the surface electrodes lying over the anatomical landmark of the anterior scalene. Of note, this activity was consistently synchronous with the intramuscular one with the exception of one case during QB (subject #2) where the intramuscular signal disappeared intermittently from the recordings in spite of the persistence of the surface one.

3.5. Surface EMG recordings

The $P_{\text{mask}}$ triggered averaging of the surface EMG signal consistently evidenced a phasic inspiratory activity of the scalene muscle. This was true in all the subjects and all the conditions, with the exception of two cases during unloaded breathing (#6 and #9). The cross correlation analysis indicated a very high degree of shape similarity of the surface and intramuscular EMG patterns ($R_0 = 0.968 \pm 0.037$; ranging from 0.818 to 0.996) (Fig. 4). With increasingly intense inspiratory efforts, the surface scalene RMS increased significantly for the two triggers fixed at $-10\%$ and $-15\%$ of $P_{i, \text{max}}$, and returned to its initial value during the washout period (Figs. 5 and 6). Of note, the baseline EMG activity increased significantly during the 15% trigger condition, suggestive of an increased tonic activity.

Fig. 5. Example, in one subject, of the inspiration-adjusted average EMGs of the scalene surface and intramuscular recordings. From top to bottom: $P_{\text{mask}}$, mask pressure; Scal i, intramuscular EMG recording of the anterior scalene (fine wire electrode); Scal s, surface EMG recording of the anterior scalene. For sake of clarity the averaged EMG patterns are smoothed (triangular Barlett window width 3001 points). “QB” stands for “quiet breathing”, “WO” for “washout”. Ventilator triggering levels (“trigger”) are expressed in % of the maximal inspiratory pressure developed by the subjects.
The repeated experiment showed a fair reproducibility (\(R^2 = 0.81\)) of the trigger-related increases in the surface scalene RMS.

### 3.6. Repeated experiments

In the five subjects who could be studied on a second occasion, the repeated experiment showed a fair reproducibility (\(\eta^2 = 0.81\)) of the trigger-related increases in the surface scalene RMS.

### 4. Discussion

This study shows that in healthy subjects receiving inspiratory pressure support via a face mask, decreasing the sensitivity of the ventilator trigger in a stepwise manner (namely increasing inspiratory loading) is associated with a progressive increase in the EMG activity level of the scalene muscle. This increase correlates with the increasing magnitude of the inspiratory effort (as assessed by \(\Delta P_{\text{Pes}}\), the \(\Delta P_{\text{Pes}}/\Delta P_{\text{di}}\) ratio, or \(P_{\text{TPes}}\)). The RMS increase is strongly correlated with an increase in the self-rated intensity of dyspnea, in line with the relationship between respiratory discomfort and inspiratory neck muscle activity (Ward et al., 1988). The contribution of the diaphragm to the inspiratory effort does not vary, in contrast with the changes in task repartition that has been described in response to other types of inspiratory loading (Aliverti et al., 1997).

The sternomastoid muscle does not appear to be recruited within the loading range studied.

Of note, the trigger pressures used in our subjects may seem very high in absolute value, in comparison of what is encountered in intensive care patients. In fact, in proportion of maximal inspiratory pressures, they are not that high. This is because critically ill patients placed under mechanical often have greatly reduced inspiratory pressures (median maximal inspiratory pressure of 30 cm H$_2$O only—with a lower interquartile of 20 cm H$_2$O—in 79 such patients studied by De Jonghe et al. (2007): a ventilator trigger of −2 cm H$_2$O can correspond to 10% of the maximal inspiratory pressure). In addition, it must be kept in mind that many patients who have to trigger a ventilator also have to overcome an intrinsic positive end-expiratory pressure due to dynamic hyperinflation that can amount to several cm H$_2$O (Petrof et al., 1990). As a result, our experimental protocol appears realistic in the perspective of future intensive care applications.

#### 4.1. Sequence of recruitment of inspiratory neck muscles

With one exception, our subjects did not exhibit sternomastoid recruitment in response to the increasing inspiratory loading in spite of a marked increase in the scalene activity. This is consistent with the current knowledge of about the recruitment of inspiratory neck muscles. Hudson et al. (2007) have shown that during both static and dynamic inspiratory efforts, the recruitment of the sternomastoid is delayed until about 20% of the maximal inspiratory pressure has been developed (and at times much later depending on the subjects). This sequence of recruitment that is in line with the greater mechanical advantage of the scalene as compared to the sternomastoid (Legrand et al., 2003) is unaffected by changes in lung volume. In our study, the higher load chosen was of 15% of maximal inspiratory pressure. The absence of sternomastoid activation is therefore not surprising. It also supports the idea, expounded by Hudson et al. (2007), that the recruitment threshold of the scalene and sternomastoid is not sensitive to the type of inspiratory efforts performed. Hudson et al. (2007) asked their subjects to perform either static efforts at constant lung volumes against a closed airway, or dynamic efforts during which lung volume increased from FRC to TLC. They found that this did not markedly change the sequence and timing of the respective scalene and sternomastoid activation. The type of effort performed by our subject was more complex in nature, the first part of inspiration being devoted to reach the ventilator trigger and performed at constant lung volume (“static”), the second part being devoted to the production of tidal volume (“dynamic”) with the aid of the ventilator. Yet, as during simpler types of efforts, the threshold of scalene activation appeared to be low, and in any case lower than that of the sternomastoid.

From another point of view, our study also concords with the idea that the nature of the neural command involved in a given inspiratory activity does not modify the scalene-sternomastoid sequence. The scalene muscles are recruited early and the sternomastoid only late in the course of the response to a stimulation of the automatic respiratory command by carbon dioxide (Campbell, 1955). In the study by Hudson et al. (2007), where the scalenes were also recruited early and first, the subjects performed volitional efforts involving the primary motor cortical representation of inspiratory muscles. Our subjects, who were naive to respiratory physiology experiments, were confronted to sustained inspiratory mechanical loading. In this setting, load compensation occurs—attested to by the maintenance of PETCO$_2$ — and involves cortical mechanisms (Raux et al., 2007a,b) that are probably behavioral rather than volitional strictly speaking. Yet, the scalenes were also recruited before the sternomastoid in our subjects. Again this supports the notion that the sequence of the inspiratory activation of the scalene and the sternomastoid is neurally preset and does not depend on the source(s) of the inspiratory command. Of note, it is consistent with the respective mechanical advantage of these muscles (Hudson et al., 2007).
4.2. Significance and usefulness of surface scalene EMG recordings

The electromyographic activity of the scalenes can be studied using intramuscular or surface electrodes. Intramuscular electrodes provide high quality signals, but they sample a limited number of motor units and their invasive nature limit their use in clinical practice. Surface electrodes give a more global picture of muscle function and are easy to use for prolonged or repeated studies, with a reasonable inter-experiment reproducibility (Duiverman et al., 2004). Their use, however, raises the question of signal contamination due to the cross-talk of adjacent muscle groups. Indeed, surface electrodes placed over the anatomical landmark of the scalene muscles will inevitably pick-up sternomastoid activity if it is present, and reciprocally, making the interpretation of the signal difficult. Of note on this, our study confirms that surface recordings are not appropriate to study the recruitment of the sternomastoid, even though they are often used with this purpose (Mananas et al., 2000; Ribeiro et al., 2002; Tassaux et al., 2002; Ratnovsky et al., 2003; Perlovitch et al., 2006). The platysma could also be a source of signal contamination. Available data however suggest that the inspiratory recruitment threshold of this postural muscle is very high (Fitting et al., 1988) and that its activation occurs in very particular clinical conditions such as tetraplegia (De Troyer et al., 1986) or very severe respiratory distress in ICU patients (personal observations, unpublished). Recruitment of the platysma is thus not likely to have occurred in our subjects. In addition, postural activity of the platysma should also not have been an issue, given the inspiratory-adjusted nature of our EMG averaging approach.

What can we infer from our results regarding the potential usefulness of surface recordings of inspiratory neck muscles in acutely ill patients? In such patients, sternomastoid activation hallmarks the severity of a given clinical condition. In the weaning study of Parthasarathy et al. (2007), sternomastoid activity was noted after the first minute of a spontaneous breathing trial in 8 out of 11 patients who failed this trial. These patients had very low maximal inspiratory pressures (32.7 cm H2O on average) and at the beginning of the spontaneous breathing trial, their inspiratory effort amounted to 11.3 cmH2O, namely more than 30% of Pimax. At the end of the trial all these patients exhibited sternomastoid activity and their average inspiratory effort amounted to 18.7 cm H2O, more than half of the available Pimax. Conversely, only three of the eight weaning success patients exhibited sternomastoid activity during the trial, in line with a ratio of inspiratory effort to Pimax that was generally well below 30%. These observations are in keeping with the results of Hudson et al. (2007) and they emphasize the notion that sternomastoid activation in respiratory distress probably has a strong negative prognostic value. Although Parthasarathy et al. (2007) did not assess the activity of the scalenes in their patients, it is almost certain that such an activity was present and would have been picked up by surface electrodes, as indicated by our results. All in all, surface recordings are not likely to provide an appropriate answer to the question “are the sternomastoid activated?” This sets a limit to their value as prognostic indicators during respiratory distress, but in practice there is no need of an EMG approach to answer this question. In contrast, identifying the activation of the scalenes can be more difficult, let alone quantifying it. Yet answering the question “are the scalenes activated and how much?” in situations where loading is not sufficient to activate the sternomastoid is putatively important. This is subverted by the correlation that exists between scalene EMG and dyspnea. This correlation was first documented by Ward et al. (1988) during experimental respiratory muscle fatigue protocols involving high level of inspiratory loading. We found a similar correlation in our subjects even though the intensity of loading was much lower than in the study by Ward et al. (1988). This suggests that the relationship between scalene activity and dyspnea is linear and has a low threshold, slight increases above the resting activity being likely to be associated with respiratory discomfort. Of note, we observed this relationship in individuals subjected to intermittent positive pressure ventilation that is known to exert a non-chemical inhibitory influence on respiratory drive (Simon et al., 1991; Leeviers et al., 1993; Fauquiers et al., 1998). This phenomenon was therefore not of sufficient magnitude to stamp out the effects of scalene recruitment on respiratory sensations. It could have changed the slope of the relationship, but the design of the study was not meant to assess this hypothesis.

In the absence of sternomastoid activation, as in our subjects, the similarity in shape and timing evidenced by cross-correlation analysis of the intramuscular and surface scalene EMG patterns that we observed (Fig. 4) indicates that the averaging method that we used can reliably identify changes in scalene activity over time, from surface recordings. This result must be seen as a mere proof of concept, and requires corroboration in mechanically ventilated patients receiving non-invasive or invasive ventilation. If such corroboration is obtained in the future, then real time scalene RMS at the bedside could allow clinicians to monitor the neural reactions to small changes in inspiratory loading. This is clinically relevant, because identifying an increase in scalene activity could then alert clinicians to subtle or impending respiratory discomfort. This would be the case even in the absence of actual patient–ventilatory asynchrony (Chao et al., 1997), and would have the advantage of occurring far earlier than inspiratory loading induced sternomastoid recruitment. This would be particularly important in mechanically ventilated patients who, for whatever reasons, are not capable of full communication with their caregivers (e.g. intubated and partially sedated patients). Real time scalene RMS could then become an adjunct to the current methods used to adjust mechanical ventilation to the demands of the patients. Of note, the evaluation the activity of extradiaphragmatic inspiratory muscles of the neck or of the chest wall must be seen as complementary of the evaluation of the activity of the diaphragm, as it is used for example using esophageal EMG probes during neurally adjusted ventilatory assistance.

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Conflicts of interest: This study did not involve any commercial or financial conflict of interest. Two of the authors (TS, MR) are listed as inventors on patent WO/2008/006963 “Device for detecting the improper adjustment of a ventilatory support machine used on a mammal”, that describes various neurophysiological approaches (EEG and EMG) to detect and quantify the neural reactions induced by “ventilator fighting”.

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