Correlations between Dyspnea, Diaphragm and Sternomastoid Recruitment during Inspiratory Resistance Breathing in Normal Subjects*

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The purpose of this study was to determine the relationship between recruitment of the DI and SM muscles measured as EMG signal amplitudes, the pattern of respiratory muscle recruitment measured with inductive plethysmography, and the intensity of the sensation of dyspnea, measured with 100 mm VAS. Eighteen normal subjects between the ages of 33 and 47 breathed under two conditions: normal controlled breathing and breathing against an inspiratory resistance at 60 percent of their maximal inspiratory pressure (MIP). The PM, RR, duty cycle (Ti/Ttot), and Vr were held constant. During resistance breathing, VAS dyspnea was increased when EMG-

Dyspnea, the distressful sensation of uncomfortable breathing, is the primary activity-limiting symptom in COPD. The physiologic mechanisms of dyspnea have been related to the intrinsic characteristics of the inspiratory muscles. Although COPD is characterized by reductions in expiratory flow, dyspnea is perceived during inspiration and better associated with inspiratory muscle function. A major focus for research in the field has been on pulmonary function. However, limited correlations have been found between dyspnea and alterations in pulmonary function. The association between dyspnea and respiratory muscle function has led to a paradigm shift in the study of dyspnea from pulmonary to respiratory muscle function.

Dyspnea is associated with the relationships between respiratory muscle force and (a) muscle fiber length, (b) the frequency of motor unit action potentials, and (c) the velocity of muscle contraction. Important determinants in the development of dyspnea are the force required of the respiratory muscles, their maximal capacity to produce force, and the ratio between these factors. Dyspnea increases with an increase in inspiratory muscle resistance and with a reduction in maximal force produced by the inspiratory muscles. Temporal parameters of muscle contraction, such as an increase in the duration of muscle contraction or duty cycle Ti/Ttot and an increase in RR, also have demonstrated relationships with the sensation of dyspnea.

Clinicians have observed that patients with COPD who recruit accessory neck and rib cage muscles in ventilation are more likely to report an increase in the sensation of dyspnea. Little is known about the relationship between specific respiratory muscle recruitment and the sensation of dyspnea, however. The purpose of this laboratory investigation was to determine the relationship between recruitment of the DI and SM muscles measured as the average peak amplitude of the EMG signal, the pattern of respiratory muscle recruitment as deduced from the pattern of thoracoabdominal motion and the intensity of the sensation of dyspnea.

DI = diaphragm; SM = sternomastoid; EMG = electromyogram; VAS = visual analog scales; PM = mouth pressures; Pdi = transdiaphragmatic pressure

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**METHOD**

Eighteen normal adult subjects participated in this laboratory investigation. The study was approved by the Committee on Human Research, University of California, San Francisco. Informed consent was obtained from each subject. Respiratory muscle recruitment and dyspnea were investigated under two conditions, normal breathing and breathing against an inspiratory resistance. While subjects were seated in an upright position electromyographic signals of the diaphragm (EMG-DI) and sternomastoid (EMG-SM) muscles and the pattern of thoracoabdominal motion were recorded continuously. Dyspnea was rated immediately after each trial by subjects using VAS. Two-minute trials were divided into four 30-s time intervals (isotimes) for data analysis. Subjects served as their own controls.

Four ventilatory parameters were maintained constant during each breathing condition. With normal breathing, Vr obtained from a calibrated pneumotach (Bionex 400) and PM measured with a differential pressure transducer (Cavlico: P551-5D-E1A) were voluntarily controlled by subjects using visual feedback from an oscilloscope. A RR of 18 breaths per minute and duty cycle T/Tm = 0.5 were held constant by having the subject breathe in time with a metronome. Concurrent calibrated measures of pulmonary parameters were continuously displayed on a strip chart recorder with other physiologic parameters for later analysis.

During the second pulmonary condition, inspiratory resistance breathing, subjects generated FMt equivalent to 60 percent of their MIP. The MIP was measured from residual volume according to standardized procedures. The resistance device was a tube with an adjustable orifice, such that inspiratory resistance could be set for the appropriate PM while subjects breathed at a constant inspiratory flow with control of breathing rate (RR = 18), duty cycle (T/Tm = 0.5), and Vr. A two-way valve allowed subjects to exhale without resistance.

The DI and SM recruitment was measured as average maximum peak to peak amplitude of the EMG signal quantified from the paper recording. The EMG-DI was obtained using an esophageal electrode (No. 8 French tubing containing nine internal electrodes spaced 1 cm apart). Specific electrodes for recording were selected for maximal EMG signal with voluntary diaphragmatic activation. The esophageal tubing was taped securely to the nose. To record EMG-SM, gold-plated bipolar surface electrodes were placed over the muscle 2 cm apart. The EMG signals were amplified (Tetronix TM 500), filtered from 10 to 10 kHz (Honeywell 1508C Visiscorder), and refiltered with a 20-Hz high-pass filter band. The five middle and consecutive EMG amplitudes within each isotime were averaged, the measurement of which was made between interfering electrocardiogram signals. The average EMG amplitude was then converted to millivolts based on the linear relationship between the amplitude and voltage of the signal at specific amplifier gains. To verify the accuracy of the EMG signal amplitude measurements, 50 randomly chosen EMG signals were independently measured by two of the investigators. The intrarater reliability coefficient in the measurement of EMG amplitudes was 0.92 (p = 0.0001).

Rib cage and abdominal excursions were recorded continuously using inductive plethysmography (Respirtrac Ambulatory Monitoring, White Plains, NY). To determine the category of chest wall movement, inductor bands were placed around the chest wall. The superior border of the rib cage band was placed at the level of the axilla and the inferior border of the abdominal band was 4 cm below the umbilicus. Standardized calibration of the phase difference between the two waveforms and spirometry was carried out. The five middle wave forms per isotime were analyzed for ventilatory motion patterns, based on the phase relationship and the direction of waveforms. Synchronous breathing was defined as simultaneous in-phase motion of the rib cage and abdomen during ventilation (Fig 1). Dysynchronous breathing was identified as waveforms showing out-of-phase rib cage and abdomen movement during ventilation (Fig 2). Dysynchronous breathing ranged from some lag between rib cage or abdominal movement to complete paradoxic breathing, such that when the rib cage moved out the abdomen moved in.

Dyspnea or breathlessness was defined as the sensation of uncomfortable breathing. The intensity of the sensation of dyspnea was measured with a VAS, a horizontal line, 100 mm in length, with descriptors of the sensation of dyspnea from "no breathlessness" at one end of the line to "extremely severe breathlessness" at the other. The subject was asked to rate the maximum breathlessness or the sensation of uncomfortable breathing felt during each trial. Subjects marked the point on the line that represented the level of breathlessness experienced immediately after each trial. Each rating was recorded on a separate scale form, eliminating visibility of previous sensation ratings.

**Data Analysis:** The differences between EMG-DI and EMG-SM during normal breathing and inspiratory resistance breathing were determined using paired t tests. Spearman rho correlations were used to determine the relationship between dyspnea and EMG-DI and EMG-SM during the last minute of the trials. The acceptable level of statistical significance for each test was a two-tailed probability value of less than 0.05 (p<0.05).

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**Figure 1.** Electromyographic signals of crural DI (EMG, DI, CRU), costal DI (EMG, DI, COS), sternomastoid (EMG, SM), parasternal (EMG, PS) and abdomen (EMG, AB); PM, rib cage motion (RC), abdomen motion (AB), and expiratory VT from a subject during arm rest. Arrows denote increase in pressures, RC and AB excursions and VT at onset of inspiration. Note synchronous excursions of the rib cage and abdomen.
RESULTS

Fourteen female and four male subjects ranged in age from 33 and 47 years, mean ± SD, (38.5 ± 4.4). Their pulmonary functions were within 80 percent of predicted values. The weight of each subject was within 20 percent of the predicted values for gender, age and height.

Compared with normal breathing, inspiratory resistance resulted in statistically significant increase in mean ± SD: dyspnea scores (4.7 ± 2.5 mm vs 37 ± 23 mm) (p = 0.0001), EMG-DI (0.10 ± 0.05 mV vs 0.27 ± 0.05 mV (p<0.01); and EMG-SM (0.08 ± 0.06 mV vs 0.32 ± 0.20 mV) (p = 0.07). In addition, there was a change in the overall pattern of respiratory muscle recruitment. In contrast to normal breathing that was associated with synchronous thoracoabdominal motion in 88 percent of the observed measures, dysynchronous breathing patterns were observed in 99 percent of the measures analyzed during inspiratory resistance.

The correlation between dyspnea and mean EMG-DI amplitudes during the last minute of inspiratory resistance breathing was r = -0.59 (p = 0.05). In contrast, resistance breathing resulted in a statistically significant positive correlation between dyspnea and mean EMG-SM amplitudes during the last minute of the trial (r = 0.31; p<0.01). As shown in Table 1, increases in the sensation of dyspnea were strongly associated with the pattern of thoracoabdominal motion.

DISCUSSION

There were three major findings from this study. Resistance breathing resulted in an inverse relationship between dyspnea and the recruitment of the DI during inspiratory resistance breathing. Conversely, during resistance breathing, EMG-SM increased in association with significant increases in the sensation of dyspnea. Finally, rib cage and accessory muscle recruitment manifested by the predominance of breathing dysynchrony was associated with an increase in the sensation of dyspnea.

The EMG amplitude of a muscle is an estimator of its drive and recruitment. Increased drive to the respiratory muscles has been hypothesized as one of the mediators in the generation of the sensation of dyspnea. In view of this, an increase in EMG amplitudes of the respiratory muscles would be expected to correlate with the sensation of dyspnea.

Recruitment of the diaphragm and sternomastoid muscles increased significantly during resistance breathing. In the maintenance of large and fatiguing resistance loads, the contribution of these muscles to the ventilatory effort also varied over time, such that, as the diaphragm decreased activity, SM recruitment increased. Coordination and synergistic activity of the respiratory muscles has been reported with increased ventilatory loads, possibly in the prevention or adaptation to the respiratory muscle fatigue process. In the present study, negative correlations between dyspnea and DI EMG signal amplitudes were observed during resistance breathing. Thus, the sensation of dyspnea decreased as the drive to and recruitment of the DI increased. In contrast, there was a positive correlation between SM recruitment and the sensation.
of dyspnea, as well as an association between dyspnea and the pattern of rib cage and accessory muscle recruitment. We speculate that the negative correlations found between dyspnea and diaphragmatic muscle EMG activity are dependent on the presence of rib cage and accessory muscle recruitment. No relationship was observed between dyspnea and DI recruitment during normal breathing (without added resistance), nor was there recruitment of the rib cage and accessory muscles.

The diaphragm may lack the sensory receptors necessary to mediate the sensation of dyspnea, so that an increase in recruitment of these muscles could not result in dyspnea. It is poorly supplied with muscle spindles, afferent receptors which some have hypothesized to mediate the sensation of dyspnea. On this theoretical basis, a correlation between dyspnea and DI EMG amplitudes would not be expected. Although these findings do not imply cause and effect relationships, they do tend to support the suggestion that accessory spindle afferents may be involved in the generation of the sensation of dyspnea.

In corroboration with the present findings, it has been demonstrated that dyspnea is not associated with dysfunction of the DI. Kontragunta and co-workers studied eight patients with moderate to severe COPD (mean FEV₁ = 1.08 L) during endurance leg cycling exercises, noting the presence of severe dyspnea in the absence of diaphragmatic fatigue. These investigators defined diaphragmatic fatigue as the inability to attain a target Pdi during the final moments of the endurance run. Mier-Jedrzejowicz and colleagues observed no relationship between reduction in Pdi and the sensation of dyspnea in 30 nonpulmonary patients with diaphragm weakness and breathlessness. Bradley and co-workers also observed that the rating of inspiratory effort, an indicator of dyspnea, by six young male subjects breathing against a resistance did not relate to the presence or absence of diaphragmatic fatigue. In a study of five normal subjects, Ward and associates recently reported an increase in respiratory effort with diaphragmatic fatigue that was not associated with the level of diaphragm activation nor the level of contraction. Finally, Celli and colleagues observed a greater intensity of dyspnea with arm activities than with leg activities at similar Pdi pressures, suggesting that diaphragmatic dysfunction may be of limited importance in the development of dyspnea.

In contrast, evidence from the present study and other investigations suggests an association between recruitment of the rib cage and accessory muscles of respiration and the sensation of dyspnea. In patients with COPD, paradoxic motion of the abdomen has been observed in association with the sensation of dyspnea as noted by Sharp and co-workers. Delgado and colleagues reported an increase in the sensation of dyspnea in association with paradox thoracoabdominal motion during exercise testing in COPD. During unsupported arm exercise, Celli and colleagues observed an increase in the sensation of dyspnea with rib cage and accessory muscle recruitment. The latter two investigations also demonstrated that rib cage and accessory muscle recruitment and the sensation of dyspnea were associated with a reduction in exercise endurance. Study of the relationship between dyspnea and drive to other primary and accessory respiratory muscles, including muscles with an abundance of muscle spindles, may enhance understanding of the complex neurophysiologic mechanisms in the generation of the sensation of dyspnea.

Clinical implications of these findings lie in the development of treatment strategies to reduce dyspnea. A commonly used treatment for dyspnea supported by these data is diaphragmatic breathing. The beneficial mechanisms in the treatment of dyspnea with diaphragmatic breathing have contributed to the reduction in RR and the increase in V̇. During periods of increased ventilatory work, this study demonstrated an inverse association between dyspnea and diaphragm activity and a positive correlation between dyspnea and rib cage and accessory muscle recruitment. In patients with COPD, a shift in the work of breathing from rib cage and accessory muscles to the DI may result in a reduction in dyspnea. Investigation, with quantitation of the sensation of dyspnea, is necessary to establish the efficacy of diaphragmatic breathing in the reduction of dyspnea in patients with COPD.

In summary, this study has demonstrated negative correlations between the sensation of dyspnea and recruitment of the DI in normal adult subjects breathing against high resistive loads. This occurred only in the presence of rib cage and accessory muscle recruitment. Dyspnea was positively correlated with recruitment of the SM muscle. Knowledge of the relationship between dyspnea and recruitment of specific respiratory muscles may guide the development of specific strategies in the clinical treatment of dyspnea. The present study indicates that a shift in ventilatory work from the rib cage and accessory muscles to the DI with voluntary diaphragmatic breathing may reduce the sensation of dyspnea. Investigation is necessary to better understand the mechanisms of dyspnea in relation to specific respiratory muscles and to develop therapeutic strategies for coping with activity-limiting dyspnea in COPD.

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