Crural diaphragm activation during dynamic contractions at various inspiratory flow rates

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Beck, Jennifer, Christer Sinderby, Lars Lindström, and Alex Grassino. Crural diaphragm activation during dynamic contractions at various inspiratory flow rates. J. Appl. Physiol. 85(2): 451–458, 1998.—The purpose of this study was to evaluate the influence of velocity of shortening on the relationship between diaphragm activation and pressure generation in humans. This was achieved by relating the root mean square (RMS) of the diaphragm electromyogram to the transdiaphragmatic pressure (Pdi) generated during dynamic contractions at different inspiratory flow rates. Five healthy subjects inspired from functional residual capacity to total lung capacity at different flow rates while reproducing identical Pdi and chest wall configuration profiles. To change the inspiratory flow rate, subjects performed the inspirations while breathing across two different inspiratory resistances (10 and 100 cmH2O · l–1 · s), at mouth pressure targets of –10, –20, –40, and –60 cmH2O. The diaphragm electromyogram was recorded and analyzed with control of signal contamination and electrode positioning. RMS values obtained for inspirations with identical Pdi and chest wall configuration profiles were compared at the same percentage of inspiratory duration. At inspiratory flows ranging between 0.1 and 1.4 l/s, there was no difference in the RMS for the inspirations from functional residual capacity to total lung capacity when Pdi and chest wall configuration profiles were reproduced (n = 4). At higher inspiratory flow rates, subjects were not able to reproduce their chest wall displacements and adopted different recruitment patterns. In conclusion, there was no evidence for increased demand of diaphragm activation when healthy subjects breathe with similar chest wall configuration and Pdi profiles, at increasing flow rates up to 1.4 l/s.

diaphragm; electromyogram; force-velocity relationship

The force-velocity relationship of skeletal muscle predicts that the maximum force generated by a muscle is a function of its velocity, as demonstrated in vitro by Hill (7). In the human in situ diaphragm, Pengelly et al. (11) demonstrated that transdiaphragmatic pressure (Pdi) decreased linearly with increasing flow for peak inspiratory flows up to 2 l/s when the contractions were elicited by submaximal unilateral stimulation of the phrenic nerve. They attributed their findings to the force-velocity characteristics of the diaphragm. The functional implication of these results is that there should be an increase in diaphragm activation (i.e., increase in the number of motor units recruited and/or in motor unit firing rate) to maintain the same mechanical output because the diaphragm seems to become weaker with increasing inspiratory flow rates.

With respect to voluntary breathing, Younes (13) has estimated that during resting ventilation (~1 l/s) the velocity of shortening is ~2% of the maximum velocity and that the diaphragm contracts at the low-velocity region of the force-velocity curve. Therefore, according to Younes (13), a small to moderate change in inspiratory flow should have little impact on the diaphragm activation required to maintain a given tension (at a given length).

To our knowledge, only Goldman et al. (6) have studied the influence of velocity of shortening [inferred from inspiratory flow with outward abdominal (Ab) displacement] on the relationship between diaphragm activation, evaluated via the diaphragm electromyogram (EMG), and diaphragm force generation, evaluated by the Pdi, at a given chest wall configuration, during voluntary contractions. Their results suggested that increases in inspiratory flow at low flow rates (flows ranged from 0.05 to 0.4 l/s) demand large increases in diaphragm activation to generate a given Pdi, which contradicts the theoretical predictions of Younes (13). These discrepancies could very well be due to the methodology and experimental design used in the study by Goldman et al. (6). At the time their experiment was performed, the EMG methodology available was not able to ensure that the distance between the diaphragm and the esophageal electrode was kept constant. Therefore, for reasons that have previously been discussed (2, 4), systematic errors could have been introduced into the signal, particularly during dynamic breathing maneuvers. In addition, the extreme voluntary chest wall configurational pathways that they studied may have easily altered the "natural" activation patterns of the costal and crural portions of the diaphragm.

We have developed diaphragm EMG acquisition and analysis methods to reliably obtain a signal with maintained muscle-to-electrode distance (12). The present study uses this improved diaphragm EMG methodology to evaluate whether there is a measurable increase in neural drive to the diaphragm when a subject is breathing at increasing inspiratory flow rates, for a given Pdi. Different from the experiment of Goldman et al. (6), our subjects performed inspirations from functional residual capacity (FRC) to TLC with spontaneously chosen trajectories of chest wall configuration on a Konno and Mead diagram (9).
METHODS

Subjects. Five healthy men agreed to participate in the protocol. Their anthropometric data are provided in Table 1. All subjects were experienced in performing respiratory maneuvers. The experimental setup is shown in Fig. 1. The study was approved by the Research and Ethics Committee of the L. C. Simard Research Center, Notre-Dame Hospital, Montreal, Quebec, Canada.

Signal acquisition. Diaphragm EMG signals were obtained via an esophageal electrode that consisted of nine rings placed 10 mm apart, creating an array of eight sequential, differential, bipolar electrode pairs. A schematic representation of the electrode is presented in Fig. 1, left.

Two Teflon tubes were placed inside the silicone tubing (diameter = 0.75 mm), and two 5-cm-long, 1.5-cm-diameter latex balloons were mounted ~10 cm below the most distal EMG ring to allow for measurements of gastric pressure and 2 cm above the most cephalad ring to allow for measurements of esophageal pressure. The two balloon catheters were connected to two differential pressure transducers (±350 cmH2O). Mouth pressure (Pm) was measured via a side port in the mouthpiece, which was connected to a third differential transducer (±350 cmH2O). Inspiratory flow was measured with a pneumotach (Fleisch no. 2). A two-way valve (model 2600, Hans Rudolph, Kansas City, MO) was connected to the pneumotach, such that we could alter the resistance on the inspiratory side while keeping expiration unobstructed. We monitored rib cage (RC) and Ab displacements (Respitrace) throughout the protocol in the form of a Konno and Mead diagram (9), with RC displayed on the y-axis and Ab displayed on the x-axis of a storage oscilloscope (see Fig. 1, middle).

Diaphragm EMG signals from electrode pairs 1–8 were amplified (Burr-Brown INA102) and high-pass filtered at 10 Hz, with an antialiasing filter at 1,000 Hz (Frequency Devices D70L8L, 8-pole Bessel filter). Diaphragm EMG signals were acquired and digitized by an analog-to-digital converter (2821, Data Translation), with 12-bit resolution, at a sampling frequency of 2,000 Hz, and stored on hard disk for offline analysis. Flow, Pm, esophageal pressure, gastric pressure, and RC and Ab data were acquired simultaneously with the EMG (DT 2811, Data Translation) at a sampling frequency of 100 Hz. All signals related to the diaphragm EMG and respiratory mechanics could be observed online during the experiment.

Experimental protocol. Subjects were studied while seated upright, facing the computer monitor, which gave them feedback about Pm, and the storage oscilloscope, which gave them feedback about chest wall displacements. Respitrace bands were positioned on the subjects and secured. The esophageal electrode was passed through the nose, swallowed, and positioned at the level of the gastroesophageal junction by feedback from an online display on the computer monitor of the diaphragm EMG signals from all eight electrode pairs. Once the diaphragm was located at the center of the electrode array, the electrode was fixed externally at the nose. The correct position of the esophageal pressure balloon was confirmed by the so-called “occlusion test” (1).

Table 1. Subjects’ anthropometric data

<table>
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<th>Subject No.</th>
<th>Age, yr</th>
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Fig. 1. Experimental setup. Left: esophageal electrode used in present study. Balloons were mounted on same catheter to measure esophageal pressure (Pes) and gastric pressure (Pga). Middle: Konno and Mead diagram [y-axis, rib cage (RC) displacement; x-axis, abdominal (Ab) displacement] displaying chest wall displacements during inspirations. FRC, functional residual capacity; TLC, total lung capacity; au, arbitrary units. Right: display of target mouth pressure (Pm) (−10, −20, −40, or −60 cmH2O).
After positioning of the catheter and the Respirace bands, subjects were asked to perform a series of three inspiratory capacity maneuvers to obtain a maximum voluntary root mean square (RMS) value (C. Sinderby, J. Beck, J. Saphija, J. Weinberg, and A. Grassino, unpublished observations). The highest RMS value obtained was considered to be the subject’s voluntary maximum. Isovolume maneuvers were performed at FRC for subsequent calibration of the RC and Ab signals.

Subjects were asked to perform inspirations from FRC to TLC at target Pms, such that the Pm profile remained constant throughout the entire inspiration. To change the velocity of diaphragm shortening, subjects were asked to repeat the same inspiration from FRC to TLC while reproducing the same chest wall displacements and while generating the same target Pm, but with a different inspiratory resistance applied to the inspiratory side of the two-way valve. Therefore, all parameters are kept constant during the inspirations from FRC to TLC, but the time base of the inspirations is altered. Target Pm of −10, −20, −40, and −60 cmH2O were tested with two different resistances of 10 and 100 cmH2O · l−1 · s−1. The range of inspiratory flows evaluated was from 0.09 to 2.49 l/s. For each resistance and each target Pm, subjects repeated the maneuvers 4–12 times, depending on the duration of the contractions. In this protocol we assumed that the elastic properties of the respiratory system were constant for the different inspirations, but what we were altering was the resistive component (by applying a resistance).

Estimate of crural diaphragm velocity of shortening and diaphragm activation. In the present study we altered the inspiratory flow rate (by altering the inspiratory resistance) to change the duration of the inspirations from FRC to TLC by ~10-fold. Only data in which Pm, RC and Ab displacements, and Pdi were reproducible were included in the analysis. (In other words, Pdi, RC, and Ab should be similar for the same relative portion of the breath.) This means that Ab displacement, a parameter proposed to reflect crural diaphragm shortening (6), occurred at a higher rate when the rate of volume change (i.e., flow) was higher. For clarity, therefore, we refer to the “faster” contractions (higher velocities of shortening) as those occurring with higher flows and the “slower” contractions (lower velocities of contraction) as those occurring with lower flows. We are aware that, depending on the thoracoabdominal pathway chosen, the velocity of crural diaphragm shortening may vary even when inspiratory flow rate is kept constant. However, because identical thoracoabdominal pathways were reproduced at two different inspiratory flow rates, we assumed that the mean velocities of diaphragm shortening were different at two inspiratory flow rates.

In the present study, the RMS of the diaphragm EMG was used as an index of crural diaphragm activation. This is based on the fact that the interference-pattern EMG constitutes a spatial and temporal summation of action potentials from the recruited motor units and their firing rate. The relationship between diaphragm activation and the RMS of the diaphragm EMG has been previously described (3).

Analysis. Diaphragm EMG signal and respiratory mechanics analyses were performed off-line. Diaphragm EMG signals were automatically processed with computer algorithms that eliminate the influence of electrocardiogram, motion artifacts, background noise, and disturbances from power lines and continuously calculate the RMS value for EMG segments of 50 ms.

For each 50-ms diaphragm EMG sample, the position of the diaphragm with respect to the multiple-array esophageal
electrode was determined. During voluntary contractions, the crural diaphragm can be considered as an electrically active region (referred to as EARd), and the center of this activity, from which the majority of the signals originate, is referred to as the EARd center (2). The relative position of the EARd center with respect to a bipolar, perpendicularly oriented electrode filters the EMG signal, and hence control of the EARd center position with respect to the electrode pair from which signals are obtained is important for a correct physiological interpretation of the diaphragm EMG. With a perpendicularly electrode arrangement, signals that are obtained on opposite sides of the EARd center, or on the same side of the EARd center, correlate with extreme values (i.e., the value is expected to be close to −1 or +1) at a 0-μs time shift. Cross-correlation analysis was performed among signals obtained from electrode pairs 1 vs. 3, 2 vs. 4, 3 vs. 5, and so on. The most negative correlation coefficient between any two pairs of electrodes indicates that the respective signals are the most reversed in polarity. The electrode pair that is located between these two most negatively correlated pairs is the electrode pair closest to the center of the EARd. After the EARd center position was determined, the signals obtained from the two electrode pairs that were located next to the EARd center, i.e., 10 mm caudal and 10 mm cephalad, were subtracted from each other. This algorithm yields a new signal, the double-subtracted signal, which is less influenced by electrode filtering and enhanced in signal-to-noise ratio (12). The double-subtraction technique was applied for every 50-ms EMG segment selected. From the double-subtracted signal, RMS was calculated as

\[
\text{RMS} = \left( \frac{\sum_{i=1}^{i_{\text{max}}} s_i^2}{i_{\text{max}}} \right)^{1/2}
\]

where \(i\) is the index over which the signal (s) is summed, \(i = 1\) is the index for the first signal data point used in the summation, and \(i_{\text{max}}\) is the index associated with the last signal data point used in the summation.

In each subject, the following parameters were compared for inspirations with the same target Pm but with different inspiratory flows: Pm, flow, RC, Ab, Pdi, and RMS. To achieve this comparison for the inspirations at different time bases, reproducible breaths (within 20% of target Pm) were averaged and expressed as the percentage of inspiratory duration. The inspirations were divided into 5% intervals.

RESULTS

Four of the five subjects were able to reproduce their inspirations (in terms of pressure profiles and chest wall displacements) at flow rates between 0.1 and 1.4 l/s. Above 1.4 l/s, pressures and chest wall displacement profiles could not be reproduced.

An example of how the protocol was performed is presented in Fig. 2 for subject 1. In Fig. 2, bottom right, RC (y-axis) and Ab displacements (x-axis) are shown for two inspirations from FRC to TLC that were performed at two different inspiratory flow rates. In this example, −10 cmH2O was the target Pm (see Fig. 2, top left) for the two different resistances, resistance A (100 cmH2O · l−1 · s−1; thin line) and resistance B (10 cmH2O · l−1 · s−1; thick line). The resulting inspiratory flows are presented in Fig. 2 (top right; note that inspiration is represented by a negative flow). An
example of the Pdi profile generated with the two resistances is demonstrated in Fig. 2, bottom left. Note that this protocol was designed for subjects to generate similar pressures and similar thoracoabdominal pathways during an inspiration from FRC to TLC, but with different time bases. In other words, we aimed to keep all parameters constant, except for the flow rate.

Figure 3A demonstrates, in subject 4, profiles of Pdi and RMS during the inspirations from FRC to TLC at two different inspiratory flow rates (open symbols, higher flow; solid symbols, lower flow) with the same target Pm (−10 cmH2O) and the same chest wall displacements. Pdi and RMS are plotted on the y-axes of Fig. 3A and B, for both the slow (closed symbols) and fast (open symbols) contractions. In Fig. 3A, time is plotted on the x-axis (s), and, in Fig. 3B, the inspiratory time has been normalized to duration (0–100% of inspiratory duration). As demonstrated in Fig. 3B, the profile for Pdi generation for the two inspirations was nearly identical, where the breaths are expressed as percentage of inspiratory duration. The RMS profile was also the same for the two inspirations, even though they were performed at different velocities of shortening. The lack of an effect of velocity of shortening on the RMS for a given pressure generation is shown in Fig. 3C, which plots the relationship between RMS (y-axis) and Pdi (x-axis) for the breaths normalized to inspiratory duration. These findings were observed in the four subjects who were able to reproduce their chest wall displacements.

All parameters (Pm, flow, RC, Ab, Pdi, and RMS) measured during the inspirations from FRC to TLC at the different flow rates are presented for the group in Figs. 4 and 5 for target Pms of −10 and −20 cmH2O, respectively. The mean (±SD) data for the four subjects is presented in the form of identity plots, with the y-axis representing the parameter for the higher flow contractions and the x-axis representing the parameter for the lower flow contraction. In Figs. 4 and 5, the points are plotted at 5% intervals of total inspiratory time (for clarity, we have omitted the first and last 5%). Differences in inspiratory flow rates (slower and faster contractions) are shown for target Pms of −10 (Fig. 4, top right) and −20 cmH2O (Fig. 5, top right). As expected from the design of the protocol, target Pms were identical for the lower and higher flow inspirations (top left in Figs. 4 and 5, respectively) and had little deviation throughout the inspiration and little deviation between subjects. As well, both RC and Ab displacements (Figs. 4 and 5, middle left and right, respectively) were similar for the faster and slower contractions (for flows below 1.4 l/s). The Pdi generated for both target Pm also fell on the line of identity (Figs. 4 and 5, bottom left). Above 1.4 l/s, subjects were not able to reproduce their chest wall displacements or Pdi in a fashion similar to that for the lower flow inspira-
tions. Figures 4 and 5 (bottom right) demonstrate that there was no evidence for increased RMS values for the inspirations from FRC to TLC at the different inspiratory flows (up to 1.4 l/s).

DISCUSSION

The results of the present study do not provide any evidence for an increase in diaphragm activation when healthy subjects breathe at increasing inspiratory flow rates (up to 1.4 l/s), when similar Pdi and chest wall configuration profiles are reproduced. In other words, the activation of the diaphragm required to produce a given Pdi was not increased for higher velocities of shortening. These data suggest that the diaphragm performs on the low-velocity region of the force-velocity curve at these flow rates. Above 1.4 l/s, chest wall configuration profiles could not be reproduced, indicating that alternate respiratory muscle recruitment patterns needed to be developed, perhaps so that the diaphragm should not be forced to operate on a less favorable portion of the force-velocity relationship.

Critique of methods. We are confident about the validity of our results for the following reasons. First, we have recently developed diaphragm EMG acquisition and analysis methods to reliably obtain a signal with maintained muscle-to-electrode distance (12). Muscle-to-electrode distance filtering has a very strong influence on the strength of the diaphragm EMG signal (2, 4). In the present study, the location of the diaphragm was determined for every instant of the inspiration, and signals were obtained such that all influences of muscle-to-electrode filtering and bipolar electrode filtering were removed (12). Second, the present study was designed such that subjects should reproduce the profiles of Pm, Pdi, and chest wall configuration for the different inspirations, while only the time base of the inspiration was altered by varying inspiratory flow, achieved by altering the resistance. This type of protocol ensures that if there were an increase in diaphragm activation with increasing flow, it was not because of increased Pdi or changes in diaphragm length. In addition, this protocol allowed direct comparisons of activation with increasing flow within a given subject. Third, no particular chest wall configuration profile was imposed on the subjects during the inspirations from FRC to TLC. Subjects were free to choose any chest wall configuration displacements during the inspirations, as long as the profile of RC and Ab displacements could be reproduced at the increasing flow rates. All subjects spontaneously chose a breathing pattern that was associated with a predominant outward Ab displacement. Fourth, in a previous investigation, we have also shown that there is no artifactual influence of lung volume on the diaphragm EMG signal strength for voluntary activation of the diaphragm (3).

We are aware that because we obtained the diaphragm EMG with an esophageal electrode, the RMS represents activation from a sample of the crural diaphragm and that interpretation of our data is limited to this region. Because we found no increase in crural diaphragm EMG signal strength with increasing flow rates, it is possible that the velocity of crural diaphragm shortening did not increase with increasing flows, and one cannot exclude the possibility that the velocity of shortening of the costal portion of the
diaphragm may have increased during the inspirations at different flows. Evidence for costal and crural diaphragm shortening during dynamic breathing has as yet only been obtained in animal experiments, in which it was shown that the mean velocity of shortening of the costal and crural diaphragm is linearly related to mean inspiratory flow (5). In our study, because there was a predominant Ab displacement and activation (up to 70% of voluntary maximum) in all subjects, we can assume that the crural diaphragm contribution to inspiratory flow was significant. We have previously shown in healthy subjects that crural and global diaphragm activation are related during static isometric contractions of the diaphragm (3). As well, costal and crural diaphragm activation were strongly related in chronic obstructive pulmonary disease patients during dynamic maneuvers, as determined by the EMG (C. Sinderby, J. Beck, J. Spahija, J. Weinberg, and A. Grassino, unpublished observations).

Force-velocity characteristics of the diaphragm. For a given level of activation, the force-velocity relationship predicts that the diaphragm should generate less pressure as its velocity of shortening increases; however, according to Younes (13), "the high impedance of the respiratory system places severe constraints on the velocity at which respiratory muscles can shorten in situ." It was predicted that, at resting levels of ventilation where flows are ~1 l/s, the velocity of shortening was estimated to be <2% of the maximum velocity of shortening and that even at maximal inspiratory flow (~10 l/s) the velocity of shortening is <20% of maximum. This is somewhat in agreement with the findings of Leblanc et al. (10), who found that the capacity of the respiratory muscles is reduced by only 5% for every 1-l/s increase in inspiratory flow, and also suggests that the flows in the present study of ~1.4 l/s are representative of the low-velocity portion of the force-velocity curve. Therefore, on the basis of the theoretical predictions of Younes (13) and the experimental findings of Leblanc et al. (10) and ourselves, flows generated during resting breathing and slightly forced inspirations should have relatively little influence on the
maximum force-generating capacity of the diaphragm. Hence, for patients with severe airflow obstruction, the force-velocity characteristics of the diaphragm should play a minor role in diaphragm function.

Pengelly et al. (11) demonstrated in humans that the diaphragm became weaker as a pressure generator with increasing (peak) inspiratory flows, when the contractions were elicited by unilateral submaximal electrical stimulation of the phrenic nerve. Extrapolation of their data revealed an ~7.5-cmH\textsubscript{2}O loss of Pdi (~5% of the maximum force-generating capacity of a healthy diaphragm) for every 1-l/s increase in (peak) flow, which is of essentially the same magnitude as that reported by Leblanc et al. (10). At increasing lung volumes, the flow-related pressure loss was even less. Although the study by Pengelly et al. (11) indicates some flow-related changes in pressure-generating capacity, the low magnitude of these changes is further evidence that the diaphragm is operating on the low-velocity portion of the force-velocity curve. An extensive discussion and critique of the Pengelly study is provided by Younes and Riddle (14).

With respect to voluntary contractions, Goldman et al. (6) demonstrated large increases in diaphragm activation with increasing flow, for a given Pdi and chest wall configuration. We believe that the differences between our results and those obtained by Goldman et al. are due to methodological differences. First and most important, the methods they used to acquire and analyze their diaphragm EMG signals could not ensure a constant position of the esophageal electrode (which consisted of only 1 pair of electrodes) with respect to the diaphragm. Although they anchored their electrode by a balloon inflated in the stomach, this would not necessarily guarantee that the diaphragm position with respect to the bipolar electrode was constant, especially during dynamic maneuvers. This is because, with an anchoring balloon, the electrode is anchored to the esophagus and not to the diaphragm (which is secured to the esophagus by a ligament) (4).
Second, these investigators imposed atypical inspiratory maneuvers (pure vertical and horizontal displacements on the Konno-Mead diagram). From our experience, there may be a dissociation between crural diaphragm activation and global diaphragm activation at extreme chest wall configurations (3). In addition, the analysis by Goldman et al. (6) was also only limited to one particular chest wall configuration, whereas the present study includes data about thoracoabdominal configuration for the entire inspiration.

From the point of view of effort, it is therefore not surprising that patients in ventilatory failure frequently adopt a breathing pattern consisting of increases in inspiratory flow with reduced (or similar) tidal volumes, i.e., rapid, shallow breathing (8). With respect to the findings of the present study, an increase in inspiratory flow from 0.5 to 1.0 l/s requires no increased diaphragm activation for the same Pdi, whereas, after an increase in tidal volume of ~15% of the inspiratory capacity, there is a 10% increase in diaphragm activation required for a given target Pdi (3). Whether our findings can be applied to higher levels of ventilation (for example, in healthy subjects during exercise) remains to be investigated.

In conclusion, the results of the present study could not provide evidence for an increase in diaphragm activation when healthy subjects breathe at increasing flows (up to 1.4 l/s) with similar chest wall configuration and Pdi profiles. At flows above 1.4 l/s, the controversy regarding changes in diaphragm activation with increasing inspiratory flow remains.

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