Effects of lung volume on diaphragm EMG signal strength during voluntary contractions

JENNIFER BECK,1,2 CHRISTER SINDERBY,3,4 LARS LINDSTRÖM,5 AND ALEX GRASSINO1,2

1Department of Physiology, McGill University, Montreal, Quebec H3G 1Y6; 2Pavillon Notre Dame, Centre Hospitalier de l’Université de Montréal, Montreal, Quebec H2L 4M1; 3Guy-Bernier Research Center, Maisonneuve-Rosemont Hospital, University of Montreal, Montreal, Quebec, Canada H1T 2M4; 4Institute for Clinical Neuroscience, University of Gothenburg, and 5Department of Medical Informatics, Sahlgrens University Hospital, Gothenburg S-41345, Sweden

Beck, J. Jennifer, Christer Sinderby, Lars Lindström, and Alex Grassino. Effects of lung volume on diaphragm EMG signal strength during voluntary contractions. J. Appl. Physiol. 85(3): 1123–1134, 1998.—The use of esophageal recordings of the diaphragm electromyogram (EMG) signal strength to evaluate diaphragm activation during voluntary contractions in humans has recently been criticized because of a possible artifact created by changes in lung volume. Therefore, the first aim of this study was to evaluate whether there is an artifactual influence of lung volume on the strength of the diaphragm EMG during voluntary contractions. The second aim was to measure the required changes in activation for changes in lung volume at a given tension, i.e., the volume-activation relationship of the diaphragm. Healthy subjects (n = 6) performed contractions of the diaphragm at different transdiaphragmatic pressure (Pdi) targets (range 20–160 cmH2O) while maintaining chest wall configuration constant at different lung volumes. The diaphragm EMG was recorded with a multiple-array esophageal electrode, with control of signal contamination and electrode positioning. The effects of lung volume on the EMG were studied by comparing the crural diaphragm EMG root mean square (RMS), an index of crural diaphragm activation, with an index of global diaphragm activation obtained by normalizing Pdi to the maximum Pdi at the given muscle length (Pdi/Pdi max@). The volume-activation relationship of the diaphragm was equally affected by changes in lung volume as the volume-Pdi relationship (60% change from functional residual capacity to total lung capacity). We observed a direct relationship between RMS and Pdi/Pdi max@ independent of diaphragm length. The volume-activation relationship of the diaphragm length was equally affected by changes in lung volume as the volume-Pdi relationship (60% change from functional residual capacity to total lung capacity). We conclude that the volume-activation relationship of the diaphragm is an artifactual influence of lung volume on the diaphragm EMG signal strength, the second aim of this study was to evaluate whether there is an artifactual influence of lung volume on the strength of the diaphragm EMG during voluntary contractions. In the event that there is no artifactual influence of lung volume on the diaphragm EMG signal strength, the second aim of this study was to evaluate the required changes in voluntary activation for changes in lung volume at a given transdiaphragmatic pressure (Pdi), i.e., the volume-activation relationship of the diaphragm.

METHODS

Approach for evaluating “artifactual” influence of lung volume on diaphragm EMG signal strength. In the present study we later use the root mean square (RMS) to quantify the diaphragm EMG.

The rationale for inferring diaphragm activation from the strength of the diaphragm EMG signal is that the signal constitutes a spatial and temporal summation of action potentials from the recruited diaphragm motor units and their firing rate (see APPENDIX A). However, it has been shown that the amplitude of the electrically evoked diaphragm compound muscle action potential (CMAP) obtained with an esophageal electrode during constant neural drive (supramaximal stimulation) is altered with changes in lung volume (11, 23). We previously showed that changes in lung volume influence the power spectrum center frequency (CF) of the diaphragm CMAPs, but not of the voluntary EMG signal (2). This suggests that one should not infer changes in the voluntary EMG signal strength with lung volume on the basis of the behavior of the CMAP signal. To our knowledge, the influence of lung volume on the diaphragm EMG signal strength obtained during voluntary contractions has not been studied.

The aim of this study was to evaluate whether there is an artifactual influence of lung volume on the strength (RMS) of the diaphragm interference pattern EMG during voluntary contractions. In the event that there is no artifactual influence of lung volume on the diaphragm EMG signal strength, the second aim of this study was to evaluate the required changes in voluntary activation for changes in lung volume at a given transdiaphragmatic pressure (Pdi), i.e., the volume-activation relationship of the diaphragm.
The crural diaphragm EMG measured with an esophageal electrode represents the activation of a sample of the crural diaphragm, whereas $P_{di}/P_{di\text{max}}$ represents global diaphragm activation. Consequently, the presence of a relationship between RMS and $P_{di}/P_{di\text{max}}$ would also suggest homogeneous activation of the costal and crural diaphragm. Where appropriate, the distinction between crural diaphragm activation (RMS) and global diaphragm activation ($P_{di}/P_{di\text{max}}$) will be made. (According to APPENDIX B, $P_{di}/P_{di\text{max}}$ is actually an index of relative global diaphragm activation, but for didactic reasons we simply refer to global diaphragm activation.)

Rationale for volume-activation relationship of the diaphragm. The classic concept of the length-tension relationship of skeletal muscle is based on measurements of tension developed at various lengths during constant activation of the muscle. The analogy of the length-tension relationship for the human diaphragm is the $P_{di}$ developed at different lengths (lung volume/chest wall configuration) for maximal evoked or voluntary activation of the diaphragm, i.e., the volume-$P_{di}$ relationship. In the present study, we wanted to measure the changes in diaphragm activation at different lengths, during constant $P_{di}$, i.e., the volume-activation relationship.

For a given increase in lung volume, we expect a relative increase in activation (to achieve a given $P_{di}$), which should be of a magnitude similar to the relative decrease in $P_{di}$ (for a constant activation). With accurate measurements of activation, it should be possible to obtain both of these relationships at submaximal activation/tension levels. In the present study, the volume-$P_{di}$ relationship was obtained by asking subjects to perform combined Müller-expulsive $P_{di\text{max}}$ maneuvers at different lung volumes. The volume-activation curve was obtained by asking subjects to perform submaximal contractions of the diaphragm while maintaining a constant target $P_{di}$ at different lung volumes.

As reported in RESULTS, it was not possible to reproduce the exact target $P_{di}$ values, and there was a small variability in the actually achieved $P_{di}$ ($P_{di\text{act}}$) for the given target $P_{di}$ levels with changes in lung volume. To avoid the influence of this variability in $P_{di\text{act}}$ on the RMS, we expressed $P_{di\text{act}}$ in relation to the RMS ($P_{di\text{act}}/RMS$) and used this ratio as the “activation” component in the volume-activation relationship. Because the variability in $P_{di\text{act}}$ was small, this ratio would be minimally influenced by the nonlinear relationship between RMS and $P_{di}$. RMS is the denominator in the expression $P_{di\text{act}}/RMS$, which expresses the reciprocal of the changes in diaphragm activation.

Subjects. Six healthy men, all familiar with respiratory maneuvers, agreed to participate in the study. Their age, height, and weight are presented in Table 1. The experimental setup is shown in Fig. 1.

Signal acquisition. Diaphragm EMG signals were obtained via a multiple-array esophageal electrode consisting of eight stainless steel rings (2 mm wide, 2 mm diameter), placed 10 mm apart, creating an array of seven sequential differential
bipolar electrode pairs, mounted on silicone tubing (2 mm diameter). A schematic representation of the electrode is presented in Fig. 1, left. The most caudal pair of rings was referred to as “electrode pair 1” and the most cephalad pair of rings as “electrode pair 7.” A two-lead differential electrocardiogram (EGC) was obtained from electrodes (model FC24, Graphic Controls) placed on the sternum, vertically and 10 cm apart, for later diaphragm EMG sample selection.

A Teflon tube was placed inside the silicone tubing (0.75 mm diameter), and a 5-cm-long, 1.5-cm-diameter latex balloon was mounted ~5 cm below the most distal EMG ring to allow for measurements of gastric pressure (Pga). Esophageal pressure (Pes) was measured via a separate catheter (1 mm diameter). The two balloon catheters were connected to a differential pressure transducer (Validyne diameter). The two balloon catheters were connected to a differential pressure transducer (Validyne diameter) to yield Pdi, which was displayed to the subject on a storage oscilloscope (model 1604, Gould; Fig. 1, middle). The Pga catheter was also connected to a separate differential pressure transducer and referenced to atmosphere. Pdi and Pga were recorded on an eight-channel strip chart recorder (model 35-V7808-12, Gould) and on magnetic tape (model 4000A, Vetter). The signals were later acquired (model DT 2801A, Data Translation) at a sampling frequency of 100 Hz (12-bit resolution).

Diaphragm EMG signals from electrode pairs 1–7 and the ECG were amplified (model INA102, Burr-Brown) and high-pass filtered at 10 Hz with an antialiasing filter at 1,000 Hz (model D708BL 8-pole Bessel filter, Frequency Devices). Diaphragm EMG signals were acquired and digitized by an analog-to-digital converter (model 2821, Data Translation), with 12-bit resolution, at a sampling frequency of 2,000 Hz, and stored on hard disk for off-line analysis. EMG signals from all seven electrode pairs were displayed to the investigator on a computer monitor.

Lung volume was assessed throughout the experiment by the method of Konno and Mead (16) (Fig. 1, middle). Two respiratory inductive plethysmography bands (Respitrace, Ambulatory Monitoring) were used to evaluate rib cage (RC) and abdominal (AB) displacement. The RC band was placed around the upper portion of the thorax, vertically centered over the nipples; the upper edge of the AB band was placed around the abdomen at the level of the umbilicus. The RC signals were amplified and displayed on the vertical axis, and the AB signals on the horizontal axis of a storage oscilloscope (model 5103N, Tektronix; Fig. 1, middle). The RC and AB signals were recorded on an eight-channel strip chart recorder (model 35-V7808-12, Gould).

Experimental protocol. Subjects were studied while seated in an upright chair, facing the two storage oscilloscopes: one for target Pdi and the other for target Pdi. Respitrace bands were positioned on the subjects and secured in place by a surgical bandage placed around the abdomen at the level of the umbilicus. The RC signals were amplified and displayed on the vertical axis, and the AB signals on the horizontal axis of a storage oscilloscope (model 5103N, Tektronix; Fig. 1, middle). The RC and AB signals were recorded on an eight-channel strip chart recorder (model 35-V7808-12, Gould).

On the day of the experiment, the esophageal electrode was passed through the nose, swallowed, and positioned at the level of the gastroesophageal junction by feedback from an on-line display of the diaphragm EMG signals from all seven electrode pairs on the computer monitor. Once the diaphragm was located at the center of the electrode array, the electrode was fixed externally at the nose. After the catheters and the Respitrace bands were positioned, subjects were asked to perform a relaxation maneuver from total lung capacity (TLC) to functional residual capacity (FRC) and a series of isovolume maneuvers at FRC, 30% of inspiratory capacity (one-third of IC), 60% of inspiratory capacity (two-thirds of IC), and TLC (Fig. 1, middle). To ensure that posture and the position of the Respitrace bands remained constant, these maneuvers were repeated throughout the experiment.

In the present study, four lung volumes were evaluated along the relaxation curve: FRC, one-third of IC, two-thirds of IC, and TLC (Fig. 1, middle). We are aware that, at a given lung volume, different chest wall configurations and, hence, different diaphragm lengths can be obtained, but in this study we restricted the configurations to the relaxation curve of the Konno-Mead diagram, and hence the changes in chest wall configuration are referred to as changes in lung volume. With this setup, we could assume that diaphragm shortening occurs from FRC to TLC (12).

Maximal voluntary Pdi maneuvers were performed at four different diaphragm lengths, FRC, one-third of IC, two-thirds of IC, and TLC (combined Müller-expulsive maneuvers), randomly throughout the experiment, with rest periods between each attempt. The highest of three attempts was considered to be maximal. The Pdi_{max} voluntary performed at any given length was referred to as Pdi_{max}.

Subjects were asked to reach one of the four predetermined points on the Konno-Mead diagram (marked on the oscilloscope), and, while keeping the beam of the scope at the target point (i.e., maintaining the same chest wall configuration), subjects performed a voluntary, static contraction of the diaphragm while actively maintaining the target Pdi (range 20–160 cmH2O). All target Pdi swings were referenced to the resting Pdi at FRC. Each contraction lasted 5–10 s and was repeated five to eight times for each lung volume. A rest period was allowed between contractions (1–5 min). We did not instruct the subjects on how to generate the target Pdi levels; i.e., we did not control the relative contribution of Pga and Pes to Pdi. Although the target Pdi was fixed, the actual Pdi generated may not necessarily have been the target Pdi. For example, the target Pdi may have been 20 cmH2O for the first lung volume, but the actual Pdi generated by the subject may have been 18–22 cmH2O. Throughout this study, the Pdi that was to be maintained by the subjects during the contractions is referred to as the target Pdi. The Pdi achieved by the subject is referred to as Pdi_{act}.

Signal analysis. Diaphragm EMG signals were automatically processed with computer algorithms that eliminate the ECG, control for signal contamination (30), and neutralize signal filtering due to changes in bipolar electrode positioning with respect to the diaphragm by implementation of the double-subtraction technique (29).

Briefly, EMG segments are selected from all seven electrode pairs between successive QRS complexes of the ECGs (R-R interval 50–75%) (30). With an array of bipolar electrodes (interelectrode distance 10 mm), the electrode pair closest to the center of the electrically active region of the diaphragm (EAR_{d,center}) can be determined by cross correlating the signals from every second pair of electrodes (e.g., pair 1 vs. pair 3, pair 2 vs. pair 4) (3). EAR_{d,center} lies between the two most negatively correlated electrode pairs. Once the EAR_{d,center} is determined, the signal from the electrode pair 10 mm caudal to EAR_{d,center} is subtracted from the signal from the electrode pair 10 mm cephalad to EAR_{d,center}. This algorithm yields a new signal, the double-subtracted signal, that is minimized in bipolar electrode filtering and enhanced in signal-to-noise (SN) ratio (29).
LUNG VOLUME AND DIAPHRAGM EMG

![Graph showing RMS of Pdiact with lung volume](image)

**Table 2. Variability in Pdiact for a given target Pdi: range and CV for all lung volumes**

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>20 cmH2O Range</th>
<th>CV</th>
<th>40 cmH2O Range</th>
<th>CV</th>
<th>60 cmH2O Range</th>
<th>CV</th>
<th>80 cmH2O Range</th>
<th>CV</th>
<th>100 cmH2O Range</th>
<th>CV</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>4.26</td>
<td>10.9</td>
<td>6.84</td>
<td>6.8</td>
<td>2.76</td>
<td>2.1</td>
<td>0.13</td>
<td>0.1</td>
<td>0.55</td>
<td>0.5</td>
</tr>
<tr>
<td>2</td>
<td>0.78</td>
<td>2.0</td>
<td>0.69</td>
<td>1.1</td>
<td>0.78</td>
<td>0.8</td>
<td>0.78</td>
<td>0.5</td>
<td>1.26</td>
<td>0.7</td>
</tr>
<tr>
<td>3</td>
<td>2.24</td>
<td>5.3</td>
<td>2.43</td>
<td>3.2</td>
<td>2.62</td>
<td>2.2</td>
<td>2.64</td>
<td>1.5</td>
<td>0.64</td>
<td>0.5</td>
</tr>
<tr>
<td>4</td>
<td>2.44</td>
<td>6.2</td>
<td>3.44</td>
<td>4.3</td>
<td>1.51</td>
<td>1.3</td>
<td>0.78</td>
<td>0.5</td>
<td>1.26</td>
<td>0.7</td>
</tr>
<tr>
<td>5</td>
<td>1.36</td>
<td>2.6</td>
<td>7.87</td>
<td>7.9</td>
<td>4.73</td>
<td>4.4</td>
<td>3.7</td>
<td>3.2</td>
<td>1.17</td>
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</tr>
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<td>6</td>
<td>3.41</td>
<td>12.2</td>
<td>4.76</td>
<td>6.4</td>
<td>1.65</td>
<td>1.5</td>
<td>1.78</td>
<td>1.3</td>
<td>0.63</td>
<td>0.5</td>
</tr>
</tbody>
</table>

Mean ± SD 2.41 ± 1.28 6.53 ± 4.22 4.34 ± 2.71 4.95 ± 2.55 2.34 ± 1.38 2.05 ± 1.26 1.81 ± 1.43 1.32 ± 1.20 0.85 ± 0.34 0.6 ± 0.14

Pdi, transdiaphragmatic pressure; Pdiact, Pdi actually achieved; CV, coefficient of variation. Values are expressed in cmH2O for range and in % for CV.

RESULTS

Outcome of experimental protocol. After a practice session, all subjects were able to coordinate the respiratory muscles to achieve the target Pdi levels while maintaining chest wall configuration constant. The outcome of the experimental protocol is demonstrated for a representative subject in Fig. 2 and shows the response of the RMS with lung volume for the various target Pdi values. Also, for a given lung volume, RMS values increased to achieve higher target Pdi values. In all six subjects the Pdiact (for a given target Pdi) varied little (Table 2) at the four lung volumes (range 2.4 ± 1.3 cmH2O). The mean coefficient of variation for the Pdiact for all subjects was 3.1 ± 2.5%.

Influence of lung volume on Pdiact. In all subjects, values of Pdiact decreased with increasing lung volume (Table 3). The maneuvers were performed while subjects maintained chest wall configuration constant, as observed on the Konno-Mead diagram display. At TLC, Pdiact values dropped on average by 60% from FRC. The relationship between Pdiact, and lung volume for the group of six subjects was fairly linear.

Lung volume does not influence the relationship between RMS and Pdi/Pdiact. When the two indexes of activation were compared, RMS was directly related to RMS value was calculated. Also, means ± SD were calculated for the Pdiact swings.

**Table 2. Variability in Pdiact for a given target Pdi: range and CV for all lung volumes**

<table>
<thead>
<tr>
<th>Target Pdi</th>
<th>Subject No.</th>
<th>Range</th>
<th>CV</th>
<th>Range</th>
<th>CV</th>
<th>Range</th>
<th>CV</th>
<th>Range</th>
<th>CV</th>
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<tr>
<td>20 cmH2O</td>
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<td>4.26</td>
<td>10.9</td>
<td>6.84</td>
<td>6.8</td>
<td>2.76</td>
<td>2.1</td>
<td>0.13</td>
<td>0.1</td>
</tr>
<tr>
<td>40 cmH2O</td>
<td>2</td>
<td>0.78</td>
<td>2.0</td>
<td>0.69</td>
<td>1.1</td>
<td>0.78</td>
<td>0.8</td>
<td>0.78</td>
<td>0.5</td>
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<tr>
<td>60 cmH2O</td>
<td>3</td>
<td>2.24</td>
<td>5.3</td>
<td>2.43</td>
<td>3.2</td>
<td>2.62</td>
<td>2.2</td>
<td>2.64</td>
<td>1.5</td>
</tr>
<tr>
<td>80 cmH2O</td>
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<td>6.2</td>
<td>3.44</td>
<td>4.3</td>
<td>1.51</td>
<td>1.3</td>
<td>0.78</td>
<td>0.5</td>
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<tr>
<td>100 cmH2O</td>
<td>5</td>
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<td>2.6</td>
<td>7.87</td>
<td>7.9</td>
<td>4.73</td>
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<td>3.7</td>
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<td>1.65</td>
<td>1.5</td>
<td>1.78</td>
<td>1.3</td>
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</table>

Mean ± SD 2.41 ± 1.28 6.53 ± 4.22 4.34 ± 2.71 4.95 ± 2.55 2.34 ± 1.38 2.05 ± 1.26 1.81 ± 1.43 1.32 ± 1.20 0.85 ± 0.34 0.6 ± 0.14

Pdi, transdiaphragmatic pressure; Pdiact, Pdi actually achieved; CV, coefficient of variation. Values are expressed in cmH2O for range and in % for CV.
to Pdi/Pdi\textsubscript{max}@L, as depicted for the six individual subjects in Fig. 3. These results also demonstrate that there is no artifactual influence of lung volume on the diaphragm EMG during voluntary contractions. In some subjects, outlier points were observed (e.g., subjects 2, 3, and 6) and represent the RMS values obtained at TLC, when the subjects were contracting the diaphragm at the 20-cmH\textsubscript{2}O target Pdi. Disregarding these outlier values, a single best curve fit was drawn through the data points (from the origin) to visually emphasize the relationship between RMS and Pdi/Pdi\textsubscript{max}@L. The decision to plot a curve and not a straight line through the data points was based on the following. 1) Linear regression analysis of the data indicated y-intercepts that were different from zero (Table 4), and the relationship was expected to go through the origin, where there should be no electrical activity when the diaphragm is not activated. 2) Appendix A predicts that the RMS varies with the square root of activation, and hence the RMS is expected to increase very little at increasing activation levels. 3) Comparison of the coefficients of determination (R\textsuperscript{2}) obtained with first- and second-order polynomial equations (Table 4) suggested a slight but nonsignificant (P \textup{=} 0.083) improvement in R\textsuperscript{2} from a linear curve fit to a second-order polynomial; R\textsuperscript{2} increased on average by 3.00 \pm 3.41\% (range 0–9\%). The nonnormalized values of the RMS varied among subjects (note different scales for the y-axes in Fig. 3) and are most likely dependent on the anatomic differences among individuals (e.g., radial muscle-to-electrode distance).

Crural diaphragm activation in response to diaphragm shortening. Figure 4A demonstrates in subject 5 the curvilinearity of the relationship between the Pdi and the RMS at different lung volumes. The best curve fit obtained for each lung volume has been drawn for visual clarity. Figure 4B demonstrates the relationship between Pdi\textsubscript{act}/RMS and lung volume for the various target Pdi values. At a given lung volume Pdi\textsubscript{act}/RMS showed a large variability, as presented for the six subjects in Table 5. (All data points, including the outlier values in Fig. 3, are included in this analysis.) Figure 4C shows the relationship between Pdi\textsubscript{act} and RMS with changes in lung volume when the data were normalized to the FRC value, with correction for differences in the intercepts. The slopes of the reduction in Pdi\textsubscript{act}/RMS with increasing lung volume were similar for the different target Pdi values.

In subject 5 the relative reduction in Pdi\textsubscript{act}/RMS was similar to the relative change in Pdi\textsubscript{max}@L with increasing lung volume (Fig. 4C). Figure 5 shows the group mean relative change in Pdi\textsubscript{act}/RMS obtained for all target Pdi values and Pdi\textsubscript{max}@L at the four different lung volumes, according to the analysis performed in Fig. 4C. The three outlier values from Fig. 3 (indicated by arrows, TLC, 20 cmH\textsubscript{2}O) are included in this analysis, which reduced Pdi\textsubscript{act}/RMS at TLC, and in part explain the slight deviation away from the line of identity (dashed line). For this group of six subjects, we found a proportional relationship between the volume-activation and volume-Pdi relationship. The data suggest that, with an increase in lung volume equal to 33\% of IC, there is a 20\% reduction in Pdi\textsubscript{max}@L (the volume-Pdi relationship) and a 20\% decrease in Pdi\textsubscript{act}/RMS.

**DISCUSSION**

The results of this study demonstrate that 1) there is no artifactual effect of lung volume on the diaphragm EMG signal strength during voluntary contractions, 2) crural diaphragm activation, as measured by the RMS of the diaphragm EMG, is related to global diaphragm activation, inferred from Pdi/Pdi\textsubscript{max}@L, and 3) the volume-activation relationship can be used to infer changes in the length-tension relationship of the diaphragm at submaximal diaphragm activation/contraction levels.

Critique of Pdi/Pdi\textsubscript{max}@L as an index of global diaphragm activation. In the present study, global diaphragm activation was inferred by the relative Pdi, which was calculated by normalizing the Pdi to the maximum Pdi obtained at the given diaphragm length (Pdi/Pdi\textsubscript{max}@L). Reliability of this index of activation was dependent on the target contractions and Pdi\textsubscript{max}@L maneuvers being performed at the same muscle length. By controlling the maintenance of chest wall configuration during the target contractions and the Pdi\textsubscript{max}@L maneuvers, it was assumed that changes in diaphragm length were kept to a minimum during these maneuvers (16). Feedback from the Konno-Mead diagram also ensured that the contractions were always repeated at the same target lung volume without displacement of the chest wall.

Another limitation of using the Pdi\textsubscript{max}@L for normalization is that the maneuver is voluntary and depends on the subject's motivation and experience. In the present study, the Pdi\textsubscript{max}@L values at FRC (from a combined Müller-expulsive maneuver performed with feedback) were well within the range that has been reported in healthy subjects (18). With respect to the higher lung volumes, we observed a relative reduction in Pdi\textsubscript{max}@L similar to that obtained by others, whether elicited voluntarily or by electrical or magnetic phrenic nerve stimulation (6, 15, 17, 32). Previous investigators used the twitch interpolation technique to show that subjects who are inexperienced in performing Pdi\textsubscript{max} maneuvers are able to voluntarily and maximally recruit their diaphragm at FRC (5) and at lung volumes above FRC (22).
Although there are limitations to using Pdi/Pdimax_L as an index of global diaphragm activation, we believed it was the most suitable for comparison to the RMS of the diaphragm EMG. Other proposed measures of activation, such as the single-twitch interpolation technique, provide information about maximal diaphragm motor unit recruitment, and not diaphragm motor unit firing rate, and hence this technique provides only one part of the information related to diaphragm activation. Another method that has been used to evaluate diaphragm activation has been to look at the firing rate of single motor unit action potentials recorded with needle electrodes (8). For evaluation of diaphragm activation in the present study, this technique would not have been suitable, because the motor unit action potentials would not have been discernible at higher

![Graphs showing RMS-Pdi/Pdimax_L relationship](image)

**Fig. 3.** There is no artifactual influence of lung volume on diaphragm EMG RMS. Comparison of RMS (y-axis) and Pdi normalized to Pdimax_L (Pdi/Pdimax_L) (x-axis) for subjects 1–6 (A–F) shows no artifactual influence of lung volume on diaphragm EMG, as well as a direct relationship between crural diaphragm activation and global diaphragm activation, up to moderate activation levels. Arrows, distinct outlier points.

**Table 4.** Statistical parameters describing RMS-Pdi/Pdimax_L relationship

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>Linear Regression Analysis</th>
<th>Polynomial Function</th>
<th>RMS, root mean square. *Subjects in whom analysis was performed with (and without) exclusion of outlier values at TLC target Pdi of 20 cmH₂O.</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Intercept</td>
<td>Slope</td>
<td>r</td>
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<td>0.93</td>
</tr>
<tr>
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<td>3*</td>
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<td>0.68</td>
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<td>6</td>
<td>11.80</td>
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</table>
contraction levels, and again, motor unit firing rate provides only part of the information related to activation. Therefore, for the purpose of the evaluation of the EMG as a measure of activation at different lung volumes, we chose Pdi/Pdimax as the most appropriate index of global diaphragm activation.

Critique of diaphragm EMG RMS as an index of crural diaphragm activation. Besides the possible influence of lung volume on the voluntary EMG signal, which was ruled out in the present study, several other factors can artifactually influence the RMS of the diaphragm EMG power spectrum (for isometric, constant-force contractions). Most importantly, the EMG signal strength is dependent on accurate methodology for acquisition and analysis, as well as electrode configuration and positioning. The RMS is also affected by factors that influence signal quality, such as cardiac activity, esophageal peristalsis, external noise, electrode motion artifacts, and aliasing. These issues have previously been discussed in detail, and methods are now available to overcome the artifactual influences (3, 4, 29, 30).

Another factor that can affect the RMS value is a reduction in the conduction velocity (CV) of the muscle fiber action potentials, which could occur during sustained forceful contractions or with reductions in temperature. The spectral moment of order zero, used in calculations of the RMS, is inversely proportional to CV (20), and hence reductions in CV will result in increases in the RMS values that are unrelated to changes in muscle activation. In the present study, a fatigue correction factor involving the CF was calculated for each lung volume and each target contraction level (we assumed that changes in CF paralleled changes in CV). The use of CF in the correction of the RMS (for changes in CV) requires that CF not itself be artifactually influenced by lung volume, as was demonstrated by Beck et al. (2, 4), or other artifacts such as signal quality (30) or electrode positioning (3, 4). A lack of

---

**Table 5. Absolute achieved Pdi act/RMS**

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>FRC</th>
<th>1/3 IC</th>
<th>2/3 IC</th>
<th>TLC</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>3.36–7.06</td>
<td>3.47–4.23</td>
<td>2.23–3.49</td>
<td>2.08–2.79</td>
</tr>
<tr>
<td>2</td>
<td>1.21–2.65</td>
<td>1.54–2.04</td>
<td>0.76–1.18</td>
<td>0.32</td>
</tr>
<tr>
<td>3</td>
<td>2.00–1.62</td>
<td>0.84–1.28</td>
<td>0.68–0.74</td>
<td>0.28–0.55</td>
</tr>
<tr>
<td>4</td>
<td>2.25–7.47</td>
<td>1.71–4.38</td>
<td>1.34–2.74</td>
<td>1.05–1.61</td>
</tr>
<tr>
<td>5</td>
<td>1.04–2.05</td>
<td>0.68–1.65</td>
<td>0.40–0.70</td>
<td>0.35–0.46</td>
</tr>
<tr>
<td>6</td>
<td>1.87–3.29</td>
<td>1.18–2.57</td>
<td>0.89–2.32</td>
<td>0.29–1.36</td>
</tr>
</tbody>
</table>

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**Fig. 4.** Crural diaphragm activation in response to diaphragm shortening. A: in subject 5, relationship between Pdi actually achieved (Pdi act) and RMS at different lung volumes in 1 subject. Pdi act/RMS relationship at different muscle lengths is curvilinear. Best curve fit obtained for each lung volume has been drawn for visual clarity. B: Pdi act/RMS with changes in lung volume for different target Pdi values. C: relative change in Pdi act/RMS for different target Pdi values plotted vs. lung volume.

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**Fig. 5.** Identity plot of relative changes in Pdi act/RMS and Pdimax. Plot describes relationship between Pdimax and Pdi act/RMS at different lung volumes (group mean data). n, No. of subjects.
correction for changes in CV would result in overestimations of the RMS value.

Theoretically, a limitation of the RMS as an index of crural diaphragm activation is that the RMS is predicted to increase less than activation at very high firing rates and/or very high numbers of recruited motor units (see Appendix A). With respect to the experimental data, we found in most subjects a fairly linear relationship between the RMS and the global diaphragm activation up to ~75% of maximum activation levels, indicating that underestimation of the RMS to reflect activation may occur above this level.

One other limitation of the use of the EMG signal strength is that because of anatomic and physiological differences, nonnormalized RMS values cannot be used to compare activation levels among different subjects. Various techniques have been presented to obtain a reference value for normalizing the RMS, such as magnitudes of increase from resting or supine levels (9, 10), percentage of value obtained during an inspiration to TLC (14) or a maximal Pdi maneuver (34), or simply normalization of the RMS to the highest value obtained at any time. None of these investigators, however, provided any rationale or evaluation to justify their reference value. It has been suggested that a simple inspiration to TLC is suitable to obtain a maximum voluntary RMS value for normalization purposes (unpublished observations). One must keep in mind that because of the limited increase in RMS at high activation levels, the maximum RMS may actually be an underestimation of the true maximal activation, providing overestimations in the relative RMS.

By using the crural diaphragm EMG measured with an esophageal electrode, signals are obtained from an area of unknown size located in the region of the crural diaphragm. Hence, the crural diaphragm EMG is only a sample of the entire diaphragmatic motor unit population. The findings of the present study, however, show that similar activation levels obtained with various contractions of the diaphragm performed at four defined lung volumes along the relaxation curve. However, subjects had the freedom to choose the Pga and Pes contributions to Pdi, and our results of a direct relationship between the RMS and Pdi/Pdi,max@ were the same for subject 3, who mainly performed the maneuvers by changing Pga, and for subject 2, who mainly changed Pes. We observed that, at an extreme chest wall configuration such as TLC, three subjects showed outlier values in the RMS-Pdi/Pdi,max@ relationship when the target Pdi was 20 cmH2O, suggesting deviations in the relationship between crural diaphragm activation and global diaphragm activation at this extreme diaphragm length; however, the data points related to TLC when the target Pdi was ≥40 cmH2O behaved as expected.

In conclusion, the use of the crural diaphragm EMG RMS value to infer global diaphragm activation can be considered to be valid at physiological activation levels and at nonextreme chest wall configurations. Our findings are in agreement with previous investigators' reports on homogeneity between costal and crural diaphragm activation during breathing (7, 21, 24, 27, 28). Effects of lung volume on interference pattern EMG signal strength. The close relation between the diaphragm EMG and global diaphragm activation contradicts previous statements that the voluntary diaphragm EMG is systematically affected by changes in lung volume (8, 13). Because of the design of the study, where a range of activation levels was evaluated at different lung volumes, it would not have been possible to obtain a relationship between the Pdi/Pdi,max@ and the EMG RMS, two indexes of activation, if there had been a systematic effect of lung volume on the RMS values. The findings of the present study are also in agreement with a previous study, where we could not demonstrate any lung volume-related changes in the frequency content of the voluntary EMG (2).

Previous allegations (8, 13) about the inaccuracy of using diaphragm EMG to assess diaphragm activation have been based on findings that diaphragm CMAP amplitude is affected by lung volume (11, 23). The diaphragm CMAP represents the summed synchronized electrical activity generated by all motor units after a supramaximal stimulus of the phrenic nerve(s). The voluntary signal represents the summed electrical activity generated by asynchronously firing crural diaphragm motor units. Because of the fundamental differences in signal characteristics of the CMAP and the voluntary signal, in this study and in a previous study, we have clearly demonstrated that the behavior of the synchronized CMAP signal cannot be used to infer the behavior of the voluntary EMG signal. With respect to changes in lung volume, we previously (2) discussed the possible reasons for the differences in behavior of the two signals (in terms of their frequency content). The results of the present study did not provide any additional information describing the differences between the two types of signals, but we have confirmed that the voluntary EMG and CMAP signals behave differently with respect to changes in lung volume. We therefore conclude that the voluntary diaphragm EMG signal, when acquired and analyzed with appropriate methodology, is adequate for evaluation of diaphragm activation, under conditions where diaphragm length changes are expected to occur or in patients with chronic obstructive pulmonary disease who are hyperinflated because of expiratory flow limitation.

Volume-activation and length-tension relationships. We have demonstrated that, given the conditions of the present study and within a physiological range of activation levels, RMS = Pdi/Pdi,max@. Therefore, the volume-activation relationship, which is the activation
required to generate a given Pdi, can be expressed as 1) RMS/Pdi, where RMS is the index used to infer activation and Pdi is the targeted Pdi, or 2) (Pdi/Pdi_{max}@\text{max})/Pdi, where Pdi/Pdi_{max}@\text{max} is the index used to infer activation. (Pdi/Pdi_{max}@\text{max})/Pdi can be simplified to Pdi_{max}@\text{max}. Therefore, RMS/Pdi \sim Pdi_{max}@\text{max}. With changes in lung volume, RMS/Pdi is a representation of the activation needed to generate a given Pdi, and Pdi_{max}@\text{max} is the pressure response for maximum voluntary activation. With increasing lung volume, the RMS increases for a given Pdi, whereas Pdi_{max}@\text{max} is reduced, resulting in a reciprocal relationship between the volume-activation and volume-Pdi relationship. In summary, measurements of activation required to generate a given Pdi or measurements of the Pdi generated for a fixed activation level can provide information about the length-tension relationship of the diaphragm at submaximal or maximal levels of activation/contraction.

Implications of a curvilinear relationship between RMS and Pdi. In the present study (Fig. 4A), as well as in previous studies (14), it has been demonstrated that the relationship between RMS and Pdi is curvilinear. The implication of a nonlinear relationship is that Pdi_{act}/RMS could vary severalfold at a given lung volume (Table 5) by varying the target Pdi. The example in Fig. 4B shows that a change in target Pdi from 20 to 120 cmH_2O produces the same change in Pdi act/ impedance in Fig. 4C, (Pdi/Pdi_{max}@\text{max})/Pdi, where Pdi/Pdi_{max}@\text{max} is the index used to infer activation. (Pdi/Pdi_{max}@\text{max})/Pdi can be simplified to Pdi_{max}@\text{max}. Therefore, RMS/Pdi \sim Pdi_{max}@\text{max}. With changes in lung volume, RMS/Pdi is a representation of the activation needed to generate a given Pdi, and Pdi_{max}@\text{max} is the pressure response for maximum voluntary activation. With increasing lung volume, the RMS increases for a given Pdi, whereas Pdi_{max}@\text{max} is reduced, resulting in a reciprocal relationship between the volume-activation and volume-Pdi relationship. In summary, measurements of activation required to generate a given Pdi or measurements of the Pdi generated for a fixed activation level can provide information about the length-tension relationship of the diaphragm at submaximal or maximal levels of activation/contraction.

APPENDIX A

Theoretical Description of the Relationship Between Activation and EMG Signal Strength

Myoelectric properties. Consider a muscle (or the part of a muscle that contributes to the observed myoelectric signal) containing a number (M) of active motor units each with a repetition rate equal to 1/T_R that, for simplicity, is assumed to have the same value in the mean for all the units. We introduce the concept of the intensity (I) of the muscle’s electrical activity

\[ I = M/T_R \] (A1)

The contributions from one motor unit are assumed to be uncorrelated with the contributions from the other units. The individual motor unit contributions are characterized by the action potential duration (T_0) and a strength measure, which will emerge from the calculations below.

Statistical preliminaries. We introduce the amplitude density function f_5(z), which describes the differential probability of finding a certain value (in our case, the voltage of the signal) z of a random variable z. The density function is related to the distribution function F_5(z), which is the integral over f_5(z)

\[ F_5(z) = \int_{-\infty}^{z} f_5(z) \, dz \] (A2)

If f_5(z) is normalized (the area under the density curve attains unity), the function F_5(z) can also be expressed as the probability (P) of finding the variable z below the level z

\[ F_5(z) = P[z < z] \] (A3)

The density function is useful, since the expected value of the random variable z is

\[ E[z] = \int_{-\infty}^{+\infty} z \cdot f_5(z) \, dz \] (A4)

which is also known as the DC value. Furthermore, the expected value of the square of the variable z is

\[ E[z^2] = \int_{-\infty}^{+\infty} z^2 \cdot f_5(z) \, dz \] (A5)

which also is the square of the RMS value. The variance of the variable z is related to the mentioned quantities as follows

\[ \text{Var}[z] = E[z^2] - E[z]^2 \] (A6)

The above expressions, with statistical basis, explain why the RMS is frequently used to describe signal properties. It is interesting to compare the characteristics of the RMS value with another measure of signal strength, the full-wave-rectified and averaged potential (FRA). The FRA value can be obtained from the amplitude density function as follows

\[ \text{FRA} = \int_{-\infty}^{+\infty} |z| \cdot f_5(z) \, dz \] (A7)

This represents a rectifier characteristic that is linear but with opposite signs for positive and negative values of the input signal. Many signal strength-measuring devices are actually measuring the FRA value but are calibrated to show the RMS value. This can only be true for one particular signal waveform; mostly, it is assumed that the signal has a sinusoidal shape.

Signal summation. To further clarify the properties of the density function, consider an action potential of duration T_D that occurs once in the observation interval of length T_0 (Fig. 6). The value of f_5(z) is proportional to the relative time the signal spends at a certain level, and thus the area under the peak of f_5(z) at zero value of z is proportional to T_0 - T_D. We can thus write the expression for the density function as

\[ f_5(z) = f_5(z) + \delta(z)(T_0 - T_D)/T_0 \] (A8)
where we have split the density function into one part describing the action potential \( p \) as such and one part describing the silent interval outside the potential (voltage level zero, modeled by the Dirac delta function, \( \delta(z) \)).

The signal summation of randomly distributed contributions can be performed as follows. A fundamental theorem concerning the sum of two random variables

\[
z = x + y
\]

(A9)

states that the amplitude density function of the sum equals the convolution of the densities of the contributing signals, which are assumed to be independent (25)

\[
f_z(z) = \int_{-\infty}^{\infty} f_x(x) f_y(y) \, dx = \int_{-\infty}^{\infty} f_x(z - y) f_y(y) \, dy \tag{A10}
\]

The sum of two motor unit action potentials, denoted \( x \) and \( y \), of the same shape and duration (\( T_0 \)) randomly occurring in the observation interval \( T_0 \) is obtained by combining Eqs. A8 and A10. If the observation interval is long in comparison with the duration of the potentials, the likelihood of obtaining overlap between the potentials is low. Also, the main contribution to the convolution of \( f_x(x) \) and \( f_y(y) \) comes from the \( \delta(x) \) and \( \delta(y) \) functions. With these assumptions the combination of Eqs. A8 and A10 can be approximated

\[
f_z(z) = \int_{-\infty}^{\infty} f_x(x) \delta(z - x)(1 - T_0/T_0) \, dx
\]

\[+ \int_{-\infty}^{\infty} f_x(z - y) \delta(x)(1 - T_0/T_0) \, dx \tag{A11}
\]

\[+ \int_{-\infty}^{\infty} \delta(z - x)(1 - T_0/T_0) \, dx \]

which is further simplified to

\[
f_z(z) = 2(1 - T_0/T_0) f_p(z) + (1 - 2T_0/T_0) \delta(z) \tag{A12}
\]

Generalization to \( N \) potentials, still with the assumption that they occupy only a small fraction of the observation interval, results in the approximation

\[
f_z(z) = N(1 - T_0/T_0) f_p(z) + (1 - NT_0/T_0) \delta(z) \tag{A13}
\]

Increasing the number of sources so that they start to overlap, we find that the density function will lose the peak \( [\delta(z)] \) at zero level and begin to diffuse into a more spread-out shape. In the case of a very large number of randomly distributed sources, the shape will become Gaussian (with the interesting property that the convolution between 2 Gaussian functions yields another Gaussian function). The summation in this case can be illustrated by two Gaussian

\[
\begin{align*}
\text{Voltage (a.u.)} & \quad \text{Time} & \quad \text{Density function} \\
0 & \quad T_0 & \quad f_z(z)
\end{align*}
\]

Fig. 6. Relation between signal as a function of time and amplitude density function. Left: action potential in time domain, observed in observation interval \( T_0 \). Right: amplitude density function \( f_z(z) \) of signal voltage \( z \). au, Arbitrary units.

\[
\text{Fig. 7. Signal strength measurements as a function of intensity of muscle activity. Plot of RMS and full rectified average (FRA, y-axis) as a function of intensity (x-axis) in log scale.}
\]

\[
distributions, denoted \( x \) and \( y \), with zero mean and variances \( \sigma_x^2 \) and \( \sigma_y^2 \), respectively. Thus, for the sum

\[
f_z(z) = \int_{-\infty}^{\infty} \frac{\exp\left[-x^2/(2\sigma_x^2)\right]}{(2\pi)^{1/2}\sigma_x} \cdot \frac{\exp\left[-(z-x)^2/(2\sigma_y^2)\right]}{(2\pi)^{1/2}\sigma_y} \, dx \tag{A14}
\]

which, after some calculations, is reduced to

\[
f_z(z) = \left[2\pi \cdot (\sigma_x^2 + \sigma_y^2)\right]^{-1/2} \cdot \exp\left[-z^2/(2(\sigma_x^2 + \sigma_y^2))\right] \tag{A15}
\]

i.e., a new Gaussian density function with zero mean and variance

\[
\sigma^2 = \sigma_x^2 + \sigma_y^2 \tag{A16}
\]

For equal variances of the contributing signals the resulting variance is

\[
\sigma^2 = 2\sigma_x^2 \tag{A17}
\]

Repeated use of the procedure shows that for \( N \) contributions the variance of the resulting Gaussian density function is

\[
\sigma^2 = N \cdot \sigma_x^2 \tag{A18}
\]

Myoelectric processes and measures. At low levels of muscle activity, i.e., low motor unit potential repetition rate and low degree of motor unit recruitment, the probability of having overlapping action potentials is low. A slight increase in repetition rate or number of recruited motor units, therefore, will add potentials that most likely are nonoverlapping. If we make the observation interval equal to the mean repetition rate of the motor units, the intensity (1) according to Eq. A1 will be the relevant quantity to describe the muscle activity at low contraction levels.

Thus, using Eq. A13, with the assumption that the individual action potential duration \( T_0 \) is negligible in comparison with the length of the observation interval, now considered equal to the repetition interval, and further observing that zero level peak \( \delta(z) \) makes no contribution to the signal strength measure, we find the following expressions for the
RMS and FRA measures at low rates of repetition and recruitment

\[\text{RMS}^2 = \int_{-\infty}^{\infty} z^2 \cdot |I \cdot f_p(z)| \, dz\]  
(A19)

\[\text{FRA} = \int_{-\infty}^{\infty} |z| \cdot |I \cdot f_p(z)| \, dz\]  
(A20)

or, more condensed

\[\text{RMS} \sim |I|^{1/2}\]  
\[\text{FRA} \sim |I|^{1}\] for \(I\) is small  
(A21)

At high levels of muscle activity, i.e., high motor unit repetition rates and a large number of recruited motor units, the probability of overlapping potentials is very high: the total signal has the character of random (Gaussian) noise. Any of the two processes of recruitment or repetition rate increase will add potentials randomly overlapping other potentials. Thus also in this case the product of the mean repetition rate and number of recruited units, i.e., the intensity, is the relevant quantity to describe the muscle's activity. In the case of a Gaussian density function, we find, from the equations above, the following expressions for the RMS and FRA values

\[\text{RMS}^2 = (2\pi \cdot |I| \cdot \sigma_n^2)^{-1/2} \int_{-\infty}^{\infty} z^2 \exp \left[-z^2/(2I \sigma_n^2)\right] \, dz\]  
(A22)

\[\text{FRA} = (2\pi \cdot |I| \cdot \sigma_n^2)^{-1/2} \int_{-\infty}^{\infty} |z| \exp \left[-z^2/(2I \sigma_n^2)\right] \, dz\]  
(A23)

which immediately show that

\[\text{RMS} \sim |I|^{1/2}\]  
\[\text{FRA} \sim |I|^{1}\] for \(I\) is large  
(A24)

It should be observed that in the above derivation of the expressions for RMS and FRA a dependence between recruitment order and motor unit size has not been taken into account.

A simulation of potential summation is shown in Fig. 7, where the RMS and FRA values are plotted as functions of the intensity (I). We observe a consistent difference (in the log scale) between the RMS and FRA values for high intensities in accordance with the theory which states that the quotient of the RMS and FRA values is a constant related to muscle length (L) and activation referred to the combined processes of diaphragm motor unit recruitment and motor unit firing rate. For the diaphragm, therefore, to generate pressure (force) at a given muscle length and radius of curvature [inferred from chest wall configuration (CWC)]

\[P_{di,CWC} = \text{activation} \cdot k_{CWC}\]  
(B2)

and

\[\text{maximum } P_{di,CWC} = \text{maximum activation} \cdot k_{CWC}\]  
(B3)

where maximum refers to a maximum voluntary (100% of max) Pdi or activation. In other words

\[P_{di,CWC(100\% \text{of max})} = \text{activation(100\% \text{of max})} \cdot k_{CWC}\]  
(B4)

If we want \(P_{di}/P_{di(100\% \text{of max})}\)

\[P_{di,CWC(100\% \text{of max})} = \frac{\text{activation(100\% \text{of max})}}{k_{CWC}} \]  
(B5)

Therefore

\[P_{di}/P_{di(100\% \text{of max})} = \text{activation}/\text{activation(100\% \text{of max})}\]  
(B6)

and is a measure of relative global diaphragm activation.

**APPENDIX B**

Theoretical Explanation for the Relationship Between \(P_{di}/P_{di(100\% \text{of max})}\) and Global Diaphragm Activation

When all factors affecting the contractile properties of muscle are constant, any change in neural drive to the muscle (i.e., activation) will provide a change in muscle force. During nonfatiguing, isometric contractions, one can assume that

\[\text{muscle force} = \text{activation} \cdot k_{L}\]  
(B1)

where \(k_L\) is a constant related to muscle length (L) and activation refers to the combined processes of diaphragm motor unit recruitment and motor unit firing rate. For the diaphragm, therefore, to generate pressure (force) at a given

**REFERENCES**


