Posture effects on timing of abdominal muscle activity during stimulated ventilation

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Abe, Tadashi, Takumi Yamada, Tomoyuki Tomita, and Paul A. Easton. Posture effects on timing of abdominal muscle activity during stimulated ventilation. J. Appl. Physiol. 86(6): 1994–2000, 1999.—In humans during stimulated ventilation, substantial abdominal muscle activity extends into the following inspiration as postexpiratory expiratory activity (PEEA) and commences again during late inspiration as preexpiratory expiratory activity (PREA). We hypothesized that the timing of PEEA and PREA would be changed systematically by posture. Fine-wire electrodes were inserted into the rectus abdominis, external oblique, internal oblique, and transversus abdominis in nine awake subjects. Airflow, end-tidal CO₂, and moving average electromyogram (EMG) signals were recorded during resting and CO₂-stimulated ventilation in both supine and standing postures. Phasic expiratory EMG activity (tidal EMG) of the four abdominal muscles at any level of CO₂ stimulation was greater while standing. Abdominal muscle activities during inspiration, PEEA, and PREA, were observed with CO₂ stimulation, both supine and standing. Change in posture had a significant effect on intrabreath timing of expiratory muscle activation at any level of CO₂ stimulation. The transversus abdominis showed a significant increase in PEEA and a significant decrease in PREA while subjects were standing; similar changes were seen in the internal oblique. We conclude that changes in posture are associated with significant changes in phasic expiratory activity of the four abdominal muscles, with systematic changes in the timing of abdominal muscle activity during early and late inspiration.

The contribution of the abdominal muscles to stimulated ventilation in the standing posture in awake humans is traditionally considered in the context of classic pulmonary mechanics. Mechanically, the assumption of the standing posture is associated with increased tonic or phasic abdominal muscle expiratory activity, and adjustments in diaphragm muscle length are related to direct changes in abdominal pressure that arise from abdominal muscle activity as well as passive changes in abdominal pressure that occur with recoil of the abdominal compartment. Recoil of the abdominal compartment is presumed to occur in a fashion analogous to recoil of the rib cage, with pressure generated during active contraction and released to advantage during periods of inactivity, i.e., inspiration. This fundamental concept of abdominal compartment recoil as an important assist to inspiration is intellectually appealing, and it originated with early measurements of rib cage and abdomen diameter (13) and pressure (15), which inferred abdominal influence on inspiration. Although the fact of significant recoil of the rib cage is certainly intuitive, given the obvious expansion and elevation of the ribs against gravity, it is much less obvious that the abdomen can efficiently store energy to assist a subsequent inspiration, especially in the supine position. Even in the standing posture, an abrupt relaxation of the abdominal compartment during the instant before the onset of inspiration would be expected to assist in the rapid descent of the diaphragm, but this would be somewhat counterproductive, because inspiratory diaphragm contraction might then begin at a shorter initial diaphragm length. Of course, this sudden relaxation of the abdominal compartment could be dampened and modified by tonic activity of the abdominal muscles. Such tonic abdominal activity has been demonstrated (7, 14), and the presence of tonic activity is usually implied. However, continuous tonic activity of the abdominal muscles during the erect posture may be absent. Recently, we recorded from all four abdominal muscles in supine and standing humans and did not record continuous tonic activity of any abdominal muscle, regardless of posture, in any subject (1).

What we found, instead, was a systematic firing of the abdominal muscles that persisted after expiratory flow had ceased and extended into the following inspiration, so-called postexpiratory expiratory activity (PEEA) (1). We also identified a similar but opposite systematic firing of the expiratory muscles; this started regularly in late inspiration preceding the onset of expiratory airflow [so-called preexpiratory expiratory activity (PREA)]. Taken together, these prominent, unexpected, early and late inspiratory firings of the expiratory muscles could provide a mechanism for precise breath-by-breath adjustment of abdominal pressure and diaphragm length both at the beginning and throughout each inspiration. Perhaps the abdominal muscles can assist inspiration without invoking either tonic abdominal activity or recoil of the abdominal compartment.

In this study, we examined the activity of each of the four abdominal muscles during CO₂-stimulated breathing, with subjects in both the supine and standing postures and with a particular emphasis on the identification and measurement of PEEA and PREA of these muscles with changes in posture. We hypothesized
that, during augmented ventilation, with change in posture, a systematic adjustment of intrabreath preexpiratory and postexpiratory firing of abdominal muscles would be associated with increasing tidal volume (Vt). We employed high-resolution ultrasound to insert, under direct vision, fine (<100-µm diameter) wires into each of four individual abdominal muscles to examine the relative activities of the four abdominal muscles during supine and standing CO₂-stimulated ventilation.

METHODS

Nine healthy young male subjects (age, 18–27 yr; height, 170–182 cm; weight, 60–79 kg) were studied. None had experience with respiratory maneuvers or experimentation, and all were unaware of the scientific purposes of this study. All subjects were in good health, with no history of pulmonary or neuromuscular disorders. Each subject gave informed consent.

Electrode insertion. Techniques are described in detail elsewhere (1). Briefly, the abdominal electromyogram (EMG) recording electrodes were fashioned from 80-µm OD, polyurethane-coated fine wire. These were inserted by a modified Basmajan and Stecko technique (5), ~10 mm apart, along the axis of the fiber bundles of rectus abdominis (RA), external oblique (EO), internal oblique (IO), and transversus abdominis (TA), under direct vision, guided by high-resolution, 7.5-MHz ultrasound echograph (model 415, Hitachi, Tokyo, J apan) while the subjects were awake and reclining in the supine position on a tilt bed. The sites of insertion for RA, EO, IO, and TA were, respectively, ~2 cm to the right of the umbilicus, right middiaphragmatic line at the level of the umbilicus, 2 cm below the level of umbilicus on the midclavicular line, and 1 cm below the right costal margin on the anterior axillary line. As described previously, after wires were inserted, a series of respiratory maneuvers was performed to confirm correct placement and recording fidelity of the fine-wire electrodes (1). At the conclusion of the experiment, these respiratory and trunk maneuvers were repeated, and we were able to confirm that the position of the wires was unchanged.

Measurement techniques. Ventilation was measured during both resting and CO₂-stimulated breathing in supine (1) and standing postures. Subjects were reminded repeatedly to remain relaxed. The experimenters were especially careful not to draw the attention of the subjects to the electrodes or the abdominal muscles. After the electrodes had been inserted, posture was changed from supine to standing by using the tilt bed; this avoided the possibility of electrode displacement by active postural movement (1). Ventilatory recordings were made with the subjects breathing through a mouthpiece, through a low-resistance breathing circuit (~1 cmH₂O·l·1·s) that was connected to a hot-wire pneumotachograph (Transducer no. 023115, Minato Medical Science, Tokyo, J apan). On the expiratory limb, CO₂ was sampled and analyzed continuously (Aeromonitor AE280, Minato Medical Science). Ventilatory and abdominal muscle responses to progressive hypercapnia were elicited by a modification of the Read technique (16) (rebreathing 6.9% CO₂ in O₂ from a 6- to 8-liter bag).

EMG signals from the electrodes were amplified (model 7512, NEC-San-ei, Tokyo, J apan), band-pass filtered (Bessel type, 20 Hz to 3 kHz; NF Filters, Tokyo, J apan), and recorded simultaneously with respiratory airflow and end-tidal CO₂ (ETCO₂) on a digital audiotape data recorder (model PC116, Sony, Tokyo, J apan). At the same time, the output signals were rectified and processed by a resistance capacitor with a time constant of 100 ms (model EI-601G, Nihon-Kohden, Tokyo, J apan) to provide a continuous moving average EMG for RA, EO, IO, and TA. These moving average signals were then gathered with respiratory airflow and ETCO₂ directly to hard disk on a microcomputer by using acquisition software (DataSponge, Bioscience Analysis Software, Calgary, Alberta) and a single-board analog-to-digital system (model MIO-16-H-9, National Instruments, Galveston, TX) for subsequent examination by using a series of dedicated analysis programs written by one of the authors (P. A. Easton).

Analysis of ventilation. The respiratory flow signal was evaluated for respiratory timing and was digitally integrated. Minute ventilation (Vi), respiratory frequency, Vt, inspiratory time, expiratory time, and total time per breath were calculated breath by breath. Indexes of breathing pattern and EMG activity were calculated during resting ventilation and at three levels of CO₂-stimulated breathing in both the supine and standing postures. In the supine posture, as in a previous publication (1), we defined “high” CO₂ (CO₂-H) as the highest ETCO₂ level endured comfortably by each subject, and medium and low levels of ETCO₂ (CO₂-M and CO₂-L, respectively) were selected at 10 and 20 Torr below maximum levels, respectively. The values of CO₂-H, CO₂-M, and CO₂-L were chosen first in the supine posture for each subject, then matching levels were identified in the standing posture.

EMG analysis. Resting EMG of these expiratory muscles was defined as the lowest moving-average EMG activity measured during inspiration. Tidal EMG was calculated as the maximum difference per breath between resting EMG and the peak height of the moving-average EMG signal. These measurements defined “tidal” activity of respiratory airflow and timing as well as EMG activity of RA, EO, IO, and TA.

Duration and magnitude of PEEA and PREA were calculated from traces of airflow and the raw and moving average EMG signal. Specifically, for each breath, the beginning of expiration was identified from the airflow trace. At the exact moment when expiratory airflow began, the value of the moving-average EMG defined the magnitude of PREA, which was expressed as arbitrary units (AU). The onset of expiratory EMG activity was identified for each breath on the raw EMG trace. The time difference between the beginning of expiration and the (preceding) onset of expiratory EMG activity was calculated in seconds as the duration of PREA.

Next, for each breath, the ending of expiration was identified from the airflow trace. At the exact moment when expiratory airflow ended, the value of the moving average EMG defined the magnitude of PEEA, which was expressed in AU. Then the termination of expiratory EMG activity was identified for each breath from the raw EMG trace. The time difference between the ending of expiratory airflow and the last point of (persistent) activity of expiratory EMG after expiratory airflow had ceased was calculated (in seconds) as the duration of PEEA. These calculations of PREA and PEEA were repeated for TA and IO. Thus any PREA of the expiratory muscles occurred during the final portion of inspiratory airflow, whereas PEEA spilled over into the inspiratory airflow of the next breath. If expiratory EMG occurred simultaneously with expiratory airflow, then the magnitude and duration of both PREA and PEEA would be zero.

Statistical analysis. After calculation, mean values were exported for review to spreadsheet software (Microsoft Excel, Microsoft, Redmond, WA), to graphic software to output figures (see Figs. 1 and 3), and to the PC version of SAS (18) for statistical analysis. The slope of the EMG response to progressive hypercapnia was calculated by linear regression.
by using the method of least squares (20) (being careful to utilize tidal EMG normalized by initial tidal EMG values to compute the slope). The mean slopes were compared between supine and standing postures for each muscle by paired t-test. Mean values for breathing pattern and tidal EMG activity were compared across the four conditions (resting and the three levels of CO2-stimulated breathing: CO2-L, CO2-M, and CO2-H) at the two postures, by three-way mixed-model ANOVA with repeated measures on two factors (12, 20). Mean values for the main effects of posture change and CO2 were compared by multiple-comparison testing, specifically Tukey’s test (20). Mean values at the same level of CO2 were also compared between supine and standing postures by individual paired t-tests. Mean values for magnitude and duration of PEEA and PREA across three levels of CO2-stimulated breathing at the two postures were also compared by three-way ANOVA, with repeated measures on two factors and post hoc multiple-comparison testing by Tukey’s test.

RESULTS

During insertion of the fine-wire electrodes or measurement of resting or stimulated ventilation, subjects reported that discomfort was minimal; no subject required analgesia. The magnitude of EMG activity recorded during head-up position and during visual feedback-controlled respiratory maneuvers was not different in any subject, when the values before and after the series of ventilatory measurements were compared. In addition, at the end of the experiment, we confirmed that the fine-wire electrode had remained at the same depth as the guide needle had been advanced initially under ultrasound.

In each posture, supine and standing, mean values were compared during resting (Rest) ventilation and across three levels of CO2-stimulated breathing that corresponded to low (CO2-L), medium (CO2-M), and high (CO2-H) levels of ETCO2. In supine posture, mean ETCO2 values were 44.1, 55.6, 64.7, and 74.1 Torr for Rest, CO2-L, CO2-M, and CO2-H, respectively, with standing values within 1 Torr of the corresponding supine values.

Ventilatory and abdominal muscle responses to postural change and CO2 stimulation. Breathing pattern changed consistently during progressive hypercapnia and with change in posture from supine to standing during resting VT (Table 1). Overall, posture had a significant effect on all components of breathing pattern (Table 1), as did an increase in CO2 in general. When we looked at specific indexes of breathing pattern, VT during Rest increased slightly from supine to standing posture, from a mean of 0.54 ± 0.12 (SD) liter in supine to 0.61 ± 0.16 liter in standing posture. VT during resting ventilation increased from supine to standing posture, from 7.6 ± 2.4 liters in supine to 10.0 ± 2.0 liters in standing posture. During progressive hypercapnia, VT increased markedly (P < 0.0001) with increasing CO2 (to 1.10 ± 0.31, 1.79 ± 0.50, and 1.99 ± 0.54 liters during CO2-L, CO2-M, and CO2-H, respectively, in supine posture; and to 1.03 ± 0.24, 1.52 ± 0.26, and 1.90 ± 0.38 liters during CO2-L, CO2-M, and CO2-H, respectively, in standing posture.) VT increased markedly (P < 0.0001) with increasing CO2 in both supine and standing postures.

Across the different conditions (Rest, CO2-L, CO2-M, and CO2-H), group mean values of EO, IO, and TA tidal EMG (Fig. 1 and Table 1) were significantly different in supine posture compared with the standing posture (P < 0.0001). Only RA tidal EMG failed to change significantly with posture change. However, the EMG activity of the four muscles at each condition was not identical. All muscles did show progressively increasing EMG with increasing hypercapnia. Mean Rest and CO2-L tidal EMG values in standing position for EO, IO, and TA were significantly higher than those in supine position (P < 0.01, P < 0.02, and P < 0.02,

Table 1. Breathing pattern and EMG

<table>
<thead>
<tr>
<th></th>
<th>Supine</th>
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<th></th>
<th>Standing</th>
<th></th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Resting</td>
<td>Low</td>
<td>Medium</td>
<td>High</td>
<td>Resting</td>
<td>Low</td>
</tr>
<tr>
<td>VT, l/min</td>
<td>7.6 ± 2.4</td>
<td>15.1 ± 5.7</td>
<td>33.9 ± 18.6</td>
<td>49.1 ± 22.7</td>
<td>10.0 ± 2.0</td>
<td>0.0002</td>
</tr>
<tr>
<td>f, breathe/</td>
<td>14.4 ± 5.1</td>
<td>13.7 ± 3.4</td>
<td>19.0 ± 7.2</td>
<td>25.6 ± 9.6</td>
<td>17.4 ± 6.0</td>
<td>(0.004)</td>
</tr>
<tr>
<td>Vt, liters</td>
<td>0.54 ± 0.12</td>
<td>1.10 ± 0.31</td>
<td>1.79 ± 0.50</td>
<td>1.99 ± 0.54</td>
<td>0.61 ± 0.16</td>
<td>(0.023)</td>
</tr>
<tr>
<td>Ti, s</td>
<td>1.77 ± 0.46</td>
<td>1.97 ± 0.47</td>
<td>1.65 ± 0.58</td>
<td>1.33 ± 0.59</td>
<td>1.42 ± 0.45</td>
<td>(0.047)</td>
</tr>
<tr>
<td>Te, s</td>
<td>2.85 ± 0.92</td>
<td>2.73 ± 0.85</td>
<td>1.95 ± 0.78</td>
<td>1.41 ± 0.62</td>
<td>2.44 ± 0.83</td>
<td>(0.182)</td>
</tr>
<tr>
<td>Tr/Tt</td>
<td>0.39 ± 0.04</td>
<td>0.42 ± 0.04</td>
<td>0.47 ± 0.04</td>
<td>0.49 ± 0.05</td>
<td>0.37 ± 0.04</td>
<td>(0.362)</td>
</tr>
</tbody>
</table>

Values are means ± SD; n, 9 men. VT, minute ventilation; f, frequency in breaths/min; Vt, tidal volume; Ti, inspiratory time; Te, expiratory time; Tr, total time per breath; Tidal EMG, maximum difference per breath between resting electromyogram (EMG) and peak height of the moving-average EMG; AU, arbitrary unit; RA, rectus abdominis; EO, external oblique; IO, internal oblique; TA, transversus abdominis. Mean values of end-tidal CO2 were 55.6, 64.1, and 74.1 mmHg for CO2-L, medium, and high, respectively, in the supine position; mean values of end-tidal CO2 in the standing posture were 56.0, 64.6, and 74.0 mmHg. Values in the parentheses are P values for individual comparisons between supine and standing at specific CO2 levels. Right 2 columns report significance for overall effects of levels of CO2 and different posture. NS, not significant.
respectively), whereas values of RA in standing and supine positions were not different during Rest and CO2-L. To reach the standing position, subjects were tilted to a vertical position by using the tilt bed, then they stood relaxed and free of the table. In this standing position, there was no tonic EMG activity remaining throughout inspiration for any muscle in any subject.

The slopes of the CO2-response curve (ΔEMG/ΔETCO2) for RA, EO, IO, and TA were not different between supine and standing postures. However, values of intercept were different for IO and TA (P < 0.01, P < 0.03, respectively) between supine and standing postures.

PEEA and PREA. Examples of raw EMG signals from one subject in supine and standing postures, obtained during progressive hypercapnia with ETCO2 of 58.5 Torr, are shown in Fig. 2, A and B, respectively. In this subject, RA, IO, and TA showed phasic expiratory EMG activity. In addition to phasic EMG activity during expiration, EMG activity was seen clearly dur-

![Image of EMG activity](https://example.com/image1.png)

Fig. 1. Peak tidal electromyogram (EMG) of transversus abdominis (TA) during CO2-stimulated breathing in supine and standing positions; y-axis shows group mean peak tidal moving average EMG activity (tidal EMG) for TA, in arbitrary units (AU). The x-axis shows resting ventilation and increasing CO2 stimulation: Low CO2, Med CO2, High CO2 correspond to end-tidal CO2 (ETCO2) of 55.6, 64.1, and 74.1 Torr, or low, medium, and high, respectively. Bars show means ± SE; solid bars, supine posture; hatched bars, standing postures. Rate of increase of phasic tidal EMG was not significantly different for TA regardless of posture.

![Image of EMG signals](https://example.com/image2.png)

Fig. 2. A: EMG activity of abdominal muscles during supine CO2-stimulated breathing. Top (2 traces): raw EMG activity of rectus abdominis (RECTUS) and external oblique (EXTERN). Middle (4 traces): moving average and raw EMG of internal oblique (INTERN) and TA (TRANSV). Bottom: airflow trace. Results are for a single subject at ETco2 of 58.5 Torr. Solid arrows, postexpiratory expiratory activity (PEEA); open arrows, preexpiratory expiratory activity (PREA). B: EMG activity of abdominal muscles during standing CO2-stimulated breathing. Abbreviations and traces as in A. Results are from same subject at same ETco2. With movement from supine to standing posture, amplitude and duration of PEEA increase, whereas amplitude and duration of PREA decrease, for both INTERN and TRANSV.
ing early and late inspiration in IO, and TA. It is evident that phasic expiratory EMG activity persisted into the following inspiratory phase as PEEA and also preceded expiratory flow as PREA. In this subject, PEEA is more prominent in the standing posture (Fig. 2B). On the other hand, in the supine posture (Fig. 2A), PREA is more prominent, whereas PEEA has diminished.

Group mean values for magnitude and duration of both PREA and PEEA of IO and TA are shown in Table 2. As seen in the right column, there was a highly significant effect of postural change, regardless of CO₂ level, on both PREA and PEEA of the two muscles. Although an overall effect of CO₂, regardless of posture, was not significant globally, as seen in Table 2, there were very significant differences for specific levels of CO₂ between postures. Specifically, the magnitude of PEEA for TA during CO₂-H increased significantly from supine to standing posture, from a mean of 0.71 ± 0.79 AU in supine to 1.42 ± 1.10 AU in standing posture. Duration of PEEA for TA during CO₂-H also increased significantly from supine to standing posture, from a mean of 0.24 ± 0.33 s in supine to 0.46 ± 0.28 s in standing posture. On the other hand, the magnitude of PREA for TA during CO₂-H decreased significantly from supine to standing posture, from a mean of 1.25 ± 1.23 AU in supine to 0.78 ± 0.62 AU in standing posture. Moreover, duration of PREA for TA during CO₂-H also decreased significantly from supine to standing posture, from a mean of 0.42 ± 0.38 s in supine to 0.20 ± 0.21 s in standing posture. Similar changes in intrabreath timing of PEEA and PREA were seen at all levels of CO₂ in TA and IO. Group mean results for duration and magnitude of TA PEEA and PREA are illustrated in Fig. 3.

Fig. 3. Magnitude and duration of PEEA and PREA of TA in different postures; levels of ETCO₂ are as defined in Fig. 1. Bars show means ± SE; solid bars, supine posture; hatched bars, standing postures; x-axis shows increase in CO₂ stimulation. A: magnitude of PEEA; y-axis shows magnitude of group mean PEEA for TA. B: duration of PEEA; y-axis shows duration of group mean PEEA for TA (in s). C: magnitude of PREA; y-axis shows magnitude of group mean PREA. D: duration of PREA; y-axis shows duration of group mean PREA for TA.
DISCUSSION

These results confirmed that the ventilatory and expiratory muscle responses to CO₂ were unchanged with change in posture from supine to standing, yet the breathing pattern and EMG at specific levels of CO₂ were significantly different according to posture. Thus the Vₖ and peak tidal EMG at any level of CO₂ stimulation were greater while standing. PEEA and phasic expiratory EMG activity preceding expiratory flow (PREA) were observed with both CO₂ stimulation and postural change. Postural change had a significant effect on intrabreath PREA and PEEA muscle activity across all levels of CO₂ stimulation. With a change to the standing posture, there were major increases in duration and magnitude of PEEA and significant decreases in PREA. Similar changes in intrabreath timing of PEEA and PREA were seen in the most internal pair of abdominal muscles, i.e., TA and IO.

Ventilatory and abdominal muscle response to progressive hypercapnia. The purpose of this study was to evaluate the effects of posture on the timing of activation of the expiratory muscles during stimulated breathing, so the investigation compared breathing pattern and intrabreath muscle activation in two postures at several matched levels of CO₂. Such a comparison rests on the reasonable assumption that any changes detected are accounted for by the change in posture and are unrelated to slope of the CO₂ response per se. This presumes that the ventilatory-response curve for CO₂ is constant, regardless of posture; otherwise, a change in the CO₂-response curve simply because of posture might confound the study. Fortunately, we found that the slope of both Vₖ and peak tidal expiratory EMG responses were not changed with standing, only offset laterally so that the intercept shifted. This result is consistent with the observation, more than 30 years ago, by Anthonisen et al. (4) that the ventilatory response to CO₂ did not change with the erect posture, and that the CO₂ ventilatory response constructed from a series of steady-state inhalations was displaced in parallel when moving from supine to standing. Similar results were noted by Rigg et al. (17) with CO₂ rebreathing; ventilatory responsiveness was unchanged from supine to standing positions. Therefore, despite the significant increase in functional residual capacity that we know occurs with standing, the change in lung volume is associated with only a modest increase in ventilatory and expiratory EMG activity per increment of ETCO₂, and the CO₂ responsiveness is not affected.

Changes in PEEA of abdominal muscles. As in our previous study (1), we observed phasic EMG activity extending into the subsequent inspiration as PEEA, especially in IO and TA. In this study, we attempted to examine the effect, if any, of posture on the timing of this intrabreath muscle activation. At matched levels of CO₂-stimulated breathing, a postural change from supine to standing was associated with a major increase in both the duration of PEEA extending into the subsequent inspiration, as defined by inspiratory airflow, as well as the magnitude of this PEEA activity. This PEEA activity is analogous to inspiratory activity that persists into the following expiration of inspiratory muscles, entitled postinspiratory inspiratory activity. In addition to our previous report on humans, PEEA has been documented in awake dogs for TA during standing (3), hypercapnia (19), or hypoxia (11).

Although the intrabreath nature of the change may appear subtle at first glance, the mechanical impact of PEEA could be significant. We base this enthusiastic appraisal on the extensive literature pertaining to the mechanics of breathing with change in lung volume and posture (2, 6, 7, 9, 10, 13). In classic studies, when humans are moving from supine to erect posture, end-expiratory lung volume increases, which is presumably accompanied by a descent and shortening of the diaphragm. Since the 1950s (6, 9), it has been recognized that the standing posture is also associated with the appearance of abdominal muscle activity. In 1983, De Troyer and Loring (7) demonstrated that abdominal EMG, presumed to be tonic, was associated with a reduction of abdominal volume throughout the respiratory cycle, with the result that standing subjects were breathing consistently to the left of their relaxed thoracoabdominal configuration, as illustrated by a Konno-Mead plot (13). This reduction in abdominal volume had important mechanical consequences (10), because, for a given EMG activity, the pressure generated by diaphragmatic contraction was related to abdominal volume, with diaphragmatic pressure that rose significantly with reduced abdominal volume. The link between abdominal volume and pressure with inspiratory transdiaphragmatic pressure was diaphragm length, or, more precisely, the beneficial effect of increased abdominal pressure to increase end expiratory diaphragm length, thus improving diaphragm mechanical efficiency according to its length-tension relationship (8). This mechanical explanation is still relevant to our computerized calculations of intrabreath abdominal expiratory EMG. With a shift from supine to standing posture, the tendency of the diaphragm to descend and shorten at end expiration will be counteracted by the increased, persistent expiratory muscle activity expressed here as PEEA. In earlier studies, any advantageous preinspiratory adjustment of diaphragm length has been attributed to abdominal muscles through either tonic abdominal electrical activity or strong phasic expiratory contraction, but both of these mechanisms could be inefficient. Even an extremely strong phasic abdominal contraction that ceased abruptly before the onset of the subsequent inspiratory diaphragm activation would be unlikely to prevent the rapid descent and end-expiratory shortening of the diaphragm. Similarly, sustained tonic activity of the abdominal muscles could help preserve end-expiratory diaphragm length, but such sustained activity would be inefficient compared with a few milliseconds of phasic PEEA that spanned a brief portion of total time per breath. These results suggest that, during stimulated breathing in the standing posture, end-expiratory preservation of diaphragm length could be achieved with-
out an increase in the maximal amplitude of phasic or sustained tonic abdominal electrical activity.

Changes in PREA of abdominal muscles. Although prominent postexpiratory activity characterized stimulated ventilation while standing, accentuated PREA was the hallmark of supine stimulated ventilation. That is, supine CO2-stimulated breathing was characterized by significant activation of the internal layer of abdominal expiratory muscles that began in the latter portion of the preceding inspiration, long before any expiratory airflow. Thus phasic EMG activity of the expiratory muscles during a substantial period of inspiration is common to both supine and standing ventilation, but the time during inspiration is a function of posture. Of course, this begs the question as to what advantage could be conferred by PREA as an apparently inefficient premature activation of expiratory muscles before inspiratory airflow has finished.

Abdominal compartment recoil and tonic abdominal muscle activity. It is of interest that, as in our previous study (1), we did not identify or record continuous tonic EMG activity of any abdominal muscle. In this discussion, we have also considered the possible actions and effects of both PEEA and PREA without any reference to either tonic abdominal EMG or abdominal compartment recoil. In lieu of these classic explanations, what these EMG recordings seem to offer is a different means of adjustment of diaphragm length and optimization of abdominal pressure and compliance regionally. The existence of complementary pre- and postexpiratory activity allows for a dynamic, breath-by-breath, real-time optimization of diaphragm length and mechanics that is hard to imagine with whole abdominal compartment contraction and relaxation.

Our observation that tonic abdominal activity was not present throughout inspiration during supine (1) or standing posture can be reconciled with previous studies. For example, the PREA and PEEA activity in Fig. 2B could have been interpreted as tonic activity if the time base resolution of the EMG did not allow clear identification of the silent portions at peak inspiration. Moreover, if the subjects had not been encouraged to remain relaxed, as was done in these studies, continuous abdominal EMG could have persisted across inspiration, superimposed on PREA and PEEA. Furthermore, the recordings may have been influenced by the regional nature of the fine-wire electrodes. Such implants sample a limited territory of the muscle, and these recordings were made from electrodes implanted relatively cranially in the abdominal musculature, whereas previous recordings that showed more tonic abdominal activity originated from more caudal portions of the abdomen (7). Thus it is not our intent to suggest that tonic abdominal EMG does not occur but only that we did not record tonic abdominal EMG in these studies.

This work was supported by Monbusho (National Ministry of Science and Education), Japan. P. A. Easton was a Scholar of the Medical Research Council of Canada.

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Received 17 February 1998; accepted in final form 19 February 1999.

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