Association of chest wall motion and tidal volume responses during CO₂ rebreathing

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Yan, Sheng, Pawel Sliwinski, and Peter T. Macklem. Association of chest wall motion and tidal volume responses during CO₂ rebreathing. J. Appl. Physiol. 81(4): 1528–1534, 1996.—The purpose of this study was to investigate the effect of chest wall configuration at end expiration on tidal volume (VT) response during CO₂ rebreathing. In a group of 11 healthy male subjects, the changes in end-expiratory and end-inspiratory volumes of the rib cage (ΔVrc,E and ΔVrc,I, respectively) and abdomen (ΔVab,E and ΔVab,I, respectively) measured by linearized magnetometers were expressed as a function of end-tidal PCO₂ (PETCO₂). The changes in end-expiratory and end-inspiratory volumes of the chest wall (ΔVcw,E and ΔVcw,I, respectively) were calculated as the sum of the respective rib cage and abdominal volumes. The magnetometer coils were placed at the level of the nipples and 1–2 cm above the umbilicus and calibrated during quiet breathing.

The VT response during CO₂ rebreathing. In a group of 11 healthy male subjects, the changes in end-expiratory and end-inspiratory volumes of the chest wall (ΔVcw,E and ΔVcw,I, respectively) were calculated as the sum of the respective rib cage and abdominal volumes. The magnetometer coils were placed at the level of the nipples and 1–2 cm above the umbilicus and calibrated during quiet breathing against the VT measured from a pneumotachograph. The ΔVrc,E/ΔPETCO₂ slope was quite variable among subjects. It was significantly positive (P < 0.05) in five subjects, significantly negative in four subjects (P < 0.05), and not different from zero in the remaining two subjects. The ΔVab,E/ΔPETCO₂ slope was significantly negative in all subjects (P < 0.05) with a much smaller intersubject variation, probably suggesting a relatively more uniform recruitment of abdominal inspiratory muscles and a variable recruitment of rib cage muscles during CO₂ rebreathing in different subjects. As a group, the mean ΔVrc,E/ΔPETCO₂, ΔVab,E/ΔPETCO₂, and ΔVcw,E/ΔPETCO₂ slopes were 0.010 ± 0.034, −0.030 ± 0.007, and −0.020 ± 0.032 l/Torr, respectively; only the ΔVab,E/ΔPETCO₂ slope was significantly different from zero. More interestingly, the individual ΔVT/ΔPETCO₂ slope was negatively associated with the ΔVrc,E/ΔPETCO₂ (r = −0.68, P = 0.021) and ΔVcw,E/ΔPETCO₂ slopes (r = −0.63, P = 0.037) but was not associated with the ΔVab,E/ΔPETCO₂ slope (r = 0.40, P = 0.223). There was no correlation of the ΔVrc,E/ΔPETCO₂ and ΔVcw,E/ΔPETCO₂ slopes with age, body size, forced expiratory volume in 1 s, or expiratory time. The group ΔVab,I/ΔPETCO₂ slope (0.004 ± 0.014 l/Torr) was not significantly different from zero despite the VT nearly being tripled at the end of CO₂ rebreathing. In conclusion, the individual VT response to CO₂, although independent of ΔVab,E, is a function of ΔVrc,E to the extent that as the ΔVrc,E/ΔPETCO₂ slope increases (more positive) among subjects, the VT response to CO₂ decreases. These results may be explained on the basis of the respiratory muscle actions and interactions on the rib cage.

carbon dioxide; chest wall mechanics; end-expiratory volume; expiratory muscles; rib cage; abdomen

ONE OF THE FEATURES of CO₂-stimulated breathing is consistent recruitment of abdominal expiratory muscles (12, 13, 18). In the upright posture, it is believed that abdominal expiratory muscle recruitment decreases end-expiratory volume of the abdomen (Vab,E), which may optimize diaphragmatic performance and assist inspiration by relaxation at the beginning of inspiration, allowing the abdominal contents to displace the abdominal wall anteriorly, causing the diaphragm to descend and reducing the elastic work performed by inspiratory muscles (12, 18, 29).

Although expiratory muscle recruitment during CO₂-stimulated breathing has been repeatedly inferred from respiratory pressures and chest wall displacement measurements (12, 18, 29) and confirmed by expiratory muscle electromyograms (26, 27), quantitative studies of changes in end-expiratory lung volume (Vl,E) during CO₂ rebreathing are not numerous in humans. It has been suggested by Henke et al. (15) with helium dilution and by Cha et al. (6) with repeated vital capacity maneuvers that Vl,E does not change significantly during CO₂ rebreathing. If the abdominal expiratory muscles are recruited, resulting in a decrease in Vab,E during CO₂ rebreathing, but fail to alter Vl,E, the end-expiratory volume of the rib cage (Vrc,E) must increase (17), resulting in distortion of the chest wall away from the relaxation configuration during expiration. The magnitude of these changes may impact on tidal volume (VT) generation at a given level of CO₂.

We hypothesize that the changes in chest wall configuration at end expiration influence the magnitude of VT response to CO₂ and might account for some of the variance in this response among individuals. The purpose of the present study is therefore to measure the compartmental changes in Vrc,E and Vab,E (ΔVrc,E and ΔVab,E, respectively) and end-inspiratory volumes of the rib cage (ΔVrc,I) and abdomen (ΔVab,I) in normal subjects and relate them to the individual VT response to CO₂.

METHODS

Eleven healthy male subjects participated in the present study. Most of the subjects were laboratory staff familiar with respiratory maneuvers, but none of them knew the purpose of the present study. Informed consent was obtained from all the subjects, and the experimental procedure was approved by the Ethics Committee of the Montreal Chest Hospital. The data for age, height, body weight, forced expiratory volume in 1 s (FEV₁), and the ratio of FEV₁ to forced vital capacity (FVC) (FEV₁/FVC) of the subjects are given in Table 1.

During the experiment, the subjects were seated comfortably in an armchair with the head and back firmly supported to assure a constant body posture. They breathed through a mouthpiece with a noseclip. Respiratory flow was measured by a Fleisch no. 2 pneumotachograph and a Validyne differential pressure transducer (MP-45−2). VT was measured by integrating the flow signal. The mouthpiece was attached via the pneumotachograph to a two-way Hans Rudolph valve.
Table 1. Subjects’ characteristics

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>Age, yr</th>
<th>Ht, cm</th>
<th>Wt, kg</th>
<th>FEV₁, liters</th>
<th>FEV₁/FVC, %</th>
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<td>164</td>
<td>68</td>
<td>3.8</td>
<td>84</td>
</tr>
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<td>4</td>
<td>32</td>
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<td>86</td>
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<td>178</td>
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<td>32</td>
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<td>80</td>
<td>3.8</td>
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<td>7</td>
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<td>2.9</td>
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<td>34</td>
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<td>60</td>
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<td>88</td>
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<td>9</td>
<td>39</td>
<td>174</td>
<td>70</td>
<td>4.3</td>
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<td>172</td>
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Ht, body height; Wt, body weight; FEV₁, forced expiratory volume in 1 s; FVC, forced vital capacity.

One port of the valve was open to the ambient air, and the other port was connected to a rebreathing bag that contained 6–8 liters of 7% CO₂–93% O₂. End-expiratory CO₂ concentrations were measured at the mouthpiece by a CO₂ analyzer system (Ametek CD-3A). Two pairs of linearized magnetometer coils were placed on the chest wall of the subjects. One pair was positioned at the nipple level and the other pair 2 cm above the umbilicus anteroposteriorly to measure rib cage and abdominal displacements and estimate their volume changes during breathing. The relative gain of the chest wall motion signals from the magnetometers was determined by the isovolumetric method (17). The motion signal was converted to volume changes by calibration during quiet breathing against VT measured by integration of the flow (28).

At the beginning of the experiment, the subjects breathed room air for a few minutes. When all the recorded signals stabilized, the subjects were switched from room air to the rebreathing bag. CO₂ rebreathing was started by an inspiratory capacity inspiration followed by spontaneous breathing (24). Rebreathing was stopped at an end-tidal CO₂ concentration of ~8%. In all the subjects, this took ~3–4 min.

All the signals were amplified and recorded by an eight-channel strip-chart recorder (Hewlett-Packard 7758B) and stored in a computer at 200 Hz after passing a 12-bit analog-to-digital converter. The data were analyzed in the computer by using Anadat-Labdat software (JHT InfoDat, analog-to-digital converter. The data were analyzed in the channel strip-chart recorder (Hewlett-Packard 7758B) and subject, changes in minute ventilation (DVT), breathing frequency, VT, expiratory time (DTE), and end-expiratory and end-inspiratory volumes of each compartment were calculated as the Vr of each corresponding compartment (Vr,rc and Vr,ab, respectively). All the volume measurements by magnetometers were corrected by the ratio of VT by magnetometers over that by pneumotachograph. The data in the text are expressed as means ± SD. For each subject, changes in minute ventilation (ΔVE), breathing frequency, VT (ΔVT), expiratory time (ΔTE), and end-expiratory and end-inspiratory volumes of each compartment were all fitted as linear functions of end-tidal CO₂ partial pressure (PETCO₂) by the least squares method, and the slopes of each function were reported. The possible association between these slopes was tested by using Pearson’s product-moment correlation. In four subjects, the difference of a given slope from repeated runs was tested by analysis of covariance for each subject. P < 0.05 was considered as indicating statistical significance.

RESULTS

At the end of CO₂ rebreathing, VT almost tripled. The group VT, breathing frequency, and VE responses to CO₂ were 0.11 ± 0.04 l/mmHg, 0.413 ± 0.445 breath·min⁻¹·mmHg⁻¹, and 2.69 ± 1.09 l·min⁻¹·mmHg⁻¹, respectively. TE was shortened with increasing CO₂ in most subjects. The group TE response to CO₂ was −0.058 ± 0.057 s/mmHg.

Figure 1 shows VT measured by chest wall displacement plotted against VT measured by integrating flow for each breath in all the subjects during CO₂ rebreathing. The two long dashed lines represent the limits of ±15% variations from identity. The two measurements were within ±15% until VT was >2.0–2.5 liters. The relationship was linear with a regression coefficient of 0.97, an intercept of −0.017 liter, and a slope of 1.04.

In Fig. 2, ΔVrCE, ΔVab,E, and changes in Vcw,E (ΔVcw,E) during CO₂ rebreathing are plotted for three subjects who behaved differently. As shown in this figure, with increasing PETCO₂, subject 1 increased VrCE and subject 3 decreased VrCE whereas subject 2 did not have any consistent change in VrCE. Thus the slopes of ΔVrCE, ΔVab,E, and ΔVcw,E as a function of PETCO₂ for each subject were calculated (Fig. 3A). Of the 11 subjects studied, ΔVrCE/PETCO₂ slope was significantly positive (P < 0.05) in five subjects, significantly negative in four subjects (P < 0.05), and not different from zero in two subjects; the group ΔVrCE/PETCO₂, and ΔVcw,E/PETCO₂ slopes were 0.010 ± 0.034 and −0.020 ± 0.032 l/Torr, respectively, which were not significantly different from zero. In contrast, the
The slope of *ΔVab,E/ΔPETCO2* was significantly negative in all subjects; the group mean *ΔVab,E/ΔPETCO2* slope was -0.030 ± 0.007 liter/Torr, which was significantly less than zero (*P* < 0.001). Therefore, *ΔVrc,E/ΔPETCO2* slopes were much more variable than *ΔVab,E/ΔPETCO2* slopes among our subjects.

Figure 3B shows *Vrc,I*, *Vab,I*, and *Vcw,I* responses to CO2. The positive *ΔVcw,I/ΔPETCO2* slope was entirely due to the *Vrc,I* response to CO2 because the group *ΔVab,I/ΔPETCO2* slope of 0.004 ± 0.014 liter/Torr was not significantly different from zero, despite a significantly positive *ΔVT,ab/ΔPETCO2* slope (Fig. 3C). Thus almost all of *ΔVT,ab* produced by CO2 was accomplished below the equilibrium volume of this compartment, whereas the *ΔVT,rc* response to CO2 was partly due to a positive *ΔVrc,E/ΔPETCO2* slope in all subjects and partly due to the recruitment of rib cage volume below the equilibrium in subjects whose *ΔVrc,E/ΔPETCO2* slope was negative (Fig. 3). This caused a variability in *ΔVT,rc/ΔPETCO2* slope that was much larger than the variability in *ΔVT,ab/ΔPETCO2* slope, a finding consistent with previous observations (7,23).

As shown in Fig. 4, the *ΔVT/ΔPETCO2* slope was negatively associated with the *ΔVrc,E/ΔPETCO2* slope (*r* = -0.68, *P* = 0.021) and with the *ΔVcw,I/ΔPETCO2* slope (*r* = -0.63, *P* = 0.037) but was not associated with the *ΔVab,E/ΔPETCO2* slope (*r* = 0.40, *P* = 0.223). The *ΔVT/ΔPETCO2* slope was not significantly correlated with the *ΔVrc,I/ΔPETCO2* slope (*r* = 0.47, *P* = 0.140), *ΔVab,I/ΔPETCO2* slope (*r* = 0.45, *P* = 0.160), and *ΔVcw,I/ΔPETCO2* slopes (*r* = 0.55, *P* = 0.079). There was no association between the *ΔVE/ΔPETCO2* slope and end-expiratory or end-inspiratory volume responses to CO2 from either the rib cage, abdomen, or the chest wall as a whole.

There was no apparent correlation of the *ΔVrc,E/ΔPETCO2* and *ΔVcw,I/ΔPETCO2* slopes with subjects' characteristics such as age, height, body weight, FEV1, FEV1/FVC, and *Te* response to CO2 (*ΔTe/ΔPETCO2*). The correlation coefficients *r* and respective *P* values are summarized in Table 2.
Reproducibility of $D_{Vrc,E}/D_{PETCO2}$, $D_{Vab,E}/D_{PETCO2}$, and $D_{VE,w}/D_{PETCO2}$ slopes was assessed for two CO2 rebreathing runs in four subjects. As shown in Table 3, there was no significant change in these slopes during repeated runs for two subjects; for the other two subjects, significant changes in these slopes were found in the second run when compared with those of the first run, which, however, were accompanied by comparable significant changes in $D_{VT}/D_{PETCO2}$ and/or $D_{VE}/D_{PETCO2}$ slope in these subjects. These results suggest that compartmental end-expiratory volume response to CO2 is not more variable than VT and/or VE response to CO2 for a given subject.

**DISCUSSION**

We demonstrated that during CO2 rebreathing $D_{Vab,E}/D_{PETCO2}$ slope was negative in all subjects, with a small intersubject variability. On the other hand, $D_{Vrc,E}/D_{PETCO2}$ slope was much more variable, to the extent that some subjects had a positive slope, whereas others had a negative slope. Thus some subjects had a progressive increase, whereas others had a progressive decrease in $Vrc,E$ during CO2 rebreathing (Figs. 2 and 3A). The variable $D_{Vrc,E}/D_{PETCO2}$ slopes had a “carry-over” effect to the $D_{Vcw,E}/D_{PETCO2}$ slopes so that the latter were also variable. We also found that there was a significant negative correlation of $D_{VT}/D_{PETCO2}$ slopes with $D_{Vrc,E}/D_{PETCO2}$ and $D_{Vcw,E}/D_{PETCO2}$ slopes among our subjects (Fig. 4).

It has been observed previously that the individual ventilatory response to CO2 is quite variable (16). The variability of VT response to CO2 plays a significant role because VT response generally dominates the VE response to CO2 (14). The factors that determine this

Table 2. Correlation coefficients for $D_{Vrc,E}/D_{PETCO2}$ and $D_{Vcw,E}/D_{PETCO2}$ to subjects' age, height, weight, FEV1, FEV1/FVC, and $D_{TE}/D_{PETCO2}$

<table>
<thead>
<tr>
<th></th>
<th>$D_{Vrc,E}/D_{PETCO2}$</th>
<th>$D_{Vcw,E}/D_{PETCO2}$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$r$</td>
<td>$P$</td>
</tr>
<tr>
<td>Age</td>
<td>0.338</td>
<td>0.310</td>
</tr>
<tr>
<td>Height</td>
<td>0.318</td>
<td>0.341</td>
</tr>
<tr>
<td>Weight</td>
<td>0.501</td>
<td>0.116</td>
</tr>
<tr>
<td>FEV1</td>
<td>-0.397</td>
<td>0.227</td>
</tr>
<tr>
<td>FEV1/FVC</td>
<td>-0.249</td>
<td>0.461</td>
</tr>
<tr>
<td>$D_{TE}/D_{PETCO2}$</td>
<td>0.350</td>
<td>0.291</td>
</tr>
</tbody>
</table>

Significant changes in $D_{VT}/D_{PETCO2}$ and/or $D_{VE}/D_{PETCO2}$ slope in these subjects. These results suggest that compartmental end-expiratory volume response to CO2 is not more variable than VT and/or VE response to CO2 for a given subject.

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It has been observed previously that the individual ventilatory response to CO2 is quite variable (16). The variability of VT response to CO2 plays a significant role because VT response generally dominates the VE response to CO2 (14). The factors that determine this
In the present study, we assumed that the volume changes of the two compartments of the chest wall (rib cage and abdomen) could be inferred from their respective anteroposterior displacements, as originally proposed by Konno and Mead (17). They pointed out that there are important limits to unitary behavior of rib cage and abdomen. Beyond the limits, estimation of volume from a single dimension becomes increasingly inaccurate. Thus accuracy may be insufficient in many breathing maneuvers such as during high levels of ventilation or with significant degree of chest wall distortion. Nevertheless, the method has proven to be a simple and acceptable measurement to estimate not only the compartmental but also the total chest wall volume changes during quiet breathing and during augmented breathing maneuvers when ventilation is moderately increased (11, 13, 22) as in the present study.

There are two types of chest wall distortion during increased ventilation: one is a shift of volume between the rib cage and the abdomen, and the other is the deformation of the rib cage itself with a phase lag of displacement between the transverse diameter and the anteroposterior diameter (2). The latter was not assessed in the present study but has previously been found to be small by different groups at the ventilatory level we achieved (2, 13, 22, 23). Nevertheless, this type of distortion limits the accuracy of volume estimates from measurements of a single dimension. The precision of our VT estimates was only $\pm 15\%$ of the true value at a VT $<2.5$ liters (Fig. 1). Thus our measurements are approximations, especially for $\Delta Vcw,E$ (25, 28). However, we believe that they provide useful information. The errors produced by chest wall distortions in the present study are unlikely to affect our major results and interpretations significantly because our chest wall VT measurement had a mean error of only $3.4\%$, and our results showing no significant change in mean $Vcw,E$ during $CO_2$ rebreathing (Fig. 3A) are consistent with previous observations of no significant change in $VL,E$ during $CO_2$ rebreathing (6, 15). Furthermore, our major conclusion that changes in $Vrc,E$ may determine VT response to $CO_2$ can be established without an accurate quantitative measurement of $\Delta Vrc,E/\Delta PETCO_2$ slopes: of our 11 subjects, 5 had a significant positive $\Delta Vrc,E/\Delta PETCO_2$ slope ($P < 0.05$) and 4 had a significant negative $\Delta Vrc,E/\Delta PETCO_2$ slope ($P < 0.05$); their VT/\Delta PETCO_2 slopes were 0.078 $\pm$ 0.019 and 0.131 $\pm$ 0.023 l/Torr, respectively, being significantly smaller in the former ($P = 0.0075$).

Changes in $VL,E$ response to $CO_2$ have been studied by Henke et al. (15) and Cha et al. (6). Both studies showed no consistent decrease in $VL,E$. These results seem to conflict with other studies (12, 13, 18, 27, 29) that have demonstrated that progressive recruitment of the expiratory muscles is a feature of $CO_2$-stimulated hyperpnea. Indeed, electromyographic analysis indicates that, for a given level of ventilation, expiratory muscle activity is significantly greater during $CO_2$ rebreathing than during hypoxia (26). Furthermore, during $CO_2$ rebreathing, a consistent reduction in $Vab,E$ (12, 13) or a fall in abdominal pressure with outward displacement of the abdomen at the start of inspiration has been observed (12, 18, 29). The latter demonstrates expiratory muscle recruitment during expiration and relaxation at the onset of inspiratory flow.

Expiratory muscle recruitment could fail to lower $VL,E$ if the inspiratory on-switch triggered inspiration before there was sufficient time to decrease lung volume or if there were significant expiratory flow limitation. However, we found no correlation between $\Delta TE$ and $\Delta Vrc,E$, and only two subjects were $>40$ yr old. Recent studies (1, 4) have suggested that expiratory flow limitation never occurs in normal subjects with a $Ve$ achieved in our study ($\sim 50$ l/min) during exercise. These possibilities cannot explain our data. Why then is there not a consistent fall in $Vcw,E$ during $CO_2$ rebreathing?

An obvious (but not mechanistic) explanation is that $Vrc,E$ tends to increase. For the group, we found an
insignificant positive $\Delta V_{RC,E}/\Delta P_{ETCO_2}$ slope and a significant negative $\Delta V_{AB,E}/\Delta P_{ETCO_2}$ slope, which is internally consistent with our results, in agreement with previous reports, that $CO_2$ does not induce consistent changes in $V_{L,E}$. More importantly, however, our results indicated that it was $V_{RC,E}$ that determined whether $V_{COM,E}$ or functional residual capacity increased, decreased, or remained the same in individual subjects. Furthermore, the changes in $V_{RC,E}$ also influenced the individual VT response to $CO_2$: when the $\Delta V_{RC,E}/\Delta P_{ETCO_2}$ slope was more positive, the VT response to $CO_2$ was less than when the $\Delta V_{RC,E}/\Delta P_{ETCO_2}$ slope was more negative (Fig. 4). In contrast, although $V_{AB,E}$ decreased progressively with increasing $P_{ETCO_2}$ in all subjects, this decrease did not bear any relationship to the VT response to $CO_2$.

These results are not surprising considering the importance of the rib cage during augmented breathing efforts. It has been frequently demonstrated that increasing VT during $CO_2$ rebreathing is achieved predominantly by increasing rib cage displacement (7, 19, 23, 29). Of greater relevance to our findings were the studies by Pengelly et al. (23) and Chapman et al. (7), who showed that the variation of the individual VT response to $CO_2$ is primarily determined by the VT,rc response, whereas the VT,ab response is more uniform. The data of the present study not only support these findings (Fig. 3C) but also extend these observations by demonstrating that the between-individual variation in the VT response to $CO_2$ may be at least partly dependent on the variation of changes in $V_{RC,E}$ but not in $V_{AB,E}$.

The underlying mechanism for the relatively greater variability of the individual $\Delta V_{RC,E}/\Delta P_{ETCO_2}$ slopes is unclear. We found no association of $\Delta V_{RC,E}/\Delta P_{ETCO_2}$ slope to age, body mass, or FEV$_1$ and FEV$_1$/FVC. For reasons discussed above, we do not believe that shortening of TE or expiratory flow limitation can explain our results. We postulate that a consistent negative $\Delta V_{AB,E}/\Delta P_{ETCO_2}$ slope and a more variable $\Delta V_{RC,E}/\Delta P_{ETCO_2}$ slope in our subjects probably reflect a more consistent and uniform recruitment of abdominal expiratory muscles but a variable recruitment of rib cage expiratory muscles. Vagal afferents are known to have a facilitating effect on expiratory muscle activity. Recent studies suggest that vagotomy or vagal blockade can abolish or attenuate abdominal expiratory muscle activity (3, 5, 8, 30) but have smaller inhibitory effects on rib cage expiratory muscle activity (3, 5, 8). Hence variation of rib cage expiratory muscle activity, if present among our subjects during $CO_2$ rebreathing, is unlikely to be satisfactorily explained on the basis of vagal afferent activity. Thus, although we believe that variability in rib cage expiratory muscle activity explains our data, we do not know why this variability exists.

If abdominal expiratory muscles were to contract, the increase in abdominal pressure (Pab) in the area of apposition would tend to expand the lower rib cage. However, because the abdominal muscles are attached to the rib cage and have a direct action on it, independent of changes in Pab, the situation becomes complicated. The direct action and the effect of Pab on the rib cage seem to depend on which abdominal muscles contract. De Troyer et al. (9) showed that in dogs stimulation of the rectus abdominis had an deflationary effect on the rib cage, whereas stimulation of the abdominal oblique and transversus muscles inevitably expanded the rib cage. In humans, passively increasing Pab by gastric compression or distension was shown to be inflationary to the rib cage during relaxation (10, 11). Thus recruitment of expiratory rib cage muscles may be necessary to prevent $V_{RC,E}$ from increasing. Conversely, if the inspiratory rib cage muscles were tonically recruited during expiration, as seen in asthmatic patients after airway challenge (20, 21), $V_{RC,E}$ could increase despite recruitment of expiratory muscles. Whatever the mechanisms might be, the tendency of $V_{RC,E}$ response to $CO_2$ seems to be relatively “fixed” for a given subject; although the $\Delta V_{RC,E}/\Delta P_{ETCO_2}$ slope showed some variability, the sign of the slope never changed from run to run in each of the four subjects tested (Table 3).

Finally, the group positive $\Delta V_{RC,I}/\Delta P_{ETCO_2}$ slope and the zero $\Delta V_{AB,I}/\Delta P_{ETCO_2}$ slope (Fig. 3) suggest that changes in end-inspiratory lung volume were primarily due to an increase in $V_{RC,I}$ resulting from inspiratory rib cage muscle recruitment. The negative $\Delta V_{AB,E}/\Delta P_{ETCO_2}$ slope and a zero $\Delta V_{AB,I}/\Delta P_{ETCO_2}$ slope would tend to maintain diaphragmatic contractility by maintaining or even increasing preinspiratory diaphragm fiber length and preventing excessive diaphragmatic shortening.

In summary, changes in chest wall configuration at end expiration are one of the factors that may determine individual VT responses to $CO_2$. We found substantial between-individual variability in $V_{RC,E}$ response to $CO_2$ in spite of a relatively uniform decrease in $V_{AB,E}$ when ventilation was moderately increased during $CO_2$ rebreathing. The subjects with a progressively increasing $V_{RC,E}$ had a smaller VT response to $CO_2$. These results support and extend previous studies showing that the VT response to $CO_2$ is largely dependent on the behavior of the rib cage compartment (7, 23). It is likely that this is due to variability in rib cage expiratory muscle recruitment under these experimental conditions.

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