The effect of posture on asynchrony of chest wall movement in COPD

Rita Priori,1 Andrea Aliverti,1 André L. Albuquerque,2 Marco Quaranta,1 Paul Albert,3 and Peter M. A. Calverley3

1Dipartimento di Elettronica, Informazione e Bioingegneria, Politecnico di Milano, Milano, Italy; 2Department of Medicine, Federal University of Sao Paulo, Sao Paulo, Brazil; and 3Clinical Sciences Centre, University Hospital Aintree, University of Liverpool, Liverpool, United Kingdom

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Priori R, Aliverti A, Albuquerque AL, Quaranta M, Albert P, Calverley PM. The effect of posture on asynchronous chest wall movement in COPD. J Appl Physiol 114: 1066–1075, 2013. First published February 14, 2013; doi:10.1152/japplphysiol.00414.2012.—Chronic obstructive pulmonary disease (COPD) patients often show asynchrony of the lower rib cage during spontaneous quiet breathing and exercise. We speculated that varying body position from seated to supine would influence rib cage asynchrony by changing the configuration of the respiratory muscles. Twenty-three severe COPD patients (forced expiratory volume in 1 s = 32.5 ± 7.0% predicted) and 12 healthy age-matched controls were studied. Measurements of the phase shift between upper and lower rib cage and between upper rib cage and abdomen were performed with opto-electronic plethysmography during quiet breathing in the seated and supine position. Changes in diaphragm zone of apposition were measured by ultrasounds. Control subjects showed no compartmental asynchrony, whether seated or supine. In 13 COPD patients, rib cage asynchrony was noticed in the seated posture. This asynchrony disappeared in the supine posture. In COPD, upper rib cage and abdomen were synchronous when seated, but a strong asynchrony was found in supine. The relationships between changes in diaphragm zone of apposition and volume variations of chest wall compartments supported these findings. Rib cage paradox was noticed in approximately one-half of the COPD patients while seated, but was not related to impaired diaphragm motion. In the supine posture, the rib cage paradox disappeared, suggesting that, in this posture, diaphragm mechanics improves. In conclusion, changing body position induces important differences in the chest wall behavior in COPD patients.

chronic obstructive pulmonary disease; chest wall asynchrony; body posture; diaphragm movement

IN HEALTHY SUBJECTS, THE COORDINATED action of the diaphragm and intercostal muscles expands the rib cage and abdomen (AB) synchronously during spontaneous breathing at rest. This is not always the case in patients with chronic obstructive pulmonary disease (COPD), where asynchronous movement of different chest wall compartments has been recognized for many years (10, 23, 31). Asynchrony within the rib cage and between rib cage and AB was quantified in one or two directions using magnetometers (6, 26) and, more recently, using opto-electronic plethysmography (OEP) (4). Rib cage-abdominal asynchrony is often evident during loaded breathing and is recognized as a sign of failure to wean from mechanical ventilation (28). Asynchronous movement within the rib cage of COPD patients is commonly described as being paradoxical (4). True paradoxical movement, however, involves the expansion of one compartment in the opposite direction with respect to the other and represents the extreme of asynchrony in COPD patients.

Chest wall asynchrony in COPD is associated with worse airflow obstruction (19), more severe breathlessness, and an earlier pattern of chest wall hyperinflation during exercise (4), although the reasons why and the conditions in which asynchronous movement occurs remain unclear. Gilmartin and Gibson (19) using magnetometers described three main types of abnormal chest wall movement in seated COPD patients: lateral rib cage paradox, which was the commonest abnormality, inspiratory indrawing of the lower sternum, and paradoxical inspiratory motion of the AB. Lateral rib cage paradox was generally attributed to the insertion action of the flattened diaphragm with radially orientated muscle fibers (19, 20), secondary to hyperinflation, which reduces the zone of apposition (ZoA) of the diaphragm (11). More recent data from animal studies, however, suggest that, even when the ZoA is zero, the diaphragm is unable to produce lower costal indrawing without a large fall in pleural pressure (27).

One relatively simple way to investigate whether rib cage asynchrony results from changes in diaphragm length or the unopposed action of pleural pressure swings is to observe the effect of postural change with its attendant change in lung volume and diaphragm position on chest wall asynchrony in COPD. Moving from a seated to supine position changes the action of the respiratory muscles and thoracoabdominal motion in healthy subjects (17, 30). To date, there have been no data about asynchronous chest wall movement and its relationship to the area of apposition in different postures, nor has the relationship between asynchrony within the rib cage and that between rib cage and AB been systematically evaluated in clinically stable COPD patients.

In this study, we hypothesized that, similar to healthy subjects, in COPD patients, the contribution of rib cage and AB to tidal volume (VT) changes when body position is altered and, consequently, the degree of asynchrony within the rib cage and between rib cage and AB change as well. More specifically, based on the suggestions of De Troyer and Pride (16), we anticipated that moving from a seated to supine position would decrease the action of the neck and rib cage muscles without affecting the lower rib cage, thereby increasing the asynchrony between upper rib cage and AB and reducing the asynchrony between the upper and lower rib cage compartments.

To test this idea, we measured the volumes of the pulmonary (RCP) and abdominal rib cage (RCa) (\( V_{RCP} \) and \( V_{RCa} \), respectively) and of the AB (\( V_{AB} \)) by OEP in COPD patients when seated and supine. We examined the relative timing of these compartmental volume changes in both positions, and, in a subset of subjects, we related them to the movement of the ZoA of the diaphragm to establish whether any changes in rib cage asyn-
chrony directly resulted from a change in diaphragm motion. To define the limits of normal behavior, we compared our data to that in age-matched healthy subjects, a group lacking in many previous studies.

METHODS

Subjects

Effect of posture on chest wall compartment asynchrony. We recruited stable patients with a diagnosis of COPD according to accepted criteria (12), with age <75 yr, with forced expiratory volume in 1 s (FEV1)/forced vital capacity (FVC) <0.7, and FEV1 < 50% predicted. All normal subjects were age-matched to the COPD patients (<75 yr old), had normal spirometry for their age (FEV1/FVC > 0.7), and had no significant health problems. The research protocol was approved by the local research ethics committee, and informed consent was obtained from each participant.

Protocol and Measurements

Protocol. After recording pulmonary function and lung volumes (Medgraphic: Autolin 1085D; Medical Graphics, St. Paul, MN), OEP recordings were made during spontaneous quiet breathing (QB) in seated position for repeated periods of 3 min (2–3 times). Subsequently, the subjects lay supine on a semirigid bed, and the measurements were repeated after the 5-min interval needed to reposition the OEP markers to allow accurate supine volume measurement.

Measurements. OPTO-ELECTRONIC PLETHYSMOGRAPHY. Chest wall and compartmental volumes were measured with OEP (OEP System; BTS, Milan, Italy), as described previously (2, 9). In brief, OEP measures the volumes of the chest wall (VCW) by use of retro-reflective markers (89 in seated and 52 in supine position) placed on the chest wall of the subject. Six TV cameras capture the markers’ positions at a frequency of 60 Hz, and the three-dimensional (3D) coordinates of the markers are calculated by stereo-photogrammetry, using motion analyzer system. Three reflective markers were placed on the probe so that a coordinate system relative to the probe and the absolute position of the probe could be obtained (3). The probe was held by the operator while sitting at the patient’s right side, carefully avoiding any interference with the TV camera’s field of view. Echo-graphic images of the diaphragm were recorded at a frequency of 10 Hz by means of a frame grabber (NI PCI-1410).

Data Analysis

VCW and compartmental volumes. An average respiratory cycle was calculated in each patient by normalizing the four volumetric signals, that is, the \( V_{RCp} \), \( V_{RCa} \), \( V_{AB} \), and \( V_{CW} \), with respect to time over at least three sequential breaths during spontaneous QB. From these data, the chest wall \( V_T \) and percent contribution to total chest wall \( V_T \) of the \( RCp \), \( RCa \), and \( AB \) \( \left( V_{TRCP}{\%}, V_{TRCA}{\%}, \text{and } V_{TAB}{\%}\right) \), respectively) were computed.

Phase difference and paradoxical motion. As described previously (1, 4), we constructed Lissajous figures to describe the degree of synchrony between the different chest wall compartments. The phase

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**Fig. 1.** From top to bottom: time courses of volume variations of the different compartments [pulmonary rib cage \( V_{RCp} \), abdominal rib cage \( V_{RCa} \), abdomen \( V_{AB} \), and total chest wall \( V_{CW} \)], during spontaneous breathing. Volume values are displayed in liters. Vertical dashed lines indicate end inspiration and end expiration, considering \( V_{CW} \) as a reference for timings of the breathing cycle. **A:** volume signals recorded in seated (left) and supine (right) position in a representative subject belonging to P+ group, i.e., with lower rib cage paradox in the seated position. **B:** volume signals recorded in seated (left) and supine (right) position in a representative subject belonging to P- group, i.e., without lower rib cage paradox in the seated position. Notice the unitary behavior of the upper and lower rib cage in the supine position in both P+ and P- patients.
shift ($\theta$) between two volumetric signals is calculated after Lissajous loop analysis, a graph that is created when the two volumetric signals are plotted against each other. $\theta$ is defined by the following formula: $\theta = \sin^{-1} \left( \frac{m}{s} \right)$, where $m$ represents the distance delimited by the intercepts of the dynamic loop on a line parallel to the X-axis at 50% of the Vr of the signal on the Y-axis, and $s$ represents the Vr of the signal on the X-axis (Fig. 2). In this case, a phase angle of 0° means that the movement between the compartments under study is completely synchronous, while 180° describes a movement that is completely asynchronous.

$\theta$ between RCp (y-axis) and RCA (x-axis) and $\theta$ between RCp (y-axis) and AB (x-axis) were calculated. By convention, a positive angle means that RCp expansion is leading on RCA (or AB) expansion; negative angles describe the reverse situation. $\theta$ between RCp and RCA provides information on the relative activation and coordination between inspiratory rib cage muscles and the diaphragm and the resulting rib cage distortion; $\theta$ between RCp and AB provides information on the relative activation and coordination of the inspiratory rib cage muscles and the diaphragm during inspiration and between the expiratory rib cage muscles and the expiratory abdominal muscles during expiration (4). As formerly proposed (4), inspiratory paradox time (IP) was also used to define paradoxical rib cage movement. It was defined as the fraction of the inspiratory time, relative to the total VCW signal, in which compartmental volume decreases and is expressed as a percentage.

Control data were used to define the normal ranges of $\theta$ and IP and to classify the subjects as patients with $\theta$ or without $\theta$ inspiratory paradoxical movement of the lower rib cage in seated and in supine positions. Threshold values of $\theta$ and IP were defined as two standard deviations beyond the $\theta$ and IP mean values obtained in the 12 healthy controls during QB in seated and in supine positions.

Movement at the margin of the ZoA of the diaphragm. The cephalic margin of the ZoA was identified on each echographic image as the point at which the diaphragm reflects from the chest wall and the lung intervenes (3). This point was selected automatically on each echo-

### COPD patient with paradox (P+)

**SEATED**

A: $\theta = 22.9^\circ$

B: $\theta = -6.2^\circ$

C: $\theta = -8.2^\circ$

D: $\theta = -43.4^\circ$

**SUPINE**

Fig. 2. Lissajous figures in the same representative P+ subject of Fig. 1. A: $V_{RCp}$ vs. $V_{RCA}$ Lissajous figure in the seated posture. The analysis for phase shift ($\theta$) calculation on the normalized breath is also shown. $\theta$ is calculated as arcsin (m/s) (see text). Notice the wide opening of the loop, showing asynchronies between the two compartments. The solid dot corresponds to the onset of inspiration. Arrows indicate the direction of the loop. As expected in a P+ subject, $V_{RCp}$ is strongly leading on $V_{RCA}$ as can be seen by the wide opening of the loop and $\theta = 22.9^\circ$, as indicated in the figure. B: $V_{RCp}$ vs. $V_{RCA}$ Lissajous figure in the supine posture. $V_{RCp}$ and $V_{RCA}$ are almost synchronous, although $V_{RCA}$ is slightly leading on $V_{RCp}$ ($\theta = -6.2^\circ$). C: $V_{RCp}$ vs. $V_{AB}$ Lissajous figure in the seated posture. $V_{AB}$ is leading on $V_{RCp}$ ($\theta = -8.2^\circ$). D: $V_{RCp}$ vs. $V_{AB}$ Lissajous figure in the supine posture. $V_{AB}$ is leading on $V_{RCp}$ as expected ($\theta = -43.4^\circ$).

COPD, chronic obstructive pulmonary disease; $\Delta$, change.
graphic image, using a dedicated software developed in Matlab (The Mathematics, Natick, MA). The images were processed to obtain the two-dimensional (2D) coordinates of the margin of the ZoA in the 3D image reference system. Successively, these 2D coordinates were mapped into the 3D space using the information on the position and orientation of the probe obtained by the markers placed on it (3). In this way, the absolute 3D coordinates of the margin of the ZoA were identified for each acquired frame, and the motion in the cranio-caudal direction was successively calculated and expressed as displacement in millimeters relative to the xiphoid process (3).

### Table 1. Anthropometric characteristics and spirometric values of control subjects and COPD patients

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>COPD</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Subjects, n</strong></td>
<td>12</td>
<td>23</td>
</tr>
<tr>
<td><strong>Age, yr</strong></td>
<td>65.6 ± 6.4</td>
<td>66.4 ± 6.9</td>
</tr>
<tr>
<td><strong>Height, cm</strong></td>
<td>173.5 ± 8.8</td>
<td>171.1 ± 5.1</td>
</tr>
<tr>
<td><strong>Weight, kg</strong></td>
<td>76.8 ± 8.3</td>
<td>72.3 ± 16.2</td>
</tr>
<tr>
<td><strong>BMI, kg/m²</strong></td>
<td>25.5 ± 2.0</td>
<td>24.6 ± 4.9</td>
</tr>
<tr>
<td><strong>FVC, liters</strong></td>
<td>4.1 ± 0.9</td>
<td>3.2 ± 0.7</td>
</tr>
<tr>
<td><strong>FVC, % pred</strong></td>
<td>100.9 ± 16.1</td>
<td>65.1 ± 11.8</td>
</tr>
<tr>
<td><strong>FEV₁, liters</strong></td>
<td>3.2 ± 0.7</td>
<td>32.9 ± 7.1</td>
</tr>
<tr>
<td><strong>FEV₁, % pred</strong></td>
<td>111.1 ± 16.2</td>
<td>32.9 ± 7.1</td>
</tr>
<tr>
<td><strong>FEV₁/FVC, %</strong></td>
<td>78.6 ± 7.7</td>
<td>40.6 ± 10.9</td>
</tr>
<tr>
<td><em><em>IC</em>, % pred</em>*</td>
<td>169.0 ± 29.8</td>
<td>153.0 ± 20.1</td>
</tr>
<tr>
<td><em><em>RV</em>, liters</em>*</td>
<td>5.5 ± 0.9</td>
<td>5.5 ± 0.9</td>
</tr>
<tr>
<td><em><em>RV</em>, % pred</em>*</td>
<td>223.5 ± 35.2</td>
<td>204.2 ± 21.7</td>
</tr>
<tr>
<td><em><em>TLC</em>, liters</em>*</td>
<td>8.2 ± 1.0</td>
<td>7.7 ± 0.8</td>
</tr>
<tr>
<td><em><em>TLC</em>, % pred</em>*</td>
<td>123.5 ± 14.2</td>
<td>113.0 ± 11.3</td>
</tr>
<tr>
<td>*<em>RV/TLC</em> **</td>
<td>6.6 ± 0.6</td>
<td>67.4 ± 4.4</td>
</tr>
<tr>
<td><em><em>RV/TLC</em>, % pred</em>*</td>
<td>15.8 ± 22.39</td>
<td>10.05 ± 22.39</td>
</tr>
<tr>
<td><em><em>SVC</em>, liters</em>*</td>
<td>3.1 ± 0.5</td>
<td>2.8 ± 0.3</td>
</tr>
<tr>
<td><em><em>SVC</em>, % pred</em>*</td>
<td>76.9 ± 12.9</td>
<td>69.7 ± 9.6</td>
</tr>
<tr>
<td><em><em>IC</em>, liters</em>*</td>
<td>2.2 ± 0.7</td>
<td>2.2 ± 0.7</td>
</tr>
<tr>
<td><em><em>IC</em>, % pred</em>*</td>
<td>71.4 ± 20.7</td>
<td>68.2 ± 14.1</td>
</tr>
<tr>
<td><strong>Total CV volume, liters</strong></td>
<td>26.9 ± 4.3</td>
<td>29.0 ± 7.5</td>
</tr>
<tr>
<td><strong>RCp volume, literes</strong></td>
<td>13.7 ± 1.8</td>
<td>14.1 ± 3.1</td>
</tr>
<tr>
<td><strong>RCp volume, %CV</strong></td>
<td>51.8 ± 6.7</td>
<td>49.4 ± 5.7</td>
</tr>
<tr>
<td><strong>RCa volume, liters</strong></td>
<td>4.5 ± 1.1</td>
<td>4.1 ± 3.1</td>
</tr>
<tr>
<td><strong>RCa volume, %CV</strong></td>
<td>16.5 ± 2.3</td>
<td>14.2 ± 4.43</td>
</tr>
<tr>
<td><strong>AB volume, kg</strong></td>
<td>8.7 ± 2.7</td>
<td>10.7 ± 3.8</td>
</tr>
<tr>
<td><strong>AB volume, %CV</strong></td>
<td>31.7 ± 5.8</td>
<td>36.4 ± 7.0</td>
</tr>
</tbody>
</table>

Values are means ± SD. P+ are subjects showing lower rib cage inspiratory paradox in seated position. P− are subjects without lower rib cage inspiratory paradox in seated position. COPD, chronic obstructive pulmonary disease; BMI, body mass index; FVC, forced vital capacity; FEV₁, forced expiratory volume in 1 s; TGV, thoracic gas volume; RV, residual volume; TLC, total lung capacity; SVC, slow vital capacity; IC, inspiratory capacity; CW, chest wall; RCp, pulmonary rib cage; RCa, abdominal rib cage; AB, abdomen. *Data are not available in control subjects. +P < 0.05 and −P < 0.01 for comparison of P+ with P−. **P < 0.05 and ***P < 0.01, and +P < 0.001 for comparison of P+ and −P− COPD with controls.

### Table 2. Ventilatory pattern, phase shift between pulmonary rib cage and abdominal rib cage, and phase shift between pulmonary rib cage and abdomen in controls and COPD patients in seated and supine positions

<table>
<thead>
<tr>
<th></th>
<th>Seated</th>
<th>Supine</th>
<th>Seated</th>
<th>Supine</th>
<th>Seated</th>
<th>Supine</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Vt, liters</strong></td>
<td>0.78 ± 0.55</td>
<td>0.61 ± 0.29</td>
<td>0.88 ± 0.28</td>
<td>0.85 ± 0.35</td>
<td>0.87 ± 0.22</td>
<td>0.81 ± 0.26</td>
</tr>
<tr>
<td><strong>V₂Ca, %Vt</strong></td>
<td>19.63 ± 5.89a,b</td>
<td>13.0 ± 7.20</td>
<td>10.27 ± 6.38</td>
<td>8.50 ± 5.84</td>
<td>7.29 ± 3.74</td>
<td>6.99 ± 4.40</td>
</tr>
<tr>
<td><strong>T₀</strong>, s</td>
<td>41.35 ± 17.09a,b</td>
<td>65.27 ± 11.85b</td>
<td>61.91 ± 17.77</td>
<td>65.72 ± 10.21</td>
<td>75.88 ± 19.70</td>
<td>57.37 ± 13.49</td>
</tr>
<tr>
<td><strong>Tₚ</strong>, s</td>
<td>1.67 ± 0.68</td>
<td>1.59 ± 0.53</td>
<td>1.56 ± 0.55</td>
<td>1.58 ± 0.46</td>
<td>1.65 ± 0.63</td>
<td>1.48 ± 0.40</td>
</tr>
<tr>
<td><strong>Duty cycle</strong></td>
<td>2.45 ± 0.78</td>
<td>2.64 ± 0.93</td>
<td>2.99 ± 1.21</td>
<td>3.61 ± 1.58b,c</td>
<td>3.06 ± 1.12</td>
<td>3.36 ± 1.27</td>
</tr>
<tr>
<td><strong>Vt/Tₚ</strong>, s</td>
<td>40.21 ± 5.67</td>
<td>38.7 ± 5.56</td>
<td>34.06 ± 5.67</td>
<td>32.12 ± 5.86</td>
<td>34.34 ± 6.23</td>
<td>32.15 ± 6.26</td>
</tr>
<tr>
<td><strong>Frequency, breaths/min</strong></td>
<td>16.31 ± 5.91</td>
<td>16.09 ± 6.59</td>
<td>15.28 ± 4.90</td>
<td>13.66 ± 5.19</td>
<td>14.65 ± 4.87</td>
<td>14.05 ± 4.55</td>
</tr>
<tr>
<td><strong>θ₂Cp</strong></td>
<td>−0.25 ± 0.57</td>
<td>−6.85 ± 11.04</td>
<td>23.68 ± 19.50a,d</td>
<td>5.17 ± 10.05f,g</td>
<td>34.71 ± 17.46f,g</td>
<td>4.01 ± 22.87</td>
</tr>
<tr>
<td><strong>θ₂Cp,Ab</strong></td>
<td>0.39 ± 4.77</td>
<td>10.0 ± 18.54</td>
<td>1.30 ± 13.29</td>
<td>−24.98 ± 18.19h</td>
<td>3.45 ± 16.57</td>
<td>−30.85 ± 21.36</td>
</tr>
<tr>
<td><strong>IP</strong></td>
<td>3.77 ± 4.27</td>
<td>11.31 ± 8.98b,c</td>
<td>18.50 ± 12.72a,c</td>
<td>10.76 ± 10.60</td>
<td>26.78 ± 10.05a</td>
<td>18.14 ± 8.22</td>
</tr>
</tbody>
</table>

Values are means ± SD. Vt, tidal volume; V₂Cp, pulmonary rib cage volume; V₂Ca, abdominal rib cage volume; V₂AB, abdominal volume; T₀, inspiratory time; Tₚ, expiratory time; θ₂Cp, phase shift between pulmonary rib cage and abdominal rib cage; θ₂Cp,Ab phase shift between pulmonary rib cage and abdomen; IP, inspiratory paradox time. +P < 0.05, −P < 0.01, and +P < 0.001 for comparison between seated and supine positions within COPD. +P < 0.05, −P < 0.01, and +P < 0.001 for comparison between seated and supine positions within controls. +P < 0.05, −P < 0.01, and +P < 0.001 for comparison between control subjects and COPD patients. IP < 0.05 and −IP < 0.01 for comparison between P+ and P−.

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Relationships between compartmental volume variations and cranio-caudal displacement. To compare the relationships between compartmental volume change and that in the ZoA, we constructed scatter plots using data acquired in both postures (seated and supine), in COPD and control subjects. The correlation coefficient ($r^2$) was calculated for each linear regression.

Statistical Analysis

Data are expressed as means ± SD, unless otherwise specified. Two-way repeated measures ANOVA was performed when variables were normally distributed. To study the posture effect and to assess differences between COPD patients to the control group, post hoc tests were based on Holm-Sidak method. Nonparametric two-way repeated measures ANOVA was used when normality test failed, and Holm-Sidak method was performed for multiple comparison. $P$ values <0.05 were considered as statistically significant.

RESULTS

We studied 23 severe COPD patients (FEV$_1$ = 32.9 ± 7.1% predicted) and 12 healthy age-matched controls (FEV$_1$ = 111.1 ± 16.2% predicted) whose demographic characteristics are shown in Table 1. Measurements of the change in ZoA ($\Delta$ZoA) and plethysmographic lung volume were performed in a subgroup of 11 COPD patients whose pulmonary function was comparable to the larger group (FEV$_1$ = 29.2 ± 5.6% predicted) and also in 5 controls.

Effect of Body Posture on V$_{CW}$, Asynchronous Chest Wall Movement, and Paradox

Chest wall volumes. Ventilatory data derived from OEP in seated and supine positions for COPD patients and control sub-

Fig. 3. A–C: mean percent contribution of compartmental volumes to tidal volume during quiet breathing in seated and supine positions, in both controls (open vertical bars) and COPD patients (solid vertical bars). *$P < 0.05$ and **$P < 0.01$ for comparison of COPD with controls. ***$P < 0.001$ for comparison of seated and supine postures in controls. *$P < 0.05$ and **$P < 0.01$ for comparison of seated and supine posture in COPD. D: mean chest wall tidal volume in seated and supine positions, in both controls (open vertical bars) and COPD patients (solid vertical bars). Tidal volume was affected by neither posture or presence of a pathological condition.
Projects are presented in Table 2. There was no difference in VT when comparing seated to supine in COPD patients or controls. The effect of posture on the compartmental contribution to VT for each chest wall compartment is shown in Fig. 3. When control subjects were supine, both the RCp (VT<sub>RCp</sub>%) and RCA (VT<sub>RCA</sub>%) contributions to VT decreased significantly compared with the seated values (\(P < 0.001\) and \(P < 0.01\), respectively), with a proportionate increase in the abdominal contribution to VT (VT<sub>AB</sub>%) (\(P < \).  

![Fig. 4](http://jap.physiology.org/)

A: effect of posture on the phase shift between RCp and RCA in the control group. Individual values of 12 subjects are displayed. The synchrony of the rib cage is not affected by posture in healthy subjects.  

B: effect of posture on the phase shift between RCp and AB in the control group. Individual values of 12 subjects are displayed. Note the greater variability in supine position.  

C: effect of posture on the phase shift between RCp and RCA in the COPD patients. Individual values of 23 patients are displayed. Notice that, when seated, the majority of patients show a positive phase shift, while when supine the intersubject variability is shifted toward values around 0°.  

D: effect of posture on the phase shift between RCA and AB in the COPD patients. Individual values of 23 patients are displayed. Notice that, in supine, all of the patients show a negative phase shift, suggesting that, in supine position, the AB expands earlier than the rib cage.  

E: mean phase shift between RCp and RCA in controls (open vertical bars) and in COPD patients (solid vertical bars) in seated and supine postures. *\(P < 0.05\), ***\(P < 0.001\) for comparison of COPD with control group.  

F: mean phase shift between RCp and AB in controls (open vertical bars) and in COPD patients (solid vertical bars) in seated and supine postures. **\(P < 0.01\) for comparison of COPD with control group.
In COPD patients, $V_{TRCp\%}$ decreased when supine compared with seated ($P < 0.05$), but $V_{TRCa\%}$ did not differ from the seated values. The overall contribution to $V_T$ of $V_{AB}$ was significantly greater in COPD than control subjects when seated and increased significantly when COPD patients were supine ($P < 0.01$).

**Effect of posture on chest wall compartmental asynchronies.**

See Fig. 4. There was no $\theta$ between $RCp$ and $RCa$ or $AB$ in control subjects when seated. Control subjects showed a slightly negative $\theta$ between $RCp$ and $AB$ when supine, but the difference did not reach statistical significance.

In almost all COPD subjects, there was a positive $\theta$ between $RCp$ and $RCa$ when seated, which decreased when supine ($P < 0.001$). When seated, the $\theta$ between $RCp$ and $AB$ of COPD patients was similar to that of control subjects, but, when supine, all COPD subjects showed a negative $\theta$ ($P < 0.001$). As can be seen from Fig. 1, these changes were not due to altered behavior in the $RCa$ compartment, but were due to the $RCp$ compartment behaving more like the lower rib cage. Although such effects were most evident in $P+$ patients, lack of coordination of $RCp$ and $AB$ compartments emerged when

![Graphs showing the effect of posture on chest wall compartmental asynchronies.](https://www.jappl.org)

*Fig. 5. Left: scatter plots and linear regression of $RCp$, $RCa$, and abdominal volumes vs. cranio-caudal displacement of the margin of the zone of apposition ($\Delta ZOA$) in a representative patient in seated position. Correlation coefficients are shown for each regression. Right: scatter plots and linear regression of $RCp$, $RCa$, and abdominal volumes vs. cranio-caudal displacement of the margin of the $\Delta ZOA$ in the same representative patient in supine position. Correlation coefficients are shown for each regression.*
supine, even in patients in whom coordination of the rib cage compartments was maintained when seated (P<). Of the 23 COPD patients, 13 met the criteria for paradox (with inspiratory paradoxical movement of the lower rib cage in seated position) when seated (θ = 10.28° and IP = 12.31%). Of the remaining 10, 4 showed a θ above the threshold, 2 patients had a high IP, and the remaining 4 were normal for both criteria. Of the 11 patients in whom ultrasound data and lung volumes were available, 6 were classified as being with inspiratory paradoxical movement of the lower rib cage in seated position. The criteria for paradox were revised when supine (θ = 15.23° and IP = 31.10%) to take account of the normal changes seen with posture, and, as a result, none of the 23 patients met both criteria for paradox in this position.

Movement at the ZoA of the Diaphragm

The movement of the ZoA in healthy subjects was not significantly different in the two postures (12.3 ± 6.3 mm seated and 16.3 ± 6.4 mm supine). In COPD patients, the displacement was 23.6 ± 9.3 mm seated and 17.5 ± 5.7 mm supine (P<0.05). COPD patients showed significantly greater diaphragmatic displacement seated compared with control subjects (P<0.01), but when supine the degree of displacement was similar in both groups.

In healthy subjects, compartmental volume change was always well related to diaphragm displacement, whether seated or supine. A significant correlation was also found in COPD patients, but to varying degrees in both postures, as illustrated in Fig. 5 (P<0.001). In COPD, mean r² of the regressions between VRCp and \( V_{AB} \) and ΔZoA were similar when seated, with a lower value of r² for VRCa (Table 3). When supine, the relationship between \( V_{AB} \) and ΔZoA was maintained, but the correlation of ΔZoA and VRCp worsened to values similar to those between ΔZoA and VRCa.

**DISCUSSION**

Most studies of lung mechanics, and especially of chest wall movement, consider subjects when seated or standing. However, healthy adults spend around one-third of their time lying down over the 24-h day, and COPD patients are supine for even longer (29). This is the first study to report a comprehensive analysis of chest wall movement in COPD patients in both positions.

The main result of the present study is that, in COPD patients, chest wall asynchronies are significantly influenced by body position. We found a reduction in the within-rib cage asynchrony when supine, especially in those patients showing paradox when seated. Conversely, the degree of asynchrony between RCp and AB increased from seated to supine in all COPD patients, with the AB consistently preceding the RCp in the expansion during inspiration. Interestingly, this was associated with similar ventilatory parameters in terms of VT and respiratory frequency between healthy subjects and COPD patients in both postures. COPD patients, however, showed a greater contribution of the abdominal compartment to VT at rest with a further increase when supine.

Several possible reasons for within-rib cage asynchrony have been proposed, including the direct inspiratory action on the lower rib cage of the contraction of diaphragmatic muscle fibers, which are oriented more radially in the presence of severe lung hyperinflation (18, 19, 20). As also seen in other studies (21, 24), however, we did not find any relationship between the presence of asynchrony and the degree of airflow obstruction or hyperinflation in our well-matched P+ and P− patients. Paradoxical indrawing of the lower rib cage has also been related to high mean inspiratory flow and inspiratory time (21), which are indirect indexes of respiratory drive, but also these parameters did not differ between patients’ groups, even when asynchrony was abolished in the supine position. Given the evidence that indrawing of the lower rib cage cannot be accomplished by diaphragm contraction alone (27), we believe it is likely that asynchronous movement of the lower rib cage results primarily from the fall in pleural pressure with inspiration in hyperinflated patients with a reduced ZoA when seated. Activation of the neck and accessory inspiratory rib cage muscles opposes this effect of pleural pressure change on the RCp when seated, and so the RCp and AB compartments move together. When supine, the degree of asynchrony between RCp and AB is increased, suggesting that the effects of pleural pressure swings are no longer being counteracted, as proposed by De Troyer and Pride (16) in their review of this topic. Thus VT from the abdominal compartment increases when supine, but this gain is a consequence of the decreased contribution of the RCp. Our results suggest, therefore, that asynchrony between RCp and AB cannot be explained by a failure of the diaphragm to shorten effectively. In fact, the ΔZoA decreased significantly in the supine posture, but the abdominal volume displaced (ΔVT) per centimeter of diaphragm shortening increased rather than fell. The changes of ΔVT/ΔZoA with posture are in keeping with an increase in the tension developed for a given degree of diaphragm shortening secondary to a change in diaphragm shape, as it accommodates the weight of the abdominal contents (18). Coupled with an increase in abdominal compliance when supine, a more effective diaphragm action on the AB is, therefore, likely to be present. This has consequences not only for the volume displaced, but also for the timing of the expansion of the rib cage and AB. As seen in Fig. 1 and in all of the other equivalent traces, the expansion of the abdominal compartment leads on the rib cage compartment at the onset of inspiration in the supine position, regardless of the presence or absence of rib cage asynchrony while seated.

The data from the patients in whom we measured the ΔZoA directly support the observations on chest wall asynchronies. Like Gorman et al. (21), we found no reduction in the movement of the ZoA in our P+ patients compared with those without asynchrony; thus during tidal breathing diaphragm motion is preserved in COPD, although the muscle itself is
known to be shorter at FRC than in healthy subjects. As was seen in earlier study of healthy subjects (3), ΔZoA were closely related to abdominal volume change in all conditions and in COPD patients and older controls. ΔVAp/ΔZoA was 0.25 l/cm compared with 0.23 l/cm in the control subjects, values almost identical to the volume displaced by diaphragm motion (ΔVdi) per ΔLap (ΔVdi/ΔLap) obtained using radiographic methods in seated emphysema and healthy subjects by Singh et al. (33). This similarity of results supports our view that the OEP measurements we report are equivalent to other estimates of Vdi, and that the ΔZoA is equivalent to ΔLap in the seated position. The RCP expanded in parallel with ZoA in healthy subjects, whether seated or supine, confirming the high degree of coordination between the rib cage muscles and the diaphragm. The same correlation was found in COPD patients when seated, but this relationship weakened when supine to a degree comparable with the RCa, which was invariably poorly correlated with ΔZoA. This loss of synchronization between expansion of the RCP and diaphragm shortening measured both by OEP and ultrasounds is the major effect of postural change in COPD patients.

The present study has several new features and some limitations. The effects of posture on thoracoabdominal asynchrony in COPD have been systematically studied for the first time by both OEP and ultrasounds, which provide a more complete analysis of the relationship of movement of the lower rib cage and the ZoA in COPD patients. In addition, the data obtained in COPD have been compared with a control group of age-matched healthy subjects, a group lacking in most of the existing studies. As noted, our data agree with results obtained using different measurements in similar subjects studied when seated, but, unfortunately, signal degradation when supine and increased patient discomfort prevented us from obtaining pressure signals simultaneously. We did not establish the initial Lap, but this is known to be shorter in COPD patients, and, given the qualitative agreement of our ultrasound measurements with these authors, we do not believe our patients were different in this regard (21). Although lung volumes and ZoA measurements were not available in all subjects, the subset studied was representative of the larger group. Although the relative ΔZoA in any posture is accurate, the absolute values for change in volume and diaphragm length cannot be considered, because absolute volumes obtained by OEP cannot be compared between postures.

In conclusion, our findings contribute to explain why asynchronous chest wall movement has been a difficult physical sign to assess. The absence of paradox when supine increases the risk of interobserver disagreement when relating asynchrony to clinical outcomes. However, it is now clear that important differences in the behavior of the chest wall occur when patients change position. Some patients with COPD report being less breathless when supine (32), and our data suggest that this may be physiologically based, reflecting the greater role of the diaphragm in sustaining ventilation with less energetically costly rib cage distortion (25, 34). Future studies should explore this in more detail and determine whether this position is more suitable for exercise training in those patients with more severe COPD.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS


REFERENCES