The Relationship between Lung Volume and Standard Scalar ECG Parameters in Normal Subjects*

John D. Parker, M.D.; John E. Fay, M.B.; F. James Brennan, M.D.; and Lutz Forkert, M.D.

Although the relationship between abnormalities in the ECG and lung volumes has been well described in patients with chronic obstructive lung disease, little is known about this relationship in normal subjects. We investigated this relationship in normal, supine subjects at lung volumes ranging from residual lung volume to total lung capacity. We found that there was a significant right shift in the frontal axis with increasing lung volume. Over the precordial, R wave amplitude decreased mainly at the lateral leads and S wave amplitude increased chiefly at the anterior leads. These results are consistent with a normal vertical shift and clockwise rotation of the heart with increasing lung volume. (Chest 1989; 95:530-34)

It has been known for decades that variations in lung volume have significant effects on the resting ECG. Einthoven et al. in 1913 recognized that normal ventilation affected the position of the heart and realized that such changes would have to be appreciated to make the distinction between normal and abnormal tracings. Whereas most interest concerning the effects of lung volume on the 12-lead ECG has been focused on patients with chronic lung disease, surprisingly little is known about this relationship in normal subjects. Before valid conclusions about possible relationships in patients may be made, however, it is important to have such information available on normal subjects. For this reason, we decided to investigate the effect of lung volume on changes in the ECG in normal subjects.

We chose electrocardiographic variables, which are commonly assessed in clinical practice and are often felt to change with lung disease.

METHODS

Subject Selection

The study population consisted of 20 young normal, nonsmoking men who had no history of cardiopulmonary disease. All subjects underwent standard pulmonary function testing and had a resting 12-lead ECG prior to the study. Their anthropomorphic data are shown in Table 1. Subjects whose pulmonary function tests indicated airway obstruction or restriction and those having abnormalities in their ECG were excluded from the study. This study received ethics approval and each subject signed a consent prior to participation.

*From the Department of Medicine, Queen's University, Kingston, Ontario, Canada.
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Reprint requests: Dr. Forkert, Connell 2, Kingston General Hospital, Kingston, Ontario, Canada K7L 2V7

Apparatus

The screening pulmonary function tests prior to the study were measured with conventional pulmonary function laboratory instrumentation. Absolute lung volumes were measured with a constant volume plethysmograph (model 09001, Collins, Braintree, MA) according to the method of DuBois et al. and maximal expiratory flow rates, with a dry-seal rolling spirometer (131 Spiroflow, Morgan, Rainham, Gillingham, United Kingdom). All ECGs were recorded using a 12-lead Marquette MAC II recorder (Marquette Electronics, Milwaukee, WI). The same recorder was used for all experiments. The recorder has an internal voltage calibration, which is checked each time it is turned on. External voltage calibrations have not revealed significant deviations. Experimental measurements were made with subjects in the supine position on an adjustable Ritter table. While the subjects nose was occluded with a nose clip and he breathed through a mouth piece, flow measurements were obtained from a heated pneumotachograph (Fleisch No. 3), which was connected to the mouth piece and suspended above the subjects face. The pressure difference across the pneumotachograph was measured with a differential transducer (MP 45±2 cmH2O, Validyne, Northridge, CA). Signals from the pressure transducer were amplified by a No. 9605C Beckman LDVT coupler, integrated to volume (No. 9673B Beckman integrator) and recorded on an eight-channel recorder (R-611, Beckman, Anaheim, CA). Prior to each study, the system was calibrated with known volume signals. Electrocardiography was performed by a trained technician.

Protocol

The subject was allowed to breathe comfortably through the breathing apparatus for several minutes before any measurements were made. The subject was then asked to hold his breath for 12 s at one of four lung volumes: RV, FRC, FRC +1, and TLC, during which time the ECG was recorded. Measurements for each lung volume were repeated five times with a total of 20 measurements. The sequence of these measurements was randomized for each subject. For FRC, the end-expiratory volume during normal tidal breathing was used. The subject's tidal pattern was monitored on the recorder and once the pattern was stable the end-expiratory volume was chosen. This end-expiratory volume was related to the plethysmographically determined FRC by having the subject inhale to TLC and to RV. If the inspiratory capacity and the expiratory reserve volume were within 100 ml of the same volumes measured plethysmographically the end-expiratory volume was accepted as
Table 1—Vital Statistics and Pulmonary Function for Electrocardiography Subjects (n = 20)

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>(±) SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>28.00</td>
<td>4.38</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>75.45</td>
<td>8.96</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>178.60</td>
<td>6.83</td>
</tr>
<tr>
<td>FVC (% predicted)</td>
<td>103.34</td>
<td>9.44</td>
</tr>
<tr>
<td>FEV₁ (% predicted)</td>
<td>109.06</td>
<td>9.02</td>
</tr>
<tr>
<td>FEF₂₅-₇₅% (% predicted)</td>
<td>111.78</td>
<td>18.81</td>
</tr>
<tr>
<td>TLC (% predicted)</td>
<td>90.27</td>
<td>15.45</td>
</tr>
<tr>
<td>RV (% predicted)</td>
<td>79.90</td>
<td>26.71</td>
</tr>
</tbody>
</table>

an index of FRC. Once FRC was determined, for TLC the subject inspired the equivalent of his inspiratory capacity, for RV he expired the equivalent of his expiratory reserve and for FRC + 1 he inspired 1 L. Once these volumes were attained, the subject held his breath while the ECG was recorded. At the end of the breath holding the subject was allowed to breathe normally. If he did not return to his previous end-expiratory volume the previous measurement was discarded. Similarly, if the volume during the breath holding did not remain constant, the run was discarded.

Data Analysis

The ECGs were read by one of the investigators (Dr. Parker). Frontal plane axis was determined according to the algorithm, used by the microprocessor in the Marquette Mac II. The computer analysis program looks at leads 1 and aVF. It identifies the maximal upward deflection and the maximal downward deflection. Once these are determined, the algebraic sums of the summits and the nadirs are subtracted from each other. The results are plotted (lead 1 plotted against lead aVF) and the mean force is identified as the axis. The R and S wave amplitude, as well as P wave width and amplitude, were measured manually with calipers on the ECG tracings in V₁, V₃, and V₆. This investigator was blinded with respect to the volume at which the ECG recordings were made. A Compaq microcomputer and a statistical package (Systat 3, Systat Inc., 1985) were used for statistical analysis. The ECG data were analyzed with the Wilcoxon signed rank-tests for significant differences between different lung volumes. A linear correlation by the least squares method was used to determine the relationships between lung volumes and ECG variables.

Reproducibility of the electrocardiographic measurements was examined by randomly selecting one tracing from each subject and re-reading it, again in a blinded fashion. The data from this second reading were compared with the original reading for differences with a paired t test.

RESULTS

All subjects had normal baseline ECGs. They had normal pulmonary function test results and were of average height and weight (Table 1).

Representative data from one subject are shown in Figure 1. With increasing lung volume the amplitude

![Figure 1](image1)

**Figure 1.** Representative electrocardiographic tracings from one subject, recorded at RV (1.38 L), FRC (3.72 L), FRC + 1 (4.72) and TLC (7.41 L). Tracings for leads 1, 2, aVF, V₁, V₃, and V₆ are shown. Calibration: vertical, 1 mv; horizontal, 200 ms.

![Figure 2](image2)

**Figure 2.** The relationship between frontal axis and lung volume. The lung volumes, at which the frontal axis was measured, are shown on the abscissa. RV represents a mean absolute lung volume of: 1.49 L; FRC: 3.59 L; FRC + 1 L: 4.59 L, and TLC: 7.24 L. The frontal axis in degrees is shown on the ordinate. Mean values of the frontal axis are presented. Standard error bars of the mean frontal axis values are shown. The symbols shown above the error bars represent significant difference (p<0.05) in the frontal axis at a given lung volume compared with FRC + 1 (o), at FRC (+), and at RV (#).
of the R wave progressively decreased in lead 1 and increased in lead aVF, indicating an increase in frontal axis. Although in V₁, R wave amplitude did not change systematically, in V₃ and in V₆ the amplitude progressively decreased. The S wave amplitude increased with lung volume in V₁ and in V₃, but showed little change in V₆. The P wave amplitude and width in lead 2 did not change with lung volume. Mean results for all subjects are shown in Figures 2 to 4.

Figure 2 demonstrates changes in frontal axis which occurred with changes in lung volume. With increasing lung volume the axis shifted progressively to the right. The frontal plane axis at any of the four different lung volumes was significantly different from that found in the remaining volumes. This trend is further supported by the moderate correlation between absolute lung volume and frontal plane axis (Table 2).

Figure 3 demonstrates the change in R wave amplitude in V₁, V₃ and V₆ with lung volumes. Although at TLC the R wave in V₁ tended to be significantly smaller than at lower lung volumes, these changes were very small. In V₃ the R wave at TLC also was significantly smaller than at lower lung volumes. In V₆ these changes in the R wave were even more pronounced.

Table 2—Regression Coefficients and Correlations between Absolute Lung Volume and ECG Variables*

<table>
<thead>
<tr>
<th>ECG Variables</th>
<th>a</th>
<th>b</th>
<th>r</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frontal axis</td>
<td>46.77</td>
<td>4.55</td>
<td>0.461</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>R wave amplitude</td>
<td>3.36</td>
<td>-0.050</td>
<td>0.064</td>
<td>0.571</td>
</tr>
<tr>
<td>V₁</td>
<td>11.24</td>
<td>-0.050</td>
<td>0.324</td>
<td>0.003</td>
</tr>
<tr>
<td>R wave amplitude</td>
<td>14.47</td>
<td>-1.11</td>
<td>0.760</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>V₁</td>
<td>7.55</td>
<td>0.212</td>
<td>0.120</td>
<td>0.288</td>
</tr>
<tr>
<td>S wave amplitude</td>
<td>7.25</td>
<td>0.434</td>
<td>0.229</td>
<td>0.041</td>
</tr>
<tr>
<td>V₃</td>
<td>1.10</td>
<td>0.02</td>
<td>0.065</td>
<td>0.568</td>
</tr>
</tbody>
</table>

*a = intercept; b = slope of volume vs ECG variable (independent and dependent variable respectively); r = correlation coefficient.
nounced. Correlation coefficients supported these findings with a weak correlation between the R wave in V1 and V6 and lung volume but a strong correlation between these parameters in V6 (Table 2).

Figure 4 gives the same data for the S wave in leads V1, V3, and V6. The S wave in V1 increased progressively with lung volume. A similar trend was present for V3. The changes in V6 were small in amplitude, although there was a trend toward increasing S wave amplitude with increasing lung volume. Correlation coefficients for S wave voltage in precordial leads and lung volumes were poor (Table 2).

The P wave amplitude and width showed mean decreases of 0.2 and 0.3 mm, respectively, as lung volume increased from RV to TLC. These changes are too small to have any practical significance.

We tested the reproducibility of the measurement of the ECG variables by comparing randomly selected tracings with the previously measured tracings with a paired t test. The measurements were not different from each other (p = 0.93).

DISCUSSION

There is surprisingly little information about the effects of lung volume on the resting ECG in normal subjects. Instead, previous investigations have focused on the effects of ventilation on the ECG. Einthoven et al1 first described changes during ventilation in the frontal plane axis and alterations in R, S and P wave voltage, along with changes in the morphology of those complexes. Subsequent studies by Woodruff and Joliet3 and Lamb4 also assessed the effects of ventilation. Dougherty4 observed changes in the frontal plane QRS axis in normal subjects when the ECG was recorded in two positions—standing at deep inspiration and supine after quiet expiration. The additional effects of posture complicated this study and the effects of lung volume were not quantitated.

The present study was not designed to elucidate the mechanisms which underlie the changes in the ECG that occur with changes in lung volume. We speculate, however, that the changes in frontal plane axis are most readily explained by changes in the relative position of the heart and chest walls. Thoracic expansion and diaphragmatic descent, which accompany increasing lung volume, would lead to a more vertical position of the heart and an accompanying shift in axis. Other factors, such as differences in autonomic tone, alterations in right ventricular volume, etc, also might contribute to these changes. The fact that lung volume has no effect on P wave characteristics in lead 2 is of interest, but the reason for this is unclear. It is unlikely that the reduction in R wave voltage in V6 is due to increasing distance between the myocardium and the V6 precordial electrode, since the S wave in V6 tended not to change or actually increased in amplitude as lung volume increased. It is more likely that the rotation of the heart about its longitudinal axis is responsible for these changes. This may lead to the movement of the left ventricle posteriorly, away from the lateral chest leads, while the movement of the right ventricle is into a position closer to these leads. In short, increasing lung volumes may lead to a degree of clockwise rotation.

Standard textbooks of electrocardiography list a variety of changes in the ECG associated with both chronic obstructive and chronic interstitial lung disease.6,7 Chronic obstructive disease, in which lung volumes may be abnormally increased, has received most of the attention. Changes in frontal plane axis, P wave axis, P wave morphology, R and S wave voltage, QRS morphology and T wave configuration all have been demonstrated in chronic obstructive disease.6,12 A relationship between these changes and the degree of airway obstruction also has been noted.13,14 Generally, these changes have not been found to be either sensitive or specific as indicators of the presence of chronic obstructive lung disease.

Our investigation may offer some insight into the changes in the ECG seen in chronic obstructive lung disease. The frontal plane axis does shift to the right in some patients with chronic obstructive lung disease. Authors have attributed this to both changes in the anatomic position of the heart and changes in right ventricular function secondary to elevated pulmonary artery pressure.6,7,11 The finding of a strong correlation between lung volume and frontal plane axis in normal subjects suggests that the increased lung volume, accompanying chronic obstructive lung disease, may play an important role in the axis shift seen in this disorder. Increases in P wave voltage in the inferior leads are seen in the minority of patients with chronic obstructive lung disease and are thought to result from right atrial enlargement secondary to pulmonary hypertension.6,7 The finding that in normal subjects increasing lung volumes does not cause changes in P wave amplitude suggests that in chronic obstructive lung disease this change may be independent of the accompanying change in lung volume. Rightward shift of the P wave axis has been found to be a much more common finding in chronic obstructive lung disease. This might be secondary to changes in lung volume and accompanying alterations in the relative position of the heart and the chest wall. We elected not to assess changes in P wave axis in normal subjects, since this is rarely applied in clinical practice. Alterations in precordial R and S wave voltage commonly are seen in chronic obstructive lung disease and the finding of clockwise rotation often leads to the suspicion of chronic obstructive lung disease.6,11 Our findings suggest that these changes may well be secondary to increases in lung volume which accompany obstructive
lung disease.

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