Electrocardiographic Study of Chronic Pulmonary Emphysema by Means of Body Surface Mapping

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Summary

In order to investigate the electrocardiographic changes in patients with chronic pulmonary emphysema (CPE), 87 unipolar electrocardiograms were simultaneously recorded in 20 normal subjects and 22 patients with CPE. The voltages and the sites of Max. R (most positive R) and Max. S (most negative S) in these 87 leads were investigated. Further, the voltages and the sites of the maximum and the minimum on isopotential maps during the QRS period were examined.

In patients with CPE, the sites of Max. R, Max. S, the maximum and the minimum were all positioned lower than in normal subjects. However, the voltages of Max. R and Max. S in CPE were not significantly different from those in normal subjects. It was considered that the most prominent change in the body surface maps in CPE was the downward shift of potential distribution. This change seems to be due mainly to the downward displacement of the diaphragm and the heart resulting from the overinflation of the lung.

Additional Indexing Words:
Downward shift in potential distribution  Downward displacement of the diaphragm and the heart  Overinflation of the lung

Many investigators have reported on the electrocardiographic changes in patients with chronic pulmonary emphysema (CPE) by means of the standard 12-lead electrocardiogram or the vectorcardiogram.1)-18) However, few studies have been made evaluating these changes by use of body surface mapping.19)-21)
In the study of the electrocardiographic changes in CPE patients, three major factors must be taken into consideration:22,23 (1) downward displacement of the diaphragm, (2) distorted intrathoracic potential field caused by increased electrical resistance from the heart to the body surface across the overinflated lung, and (3) altered cardiac generator due to the hemodynamic overload on the right ventricle secondary to the pulmonary disorders. These factors affect the electrocardiographical phenomena over the entire thoracic surface to various degrees.

In body surface mapping, electrocardiographic signals are recorded from many lead points spreading over the anterior chest and the back. In this way we can obtain information about the upper and lower anterior chest and the back which are hard to obtain using the conventional electrocardiogram or the vectorcardiogram. Therefore, body surface mapping is useful for analyzing the electrocardiographic changes in such diseases as CPE, because these changes are supposed to spread extensively over the thoracic surface.

We performed body surface mapping on patients with CPE who showed no evidence of manifest cor pulmonale and analyzed the electrical potential distribution on the body surface during the QRS period.

**Materials and Methods**

*Subjects*

The subjects of this study were 22 patients with chronic pulmonary emphysema (group CPE) and 20 normal subjects (group N).

Group CPE consisted of 20 males and 2 females, aged from 51 to 75 years (mean 63.1 years). The diagnosis of CPE was based on clinical symptoms, history, physical examinations, chest roentgenogram and spirometric study.24 In the spirometric study, forced vital capacity (FVC) and forced expiratory volume in one second (FEV$_{1.0}$) were measured by a hot wire flow meter, Auto Spirometer (Minato Medical Science Co).25 Cases with ischemic heart disease, cardiac valvular disease, myocardial disease, congenital heart disease or other heart disease were excluded. Cases in whom the electrocardiogram indicated right ventricular hypertrophy (RVH), incomplete right bundle branch block or a QRS duration longer than 0.12 sec were also excluded. RVH was considered to be present when two or more of the following criteria were met: (1) R/S ratio in lead V$_1$ greater than 1, (2) R/S ratio in lead V$_5$ or V$_6$ less than 1, (3) ST segment depression and T wave inversion in leads V$_{1,2}$, (4) delayed onset of intrinsicoid deflection in lead V$_1$ (greater than 0.04 sec).26

The results of the spirometric measurements in group CPE are listed in
Table I. Clinical Characteristics of the Subjects

<table>
<thead>
<tr>
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<th>Group CPE</th>
<th>Group N</th>
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<tbody>
<tr>
<td>Number</td>
<td>22</td>
<td>20</td>
</tr>
<tr>
<td>(male/female)</td>
<td>(20/2)</td>
<td>(20/0)</td>
</tr>
<tr>
<td>Age (years) range</td>
<td>51-72</td>
<td>35-51</td>
</tr>
<tr>
<td>mean</td>
<td>63.1</td>
<td>40.5</td>
</tr>
<tr>
<td>FVC (l)</td>
<td>1.96±0.68*</td>
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<tr>
<td>FEV₁,0 (l)</td>
<td>0.76±0.27*</td>
<td></td>
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<tr>
<td>FEV₁,0% (%)</td>
<td>38.1±7.03*</td>
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</table>

* mean±SD

Table I. All subjects in group CPE showed typical obstructive changes on spirometry. On the chest roentgenogram, manifest downward displacement of the diaphragm and overinflation of the lung were observed in all members of this group.

Group N consisted of 20 males, aged from 35 to 51 years (mean 40.5 years), who clinically showed no evidence of cardiac or pulmonary diseases. Those with abnormal electrocardiographic findings were excluded.

Informed consent was obtained from all subjects before the study.

Map recording and processing

Body surface mapping was performed using a body surface potential mapping system, HPM-5100 unit (Chunichi Denshi Co) within a week after the spirometric examinations. Since the procedure for data sampling and processing has been described in detail elsewhere, it will be reviewed briefly. Eighty-seven unipolar electrocardiograms (ECGs) distributed over the entire thoracic surface with Wilson's central terminal as a reference (Fig. 1), standard 12-lead ECGs and Frank X, Y, Z ECGs were sampled simultaneously. Then, the stored signals of each ECG were displayed on a graphic terminal (TEKTRONIX 4006-1). If noise was detected in any of the signals, data sampling was repeated. The flat portion of the PQ segment was chosen as the baseline. After baseline adjustment, the data were recorded in digital format on a magnetic cassette tape. This system has a resolution of 10 μV in the dynamic range ±5mV, with a sampling rate of 1000 samples/sec/channel. The data sampling was done at the resting expiratory level with the subject in the supine position.

Data analysis

In the 87 leads (map leads), the voltages and the sites of the tallest R
Fig. 1. Electrode sites on the body surface in mapping. Eighty-seven lead points were arranged lattice like ($13 \times 7$ matrix), except for 4 lead points in both midaxillary regions, and covered the entire thoracic surface. Columns A, E and I were positioned in the right midaxillary, midsternal and left midaxillary lines, respectively. Columns B-D and F-H were evenly spaced between columns A-E and E-I, respectively. Column J was located so as to make the distance between columns I and J equal to that between columns H and I. Similarly, column M was located. Columns K and L were evenly spaced between columns J and M. Lead points $E_6$ and $E_4$ were located on the 2nd and 5th intercostal space, respectively. Row 5 was located in the center of rows 6 and 4. Row 7 and 3-1 were located so as to make the distance between adjacent rows equal.

(Max. R) and the most negative S (Max. S) were investigated. Similarly, the voltages of Max. R and Max. S were also examined in leads $V_1-V_6$.

Next, isopotential maps were constructed from data recorded at 10, 20, 30, 40, 50 and 60 msec after the onset of the QRS ($QRS_{10}$, $QRS_{20}$, $QRS_{30}$, $QRS_{40}$, $QRS_{50}$ and $QRS_{60}$). The voltages and the sites of the maximum (max) and the minimum (min) were determined on each map. For the purpose of this study, when multiple maxima (or minima) were observed on a map, the most positive one (or the most negative one) was defined as the max (or min). The time of QRS onset was determined from the superimposed Frank X, Y and Z ECGs.

Statistical comparisons were done by the unpaired $t$-test; $p<0.05$ was considered significant. Quantitative data were expressed as mean±SD.
RESULTS

Max. R and Max. S

Fig. 2 depicts the voltages of Max. R in leads $V_1$–$V_6$ (left panel) and in Max. R

![Graph of Max. R](image)

Fig. 2. The voltages of Max. R in leads $V_1$–$V_6$ and in the map leads. Open circles indicate the voltages of group N and closed circles group CPE. Bars indicate the standard deviations. Numbers in parentheses represent the number of the subjects. N = group N; CPE = group CPE.

Max. S

![Graph of Max. S](image)

Fig. 3. The voltages of Max. S in lead $V_1$–$V_6$ and in map leads. Abbreviations are as in Fig. 2.
the map leads (right panel). The mean value (±SD) was 2.02±0.46 mV for
group N and 1.08±0.52 mV for group CPE in leads V₁-V₆. In the map
leads, it was 2.04±0.41 mV for group N and 1.73±0.54 mV for group CPE.
In leads V₁-V₆, group CPE had a significantly smaller Max. R as compared
with group N (p<0.001). In contrast, although the tendency of diminished
Max. R in group CPE still existed, the difference was not statistically signifi-
cant in the map leads.

Fig. 3 represents the voltages of Max. S in leads V₁-V₆ (left panel) and
in the map leads (right panel). The mean value (±SD) was -1.84±0.54 mV
for group N and -1.66±0.58 mV for group CPE in leads V₁-V₆, and -1.87
±0.45 mV for group N and -2.07±0.63 mV for group CPE in the map
leads. The difference between the 2 groups was not statistically significant
either in leads V₁-V₆ or in the map leads. However the value of group CPE
had a tendency to increase (become more negative) in the map leads, although
it had a tendency to decrease (become less negative) in leads V₁-V₆.

The distribution of the sites of Max. R and Max. S on the body
surface is shown in Fig. 4. In group N, Max. R (left upper panel) was mostly
located around G₄, which corresponds to V₄ in standard 12-lead ECG. Max.
S (left lower panel) was, in most cases located on E₉ or F₅, which are near

Fig. 4. The distribution of the sites of Max. R and Max. S on the body
surface in each group. In this figure only the anterior portion of the map
leads is shown. The numbers on the map represent the number of subjects
whose Max. R or Max. S is located on the corresponding lead point. See
text for further explanations.
Fig. 5. The mean sites of Max. R and Max. S in group N (open circles) and in group CPE (closed circles). Bars indicate the standard deviations. The sites of leads V1–V6 are plotted on the figure.

lead V2. But in group CPE, the sites of Max. R (right upper panel) and Max. S (right lower panel) were shifted downward by 1 or 2 rows compared with those in group N.

Fig. 5 demonstrates the mean sites of Max. R and Max. S in each group. In group N, the mean sites of Max. R and Max. S were found near leads V4 and V2, respectively. However, in group CPE, the mean sites were both located about 1.5 rows (about 2 intercostal spaces) lower than in group N. These differences were significant (p<0.001, p<0.001). Lateral displacement of Max. R and Max. S was not observed in patients with CPE.

Max and min on isopotential maps

The mean sites of max and min at QRS10, 20, 30, 40, 50 and 60 in each group are shown in Fig. 6. In group N, max was located initially on the middle anterior chest, moved to the left and slightly downward thereafter and eventually reached the back. Min was located on the back at the onset of the QRS, moved to the right upper chest and finally settled on the central sternal region during the latter half of the QRS. In group CPE, although the movement was almost the same as in group N, both max and min were located lower than in group N throughout the QRS period. The downward displacements were statistically significant for max at QRS10, 20, 30, 40 and for min at QRS30, 40, 50, 60.

The mean voltages of max and min in each group are listed in Table II. In group CPE, max at QRS10–60 tended to decrease and min at QRS20–
40 tended to increase in voltage as compared with those in group N. However, differences between the 2 groups were not statistically significant, except for max at QRS40 (p<0.05).

Fig. 6. The mean sites of max (upper panel) and min (lower panel) at QRS10, 20, 30, 40, 50 and 60. Open circles represent those in group N and closed circles group CPE. Left half of the map represents the anterior chest, and right half the back. The numbers in the figure indicate the time after the onset of the QRS in msec. Asterisks indicate that the downward displacement was statistically significant.

Table II. The Voltages of the Maximum and the Minimum

<table>
<thead>
<tr>
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<th>Time after the onset of QRS (msec)</th>
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<tbody>
<tr>
<td></td>
<td>10</td>
</tr>
<tr>
<td>Maximum</td>
<td></td>
</tr>
<tr>
<td>N</td>
<td>0.39±0.15</td>
</tr>
<tr>
<td>CPE</td>
<td>0.27±0.13</td>
</tr>
<tr>
<td>Minimum</td>
<td></td>
</tr>
<tr>
<td>N</td>
<td>−0.10±0.03</td>
</tr>
<tr>
<td>CPE</td>
<td>−0.08±0.04</td>
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</tbody>
</table>

Values are expressed as mean±SD. * p<0.05 as compared to group N. Abbreviations: N=group N; CPE=group CPE.
The purpose of this study was to evaluate the distribution of the cardiac potential on the body surface in patients with CPE without manifest evidence of right ventricular hypertrophy on ECG or chest roentgenogram.

The results of our study indicated that the body surface potential distribution of the CPE patients during the QRS period was characterized by a downward shifted pattern. As was shown in Fig. 5, Max. R and Max. S in normal subjects were located near V4 and V2, respectively, while in patients with CPE these were shifted downward by about two intercostal spaces. Furthermore, the downward shift of the potential distribution was observed throughout the QRS complex, as indicated by the downward displacement of max and min in isopotential maps (Fig. 6).

It is assumed that during the ventricular depolarization the electrical wavefronts move through the various parts of the myocardium. Current is projected outward from the source side of the generator and reenters the sink side. Therefore, on the isopotential maps, the maximum occurs in the area where the greatest density of current is flowing from the source side of the cardiac generator. The minimum occurs where the greatest density of current is reentering the sink side of the cardiac generator. It was thought that the shift of max and min in our study indicated a geometric change of the cardiac generator. Considering the results of the chest roentgenogram, which revealed overinflation of the lungs and downward displacement of the diaphragm and the heart, the shifts of Max. R, Max. S, max and min in CPE were concluded to be the result of the lowered cardiac position.

Few reports have been made of the changes in the body surface maps in patients with chronic lung disease. Flaherty et al performed body surface mapping in children with cystic fibrosis, a congenital disease which results in marked overinflation and diffuse interstitial fibrosis of the lung. They observed an inferior shift of potential maxima and minima during the QRS complex and concluded that this change was due to the effect of the overinflated lung. This finding was very similar to ours. CPE and cystic fibrosis are different in many aspects but similar in that they both cause emphysematous changes in the lung and a downward displacement of the diaphragm. Consequently, it was not surprising that a similar characteristic, i.e., the downward shift of the potential distribution was observed in both diseases.

Matsushita et al performed body surface mapping in patients with various chronic pulmonary diseases. They found that the points of initial R and maximum R were located relatively lower than those of normal subjects. This finding coincides with our CPE patients. However, their subjects in-
cluded not only patients with obstructive, but also those with restrictive lung diseases. Therefore it was difficult to determine the cause of the changes in the body surface maps in their study.

The voltage of Max. R in leads V1-V6 in the CPE group significantly decreased in our study and this finding corresponded to the change in ECG called "the tendency of low voltages in V4-V6". But in the map leads, the voltage was not significantly different between the 2 groups. This discordance was considered to be due to the shift of Max. R in CPE. As was mentioned before, the sites of Max. R were located lower than in the conventional precordial leads in patients with CPE, so the recorded R waves of V4-V6 were apparently diminished.

In patients with CPE, the tendency of low voltage or decreased R waves in leads V4-V6 has been observed by many investigators. Two major factors were considered as causes of this change: (1) downward displacement of the diaphragm, (2) decreased electrical conductivity from the heart to the body surface caused by the over-inflated lung. Toyama et al showed that experimentally induced overinflation of the lung could produce a decreased R in lead X in dogs. They concluded that the increased electrical resistance across the lung may be a major cause of the change in CPE. If the effect of the decreased conductivity was large, the body surface potential on the left precordium would be expected to decrease. However in our clinical study, the potential did not always decrease in CPE patients. Thus the downward displacement of the diaphragm was thought to be the major cause of the diminished R in leads V4-V6. This conclusion coincides with that of Wasserburger et al. They showed that by replacement of the unipolar chest leads several intercostal spaces lower than the conventional precordial leads, normal voltage left ventricular potential could be registered.

On the other hand, in the present study changes other than the downward shift in potential distribution were found: i.e., the decreased voltage of max at QRS40 and the tendency of decreased Max. R, increased Max. S, decreased max at QRS10, 20, 30, 50 and 60, and increased min at QRS 20-40. These changes may have resulted from the downward displacement of the diaphragm and the rotation of the heart. However, other factors such as decreased electrical conductivity or right ventricular overloading may have contributed in part to these changes.

In experimentally induced overinflation of the lung, decreased R in lead X and increased S (posterior deflection) in lead Z were observed. Therefore decreased electrical conductivity may play a part in the changes in clinical cases. Furthermore it is well known that right ventricular hypertro-
Body surface maps in CPE

Phy is sometimes found during autopsies of CPE patients without evidence of RVH on ECG or chest roentgenogram. The clockwise rotation of the QRS loop on the VCG or decreased R/S ratio in V5 and V6 were reported as indicative of pulmonary artery hypertension or RVH. The above observed changes of Max. R and Max. S may be partially caused by pulmonary artery hypertension and right ventricular overloading. A more detailed examination of these factors must be carried out in the future.

Because the mean age of group CPE was older than that of group N in the present study, a precise comparison between the 2 groups may be difficult. But it has been reported that the voltages of R and S waves in the precordial leads are not significantly different between middle aged and elderly Japanese subjects. Therefore, we feel that the group difference in age was not a great problem in this study.

In conclusion, the most prominent change on the body surface maps in patients with CPE was a downward shift of potential distribution resulting from the downward displacement of the diaphragm and the heart and this was probably the major cause of the decreased R observed in leads V4–V6.

Acknowledgment

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References

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