Sequential Change in the Difference of Potential Distribution between a Normal Subject and Simulated Torso Model

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Summary

Isopotential map was obtained every 3 msec after the onset of ventricular activation from 85 unipolar lead ECGs of a normal subject (Measured map) and similar map at the corresponding instant (Simulated map) was also obtained by means of mathematical calculation under the assumption that the cardiac electromotive force can be represented by a single electric dipole fixed at the heart center. These 2 maps were quantitatively compared and difference was delineated on a map (Difference map).

Although, in major aspect, there was a fairly good agreement between Measured and Simulated maps during early stage of ventricular activation, a remarkable difference of potential distribution began to appear between them around the time of occurrence of epicardial breakthrough of the ventricular activation front.

From that time on, it became impossible to represent the cardiac electromotive force by a single electric dipole fixed to the anatomical heart center and there appeared a map pattern suggestive of the separation of electromotive force into 2 or more electric dipoles in Difference map.

Difference maps around the instant of epicardial breakthrough of the ventricular activation were supposed to be helpful for the estimation of the spread of ventricular activation.

Additional Indexing Words:

Measured map Simulated map Difference map Maximum Minimum Saddle Epicardial breakthrough Lead vector Single fixed-location dipole

IN 1955 a single fixed-location dipole hypothesis was advanced by Frank\(^1\).\(^2\) in the field of electrocardiography and vectorcardiography based on various findings in his experiments performed on a human body and his torso model. According to him, this hypothesis was correct in approximately 95% of the QRS pattern at any point of body surface.\(^3\) Schmitt\(^4\)–\(^6\) also supported the validity of this hypothesis on both cardiac patients and normal subjects from his
experimental data on cancellation method.

On the contrary, McFee,\textsuperscript{7,8} basing on the data in mathematical study stated that it was impossible to represent the cardiac electromotive force by a single electric dipole fixed at a certain point and that there was local information, or proximity potential, in precordial or esophageal lead ECG which could not be extracted from vectorcardiogram. Some other researchers\textsuperscript{9-11} recognized a potential distribution with multiple "maxima" and/or "minima" and sometimes "saddle" distribution on body surface isopotential maps, and suggested that these potential distributions could not be explained by the single fixed-location dipole hypothesis. There have been also many reports\textsuperscript{12-14} which the appearance of these phenomena was recognized in. The stage and area when and where those phenomena were observed were different depending upon the nature of basic heart diseases, so they could present a valuable clue for the clinical diagnosis of those heart diseases.\textsuperscript{15-17}

Recently, much effort has been done to obtain the informations which can not be explained by the single fixed-location dipole hypothesis with the aid of mathematical technique, and multipolar components such as quadripole, octapole, and so on have been analytically obtained as a source of cardiac electromotive force.\textsuperscript{18,19}

In the present paper, the author tried quantitative comparison of the isopotential map obtained from numerous unipolar thoracic lead ECGs of a normal subject and the one obtained by mathematical potential calculation in order to check the validity of the single fixed-location dipole hypothesis. It was also tried to discuss the causes of the appearance of such a difference between these 2 maps from a point of view of ventricular activation process reported by Durrer et al.\textsuperscript{20}

**Methods**

To observe the changes in body surface potential distribution, 3 kinds of maps mentioned below were obtained from the data on unipolar lead ECGs and those on torso model experiments.

1) Delineation of body-surface isopotential maps from the recorded unipolar thoracic lead ECGs

Eighty-five lead points were selected on the thoracic surface of a Japanese youth, aged 26, without any heart disease, with body length of 175 cm, body weight of 68 Kg, thoracic circumference of 95 cm, thoracic thickness of 23 cm, and thoracic width of 34 cm. Those 85 lead points selected on the thoracic surface showed a grid-like distribution (Fig. 1). The longitudinal distance between each 2 adjacent lead points was about 4.5 cm, while the circumferential distance between them was about 5.5 cm at the anterior and lateral aspects of the thorax and 12.5 cm at the dorsal aspect.

Each 2 unipolar lead ECGs were recorded by means of Wilson central ter-
Fig. 1. The 85 lead points for the unipolar lead ECGs which were recorded to obtain the body surface isopotential maps.

Finally, we obtained the body surface isopotential maps by using the above methods. The procedures described above were repeated twice on the same subject keeping a long interval between those 2 procedures in order to check the reliability of the map pattern obtained.

2) Delineation of torso model surface isopotential map by mathematical potential calculation

In order to obtain isopotential maps by mathematical calculation, a hollow acrylic resin torso model was molded after the same living subject. The thickness of the wall of the resin model was 3 mm. Silver electrodes, 1.5 mm in diameter, were planted through the wall of the torso model to simulate the location of the 85 lead points, as well as 7 lead points for Frank lead system, of the original living subject.

The heart center of the living subject was determined from the cardiac shadow in the transverse axial X-ray tomogram of the subject at the level of 5th intercostal space by the method as follows. The center line inbetween the 2 sagittal lines drawn at the left and right margins of the heart shadow and the one inbetween the 2 transverse lines drawn at the anterior and posterior margins of the heart shadow were drawn on the film. Then, cross point of these 2 center lines was obtained and regarded as the anatomical heart center. The heart center thus obtained was estimated to be 6.9 cm backward from the foremost inner surface of the torso model.
in the sagittal direction and 13.9 cm rightward from the leftmost inner surface of the model in the transverse direction at the level of the 5th intercostal space.

After a unit of electric dipole model, equipped with 3 pairs of artificial dipoles oriented perpendicularly each other (diameter of polar electrode; 1.2 cm and interpolar distance; 1.5 cm), was settled to the anatomical heart center in the torso model and adjusted so that the 3 dipole axes coincided with the 3 standard axes, transverse, longitudinal, and sagittal axes, of the torso model, respectively, the model was filled with 0.1% saline solution (solution temperature; 20°C, specific resistance; 604 Ohm cm). Each electric dipole of the unit was energized in each of the 3 anatomical directions in turn by alternating current of 3 mA (100 Hz) through the use of an oscillator (Yokogawa-Hewlett-Packard Ltd, Model 204B), and the potential difference between each lead point and the Wilson central terminal was measured point by point by an AC voltmeter (Yokogawa-Hewlett-Packard Ltd, Model 403B). The potential difference between the Wilson central terminal and each of the 85 lead points as well as the 7 lead points for Frank lead system was registered as the lead vector of the corresponding unipolar lead.

An image surface of the torso model was obtained by plotting the tips of the lead vectors on a section paper, and the X, Y, and Z lead vectors of the Frank lead system were obtained by means of the drawing method by Frank from the image surface obtained.

Frank lead scalar X, Y, and Z electrocardiograms were also recorded together with Lead II ECG from the subject and digitized every 3 msec through the same A/D converter that used when the unipolar lead ECGs were treated. Thus, the lead vectors of those 85 unipolar leads and of the orthogonal standard X, Y, and Z leads of Frank lead system and the potential differences in X, Y, and Z leads at respective instant were obtained and prepared for the mathematical potential calculation at those 85 lead points.

According to the single fixed-location dipole hypothesis, the electric potential at each of those 85 lead points on the surface of the torso model with respect to that of the Wilson central terminal can be obtained by calculation through the use of equation mentioned below provided that the lead vectors of Frank lead system were rectangular each other and took the directions of their corresponding standard axes, respectively.

\[
V_P = \frac{F_X \cdot L_{PX}}{L_{PX}} + \frac{F_Y \cdot L_{PY}}{L_{PY}} + \frac{F_Z \cdot L_{PZ}}{L_{PZ}},
\]

where \(V_P\) is the relative electric potential at a given lead point P with respect to that of the Wilson central terminal, or the electric potential difference between these 2 points. \(F_X, F_Y,\) and \(F_Z\) are electric potential differences obtained from X, Y, and Z lead ECGs of Frank lead system, respectively. \(L_{PX}, L_{PY},\) and \(L_{PZ}\) are X, Y, and Z components of the lead vector of the unipolar lead at a given point P, respectively. \(L_{PX}, L_{PY},\) and \(L_{PZ}\) are magnitudes of lead vectors of Frank X, Y, and Z leads, respectively (see appendix). \(V_P\) was calculated on all of the 85 lead points.

After the electric potential, not absolute but relative to that of the Wilson central terminal, at each lead point was obtained on all of the 85 lead points by calculation, the equipotential lines were drawn by use of the X-Y plotter stage by stage. This map was referred to as Simulated map.
Hereafter the word electric potential at a point means relative potential, not absolute one.

3) Delineation of Difference map
The calculated electric potential at each of 85 lead points was subtracted from the measured one at the corresponding lead point and the difference thus obtained was also plotted point by point on the map indicative of the torso surface and equi-difference lines were further delineated. This map was referred to as Difference map.

4) Experiments for the estimation of the variations in lead vectors of Frank lead system caused by locational change of the artificial dipole model.
Experiments were further performed to check the range of the variation in the electric potentials calculated at those 85 lead points mentioned above. The unit of electric dipole model was tentatively shifted 2.4 cm from the original spot to the one in the 6 different directions, that is, toward the left, the right, the superior, the inferior, the front, and the back, and the variations in the magnitudes and directions of the X, Y, and Z lead vectors of Frank lead system caused by locational change of the electric dipole were checked.

RESULTS

1) On the map patterns of Measured, Simulated, and Difference maps
There was a good agreement of map pattern except slight differences in potential values between the 2 series of Measured maps obtained on 2 different occasions from the same subject keeping a proper interval, and the potential values used in the present paper were those obtained in the second series.

In major aspects, Measured map showed a serial pattern typical to that of normal subject throughout the period of QRS duration. Since it was impossible in this method to determine the exact onset of ventricular activation in the map, the earliest time when the electric potential in one of the 85 unipolar lead electrocardiograms reached 0.1 mV after the onset of QRS deflection was conventionally regarded to be the onset of ventricular activation. The zero line in each of Measured and Simulated maps simply means the plotting of the spots where the electric potential is equal to that of the Wilson central terminal. In Difference map, however, the zero line was obtained connecting the points where the electric potential in Measured map was equal to that in Simulated map, and hence it does not have the same meaning as that in Measured and Simulated maps.

In the early stage of the ventricular activation, the stage up to the 24 msec after the onset of ventricular activation, there was a fairly good agreement of map pattern between Measured and Simulated maps, so the description will be made on the maps obtained at the instants of 21 msec, 27 msec, 36 msec, and 45 msec after the onset of ventricular activation to show the outline of the time course of the change in Difference map as well as that in Measured and Simulated maps.
In Measured map obtained at the instant of 21 msec after the onset of ventricular activation (Fig. 2, upper map), one maximum and one minimum were observed (the terms “maximum” and “minimum” have the same meaning as those defined by Taccardi.9)). The maximum situated at the center level of the left parasternal line and the electric potential was as high as 0.85 mV and the minimum, at the lower part of the back near the dorsal

Fig. 2. Isopotential maps and Difference map obtained at the instant of 21 msec after the onset of ventricular activation.

The thick solid line in each of Measured and Simulated maps represent the plotting of the spots where the potential is equal to that of the Wilson central terminal and is called zero line. The thin solid lines are equipotential lines of 0.4 mV step. The area with the positive or negative sign show the place where the maximum or minimum is located and figure under sign is the value of electric potential in mV at that point with respect to that of the Wilson central terminal.

Difference map delineates the difference of electric potential between Measured and Simulated maps. The area with positive or negative sign represents the place where the difference between them is maximum, respectively, and the underlying figure represents its value. The thick solid line represents a series of the spots where there is no difference of electric potential between those 2 maps.
mid-line showing $-0.17\, \text{mV}$ in its potential. There were 2 zero lines running longitudinally on bilateral surfaces of the thorax. In Simulated map (Fig. 2, middle one), the maximum was $0.88\, \text{mV}$ in its potential and the minimum, $-0.11\, \text{mV}$, and they were seen at the similar places with those in Measured map. The differences in potential values between Measured and Simulated maps were very small and the values in Difference map were not more than $0.26\, \text{mV}$ even in the maximal value (Fig. 2, lower one).

At the instant of 27 msec, the maximum increased in its potential up to $1.06\, \text{mV}$ without changing its location in Measured map (Fig. 3, upper one). The minimum decreased in its potential down to $-0.33\, \text{mV}$ and changed its location to the right shoulder on the back. One zero line which

![Isopotential maps and Difference map](image)

**Fig. 3.** Isopotential maps and Difference map obtained at the instant of 27 msec after the onset of ventricular activation. The thin solid lines in each isopotential map are equipotential lines of $0.4\, \text{mV}$ step. Dotted line in Difference map is the plotting of the spots where the difference of the electric potential between Measured and Simulated maps is $-0.4\, \text{mV}$. Refer to legend of Fig. 2 for other explanations.

Note the appearance of the circumscribed area of negative value at the center of precordium in Difference map.
was observed in the right lateral aspect of the thorax at the instant of 21 msec shifted to the right anterior aspect of the thorax almost in parallel. The other one which was observed at the left lateral aspect of the thorax at the instant of 21 msec shifted to the left back almost in parallel, too. In Simulated map (Fig. 3, middle one) the maximum was observed at the same place with that in Measured one, but its potential of 1.42 mV was higher than that in Measured map. The location of minimum observed at the center of the back was rather different from that observed in Measured map. The

Fig. 4. Isopotential maps and Difference map obtained at the instant of 36 msec after the onset of ventricular activation. The dotted line in each of Measured and Simulated maps is the isopotential line of $-0.4 \text{ mV}$. The thin solid line in Difference map is the plotting of the spots where the difference between Measured and Simulated map is 0.4 mV. Refer to legend of Fig. 2 for other explanations.

In Measured map, zero line in the precordium shows a bulging (black arrow) toward left at the center level, while that in Simulated map is almost straight. Two minima are observed in Measured map, while only one minimum, in Simulated one. In Difference map 2 maxima of difference are seen. Isopotential line of $-0.4 \text{ mV}$ shows an upward bulging (white arrow) at the right anterolateral aspect of the thorax in Measured map while there is no such a bulging in Simulated map.
electric potential of the minimum was as low as $-0.17 \text{ mV}$. The dorsal zero line situated much nearer to the mid-line, so the area occupied by the positive electric potential in Simulated map became smaller in its size compared with that in Measured map. In Difference map (Fig. 3, lower one), a circumscribed area with negative value was observed around the middle of precordium where the maximum was observed in Measured and Simulated maps.

At the instant of 36 msec after the onset of ventricular activation, the maximum further increased in its potential up to $2.08 \text{ mV}$ and shifted its location to the left lower part of the precordium in Measured map (Fig. 4, upper one). Two minima were observed. One of them situated at the back of left shoulder showing the electric potential of $-0.62 \text{ mV}$, while the other one appeared newly at the center level of the right parasternal line showing the electric potential of $-0.50 \text{ mV}$. There were shiftings of zero lines. The one on the precordium shifted toward the left and the other on the back, toward the right. Thus, the former became to run down obliquely from the middle of the right clavicle to the right hypochondriac region displaying slight bulging (black arrow) toward the left at the mid-thoracic level, and the latter, to run down obliquely from the left shoulder to the mid-line on the back.

In Simulated map of that instant (Fig. 4, middle one), the maximum appeared at similar region to that in Measured map. The potential as high as $1.88 \text{ mV}$ was slightly lower compared with that in Measured map. The expansion of the area occupied by positive equipotential lines toward the left upper aspect of the thorax was less remarkable compared with that observed in Measured map. Only one minimum was present at the central part of the right precordium. There was no such a bulging of zero line or of $-0.4 \text{ mV}$ equipotential line as that in Measured map.

In Difference map (Fig. 4, lower one), the negative area at the central part of the precordium where the bulging of zero line was observed in Measured map became more negative in its potential compared with that of the instant of 27 msec. A conspicuous positive area was observed in the left upper thoracic area where the expansion of the area occupied by positive equipotential lines was observed in Measured map, and another positive area was observed in the right anterior aspect of the thorax where the bulging of $-0.4 \text{ mV}$ equipotential line was observed in Measured map.

Afterward, the author could not find good agreement between Measured and Simulated maps at any stage of ventricular depolarization. As an instance, the 3 maps obtained at the instant of 45 msec after the onset of ven-
Fig. 5. Isopotential maps and Difference map obtained at 45 msec after the onset of ventricular activation.

Difference in potential distribution between Measured and Simulated maps is much more remarkable than that of 36 msec.

tricular activation were shown in Fig. 5. As may be seen in the figure, the difference between those 2 maps was much more remarkable.

2) Changes in magnitudes and directions of lead vectors of Frank X, Y, and Z leads caused by the locational change of the unit of electric dipole model

In lead X, the magnitude of the lead vector presented the maximal change in the leftward displacement and was greater as much as 14% of the control which was obtained when the dipole model was placed at the anatomical heart center. In lead Z, the maximal change was observed in the forward displacement and was greater as much as 26% of the control. On the contrary, in lead Y, the change was very small, being less than 4% of the control in every displacement.

The angles between each 2 of the 3 lead vectors obtained at each 7 different spots mentioned above were shown in the table. The mean ± S.D.
Changes in the Directions and Magnitudes of Lead Vectors of Frank X, Y, and Z Leads Caused by the Locational Change of the Unit of Electric Dipole

<table>
<thead>
<tr>
<th>Dipole location</th>
<th>Angles between lead vectors</th>
<th>Magnitudes of lead vectors</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>X and Y</td>
<td>Y and Z</td>
</tr>
<tr>
<td>Heart center</td>
<td>84°</td>
<td>84°</td>
</tr>
<tr>
<td>2.2 cm left</td>
<td>84°</td>
<td>82°</td>
</tr>
<tr>
<td>&quot; back</td>
<td>85°</td>
<td>87°</td>
</tr>
<tr>
<td>&quot; right</td>
<td>81°</td>
<td>84°</td>
</tr>
<tr>
<td>&quot; front</td>
<td>84°</td>
<td>84°</td>
</tr>
<tr>
<td>&quot; superior</td>
<td>78°</td>
<td>69°</td>
</tr>
<tr>
<td>&quot; inferior</td>
<td>89°</td>
<td>101°</td>
</tr>
<tr>
<td>Mean±S. D.</td>
<td>83.6°±3.2°</td>
<td>84.4°±8.7°</td>
</tr>
</tbody>
</table>

of each 7 values of angle was 83.6°±3.2° with the one between lead vectors X and Y, 84.4°±8.7° with the one between lead vectors Y and Z, and 94.6°±7.9° with the one between lead vectors Z and X.

**DISCUSSION**

For the single fixed-location dipole hypothesis, it is indispensable that the resultant dipole which is equivalent to the cardiac electromotive force stays at a fixed location throughout the whole stage of the ventricular activation. There is, however, a report stating that the cardiac electromotive force is represented more accurately by a moving dipole which changes its location following the process of ventricular activation. Toyoshima et al., reconstructing VCGs and the 12 standard lead ECGs following the propagation of ventricular activation, found fairly good agreement between the experimental ECGs and reconstructed one on both normal and various patients with cardiac disease.

As well known and as seen in the present experiment, dislocation of the electric dipole from the anatomical heart center causes the changes of the lead vector and hence of the value of represented electromotive force. In addition, the lead vectors of the given 85 leads in the present experiment are valid only when the equivalent dipole is situated at the anatomical heart center. Therefore, the more the equivalent dipole dislocates from the anatomical heart center, the more were invalid the lead vectors and hence the calculated potentials. If the lead vectors of the orthogonal 3 component leads
of Frank lead system, however, do not show any marked change in their magnitudes and directions even though the equivalent dipole is dislocated from the anatomical heart center, the equivalent dipole can be treated as fixed locational one with minor range of error and the validity of calculated potential is sustained.

As far as the present experiment is concerned, the change in the magnitude of each lead vector of the 3 component leads of Frank lead system did not exceed 26%, whatever direction the dipole was dislocated in so far as it was located within a sphere of 2.4 cm in radius. Since the sphere covers almost the full region occupied by the ventricular mass, it was considered that this range might be large enough to cover the error range caused by the locational change of the equivalent dipole. Therefore, the discussion was limited to the phenomena which were always common to 2 series of Difference maps obtained on different occasions so far as the variations of the calculated potentials were limited within the range of 26%.

As may be seen in the present data, there was good agreement of potential distribution, and accordingly of locations and potentials of maximum and minimum of Measured (Fig. 2, upper one) and Simulated (Fig. 2, middle one) maps at the instant of 21 msec after the onset of ventricular activation. In Difference map (Fig. 2, lower one), the maximal value of the difference was as great as 0.26 mV and this was supposed to be within the error range so far as the variations of 26% in the calculated potential were admitted as a possible error range.

During the stage from the onset of ventricular activation up to the instant of 18 msec, the difference between Measured map and Simulated one was less remarkable than that at the instant of 21 msec. Good agreement was also seen at the instant of 24 msec. Therefore, it might be considered that the cardiac electromotive force could be well represented by a single electric dipole fixed to the heart center without any significant error during the stage up to this instant.

At the instant of 27 msec after the onset of ventricular activation, however, the electric potential at maximum and nearby region in Measured map (Fig. 3, upper one) was lower than that in Simulated one (Fig. 3, middle one) although the location of maximum in the former map coincided with that in the latter one. This difference in electric potential resulted in the appearance of a circumscribed negative area in Difference map and the difference was as great as -0.52 mV at the nadir. This difference was too great to represent the cardiac electromotive force by a single electric dipole fixed to the heart center.

According to the study by Durrer et al,20 this stage is the one around the
occurrence of epicardial breakthrough of the activation front at the anterior wall of the right ventricle. Once the epicardial breakthrough occurs, the depolarized area of the subepicardium makes a localized deficit of electromotive force, or an opening of electric double layer, and extends its area along with the propagation of activation front causing the abrupt decreasing of electric potentials at lead points facing to the deficit area, or the opening. On the other hand, it is reported that in most of normal subjects there appears a potential distribution similar to a "niche", that is, a second minimum surrounded by a relatively higher potential, and this potential distribution is tentatively explained to be caused by the deficit of electromotive force produced by the epicardial breakthrough. This phenomenon was verified by Spach et al by comparing the body surface isopotential map of a patient with his epicardial sequence of ventricular activation obtained at the cardiac surgery.

In the present study, however, such a change in potential distribution of a localized area suggestive of the occurrence of the epicardial breakthrough was not observable both in Measured and Simulated maps of this instant, especially in the latter (Fig. 3, upper and middle ones). This is probably because the distribution of the selected lead points was too sparse to reflect the potential change in localized body surface area.

It was also supposed that the most important reason for the lacking of such a change in Simulated map was in the procedure of mathematical potential calculation. In the present experiment, every lead vector necessary for the calculation was obtained by replacing the electromotive force of the heart by a single electric dipole fixed to the cardiac center. So, the proximity potentials were much less marked to be found in the Simulated map.

Despite the absence of the discernible potential distribution suggestive of the epicardial breakthrough of ventricular activation in Measured and Simulated maps of this subject at this instant, a conspicuous circumscribed area was observed on the middle precordium in Difference map in the present study. From the appearance of this negative area, it was supposed that there was a latent local decrease in potential in Measured map at the place where the negative area was observed in Difference map. It was also an important finding that the negative area in Difference map situated at the place under where the epicardial breakthrough was most likely to occur in normal hearts. According to the experimental results by Toyama, a great deal of non-dipolar components, the components which can not be represented by a fixed-location electric dipole, were contained in the unipolar lead ECGs recorded from the precordium near the heart. Therefore, such a latent decrease of potential in Measured map as revealed by a circumscribed negative area in Difference
map may be due to the occurrence of the epicardial breakthrough.

In Measured map obtained at the instant of 36 msec after the onset of ventricular activation (Fig. 4, upper one), the area with relatively higher potential between 2 minima, one on the back and the other on the precordium, was similar to "saddle" distribution by Taccardi.\(^9,10\) Taccardi et al\(^9,10\) as well as Spach et al\(^9\) and Boineau et al,\(^27\) who, the latter 2, studied the relationship between the isopotential map and the sequence of ventricular activation, stated that "saddle" was a phenomenon closely related to the activation front which is continuing to spread in the right ventricle after the occurrence of epicardial breakthrough.

Contrarily to the complexity of Measured map, Simulated one displayed the smooth equipotential lines with single maximum and minimum at this instant.

In Difference map obtained at this instant, the differences of 0.30 mV and 0.73 mV in the respective positive areas on the right precordium and left lateral thorax and the difference of \(-0.98\) mV in the negative area on the mid-precordium were values out of the error range. Therefore, the electromotive force could hardly be represented equivalently by a single fixed-location dipole at this instant.

Presumably, the appearance of the negative area on the precordium in Difference map was caused by further extension of the deficit area of the activation front in the right ventricle on the same ground stated on Difference map obtained at the instant of 27 msec after the onset of ventricular activation. The positive areas to the right of the right parasternal line and on the left upper thorax may be related to the proximity potential of the activation front which is spreading mainly affecting the anterior aspect of the right and left ventricles. Thus, Difference map seems to suggest the outline of activation front at a glance.

It is also possible, however, to surmise the appearance of the circumscribed deficit area of activation front at the anterior ventricular wall from Measured map obtained at this instant on account of the next 2 reasons. First, the appearance of new minimum about the middle of precordium seems to suggest the appearance of the deficit area of activation front at the underlying cardiac region. Second, the area with relatively high electric potential at the right precordium as expressed by the bending of 0.4 mV equipotential line seems to suggest the existence of activation front which is spreading affecting the anterior wall of the right ventricle as stated by other researchers.\(^9,12\) Anyway, at this stage it is considered that the deficit area of the activation front has acquired a rather extensive area.

Although the potential distribution suggestive of the occurrence of
epicardial breakthrough of the activation front was observed on Measured map so late as the instant of 36 msec, this kind of distribution would be observable at somewhat earlier stage if more numerous lead points were selected on the body-surface. In Difference map, however, the occurrence of epicardial breakthrough was already observed in the earlier stage, the stage of 27 msec. It was further observed in Difference map at the instant of 36 msec that there was a potential distribution suggestive of the existence of the separated 2 major activation fronts which were spreading affecting the right and left ventricular walls, respectively. Therefore, it was considered that Difference map was helpful for the estimation of the spread of ventricular activation.

In this experiment, the torso model used for the measurement of lead vectors was molded after a living subject as precisely as possible, but it was filled with the homogeneous saline solution. Therefore, the lead vectors obtained under these experimental conditions can not necessarily be applied to the torso of the original living subject as a linear constant, since there are tissues with different electric conductivity such as intracavitary blood, lungs, and so on which influence the lead vector. Brody,28) for instance, stated that the lead vectors greatly differed according to the physical conditions such as the activation front lay in contact with the intracavitary blood or it lay remote form the blood and that so-called mean lead vectors could not exist throughout the whole stage of ventricular activation. There are also some experimental studies which verified29) that even the single dipole could produce multiple maxima on the torso surface when the substance with different electric conductivity was put into the volume conductor to imitate a couple of lungs, or verified30) that Simulated isopotential maps obtained on a canine torso containing lungs and heart became more similar to Measured maps than Simulated maps obtained on the canine torso model without lungs and heart.

The phenomena seen on Difference map in the present paper was the one observed around the stage when the activation front was spreading near the epicardium of the anterior ventricular wall where there is a thin layer of lungs wedged into the space between the anterior thoracic wall and the cardiac surface. The tissues and organs in the living torso make an electrically inhomogeneous field unlike the saline solution in the experimental torso model. In the mathematical potential calculation in the present report, however, the effect of inhomogeneity is already taken into consideration to a certain degree in the equation presented in this paper (see appendix). Nevertheless, there was marked difference between Measured and Simulated maps depending upon the stage of ventricular activation. That is, there was good agreement
between those 2 maps in early stage but marked difference in later stage. Therefore, it was supposed that the differences between these 2 maps were caused mainly by the changes in electromotive force itself of the heart, not by the existence of intracavitary blood or the lungs. This means the electromotive force in the later stage could not be represented by a single fixed-location dipole.

**APPENDIX**

According to a single fixed-location dipole hypothesis, electric potential \( V_P \) at a given lead point \( P \) can be determined by the next equation:

\[
V_P = H_x \cdot L'_p X + H_y \cdot L'_p Y + H_z \cdot L'_p Z
\]

This equation is transformed into

\[
V_P = \frac{H_x \cdot L'_p X \cdot L'_p X}{L'_p X} + \frac{H_y \cdot L'_p Y \cdot L'_p Y}{L'_p Y} + \frac{H_z \cdot L'_p Z \cdot L'_p Z}{L'_p Z}
\]

where, \( H_x, H_y, H_z \); \( X, Y, \) and \( Z \) components of cardiac dipole moment, respectively,

\( L'_p X, L'_p Y, L'_p Z \); \( X, Y, \) and \( Z \) components of the lead vector of unipolar lead at the lead point \( P \), respectively,

\( L'_p X, L'_p Y, L'_p Z \); lead vectors of Frank X, Y, and Z scalar lead, respectively.

Electric potential differences, \( F_x, F_y, \) and \( F_z \) of component \( X, Y, \) and \( Z \) leads of Frank lead system are measurable on the living human subject, and they are also expressed by next equations, provided that the lead vectors of Frank lead system are rectangular each other and take the directions of their corresponding standard axes, respectively.

\[
F_x = H_x \cdot L'_p X, \quad F_y = H_y \cdot L'_p Y, \quad \text{and} \quad F_z = H_z \cdot L'_p Z
\]

Applying equations (3) to equation (2),

\[
V_P = \frac{F_x \cdot L'_p X}{L'_p X} + \frac{F_y \cdot L'_p Y}{L'_p Y} + \frac{F_z \cdot L'_p Z}{L'_p Z}
\]

In the present simulation experiment, the calculation of electric potential at the lead point \( P \) on the torso model was made using the potential differences \( F_x, F_y, \) and \( F_z \) measured on the living subject. The lead vectors \( L'_p X, L'_p Y, L'_p Z, L'_p X, L'_p Y, \) and \( L'_p Z \), however, can not be obtained from the living subject. Therefore, the lead vectors \( L'_p X, L'_p Y, L'_p Z, L'_p X, L'_p Y, \) and \( L'_p Z \) were obtained by the torso model experiment in place of the former.

Substituting the latter for the former in equation (4),
This is the equation used for the calculation of the potential at a given lead point P.

To consider what this equation implies, the equation was again transformed by using equation (3).

\[ V_P = \frac{H_X' \cdot L''_{PX}}{L_{PX}} + \frac{H_Y' \cdot L''_{PY}}{L_{PY}} + \frac{H_Z' \cdot L''_{PZ}}{L_{PZ}}. \]

This is further rewritten as

\[ V_P = H_X' \cdot L''_{PX} + H_Y' \cdot L''_{PY} + H_Z' \cdot L''_{PZ}, \]

where,

\[ L''_{PX} = \frac{L'_{PX} \cdot L_{PX}}{L_{PX}}, \quad L''_{PY} = \frac{L'_{PY} \cdot L_{PY}}{L_{PY}}, \quad \text{and} \quad L''_{PZ} = \frac{L'_{PZ} \cdot L_{PZ}}{L_{PZ}}. \]

Here the equation shows a form similar to equation (1). \( L'_{PX}, L'_{PY}, \) and \( L'_{PZ} \) are the values obtained by multiplying \( L_{PX}, L_{PY}, \) and \( L_{PZ} \) by respective ratio of the lead vector of each component lead of Frank lead system in the living subject to that in the torso model. Hence the calculated potential \( (V_P) \) reflects to a certain degree the effect of the inhomogeneity of the living subject.

Therefore, the differences which appeared in Difference map in the later stage are considered to be mainly due to the changes in cardiac electromotive force itself. That is, the cardiac electromotive force in the later stage can not be represented by a single fixed-location dipole.

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