THE BODY SURFACE ISOPOTENTIAL MAPS OF THE NON-TRANSMURAL INFARCTIONS

— A Simulation Study of Excitation Spread in a Ventricular Model —

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Simulation by a digital computer of excitation spread in a human ventricular model produced displays of body surface isopotential maps.

Localization of model myocardial infarctions to non-transmural and transmural sites produced distinctive differences in these displayed isopotential maps. The body surface directly over the infarcted lesion was negative in potential only in the first half period of the QRS complex and then became positive.

The model demonstrated that this later positive potential was due to delayed arrival of excitation to the subepicardial layer outside of the subendocardial lesion.

While, in transmural infarction, the overlying body surface remained negative in potential throughout the QRS complex.

It is expected that body surface isopotential maps will become clinically available and will permit helpful differential diagnoses between non-transmural and transmural myocardial infarctions.

IN general, transmural infarction is easily diagnosed by the appearance of abnormal Q waves on a conventional ECG.^{1,2} In non-transmural infarction, it has been disputed whether there are the same QRS changes as those in transmural infarction.³⁻⁶ Durrer et al⁷ reported that abnormal Q waves were observed in electrograms at the epicardial sites overlying the subendocardial infarcted lesion, although its size was

limited to only 1 cm in diameter, over the one-fourth of the whole wall thickness.

As for abnormal Q waves in non-transmural infarction, clear evidence was not given in animal experiments because it was impossible to produce non-transmural infarction. Okajima et al⁸ and Kono⁹ devised a simulation of ventricular propagation using a digital computer with a human heart model. They reported that abnormal Q waves appeared on a conventional ECG in anterior non-transmural infarction. In 1968, Boineau et al¹⁰ described that delayed activation of the intact subepicardial region outside of the inner lesion was a characteristic phenomenon in

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Fig.1. Comparison of simulated control maps (A) with those obtained from a normal man (B).

Each body surface map is cut at the right axillary line and extended to a plane. The anterior, the left lateral chest and the back stand side by side from left to right of each map. The central horizontal level shows the 5th intercostal space. The dotted line is at the level of null potential, which is isopotential to Wilson's central terminal. The shaded area shows positive potential and the clear one, negative. The plus and the minus marks are at the peak of positive (maximum or maxima) and negative (minimum or minima) potential area, respectively.

Simulated maps resemble those obtained from a normal man.

Abbreviation: RAL=right axillary line, MSL=mid-sternal line, LAL=left axillary line and 5th i.c.s.=the 5th intercostal space.

anterior non-transmural infarction, which was recognized as a late positivity in the left anterior chest. It is the purpose of this paper to confirm whether the aforementioned late positivity due to delayed activation of the intact subepicardial layer as well as abnormal Q wave are common findings on body surface isopotential maps (map) in non-transmural infarctions at the miscellaneous sites of the left ventricle. Therefore, we carried out the simulation of ventricular excitation using the heart model with some fictitious infarcts.

METHOD

The model of the ventricles

As previously described⁸ a fresh heart was obtained by autopsy from an old man without heart disease. It was taken from the thorax, maintaining its intrathoracic orientation. Then, it was divided into twenty-seven thousand, 3 mm cube blocks. They served as a ventricular model

for the digital computer. Specialized conduction systems such as the Purkinje network in the ventricles were arranged on the basis of the distribution of a dog. The spread of ventricular excitation within the specialized conduction system was determined at a velocity 7 times that of ordinary cardiac muscle.

Reconstruction of the body surface isopotential maps

Simulation of ventricular excitation spread in a human heart model with miscellaneous infarctions fictitiously produced was carried out using a HITAC digital computer system at Tokyo Univ. and a FACOM 230-60 at Nagoya Univ.

The map in each instance was reconstructed from the potentials obtained from 85 lead points (59 of them in the anterior chest, the others in back). The potential in each lead point was calculated as follows, the heart vector in each

Fig.2. Sequential maps in normal (A), anterior non-transmural (B), and anterior transmural (C) infarctions.

Note that, in B, the upper portion of the left anterior chest was occupied by a negative potential in the early stage (20 msec after the onset of QRS complex) and displaced by a positive potential in the late stage (60 msec).

While in C, negative potential occupied the upper portion of the left anterior chest throughout the period of QRS complex.

With the V3 lead on, a late R wave appeared, resulting in rSR' pattern in non-transmural infarction, although RS and rS patterns were observed in normal and transmural infarction, respectively.

instance was calculated from the area of the excitation front and was multiplied by each transfer impedance vector.¹¹ It was assumed that in the infarcted lesion, the excitation did not propagate to any blocks of ordinary cardiac muscle and specialized conduction systems, yielding no cardiac electromotive force there. The simulated body surface isopotential maps with a normal heart model resembled those actually recorded from a normal man (Fig. 1) and those shown in some reports,¹²⁻¹⁴ although the details were slightly different. Accordingly, this simulation study was thought to be an appropriate method in order to study and discuss the relationship between body surface potential

and ventricular excitation process in myocardial infarctions.

Arrangement of infarction in a ventricular model I) Anterior infarction model (Fig. 2)

A. Non-transmural model: The infarction was arranged over about one-half to three-fourths of wall thickness in the subendocardial side at the anterior free wall of the left ventricle. Its volume was about 3.9 cm³ with an area of 5.5 cm² on the endocardial surface (12% of the total endocardial area in the left ventricle). The Purkinje networks in the impaired wall were all involved in the "infarction". This assumption holds true in all of the following infarct models.

Fig.3. Sequential maps in normal (A), lateral non-transmural (B), and transmural (C) infarctions.

In B and C, the upper part of the left lateral chest was occupied by a negative potential in the early stage. In the late stage, it was displaced by an increased positive potential with a maximum in B. This late displacement was not observed in C.

The duration of QRS complex was remarkably prolonged in B. With the aV_L lead on, increased depth and duration of Q wave and reduced R wave were shown in B and C.

B. Transmural model: The transmural infarction was set up at the same site as that of I-A model. Its volume was about 6.6 cm^3 .

II) Lateral infarction model (Fig. 3)

A. Non-transmural model: The infarction was arranged over about one-third to three-fourths of wall thickness in the subendocardial side at the lateral wall of the left ventricle. Its volume was about 7.2 cm^3 with an area of 13.2 cm^2 (29% of the total endocardial area).

B. Transmural model: The transmural infarction was set up at the same site as that of II-A model, which volume was about 11.8 cm^3 .

III) Inferior infarction model (Fig. 4)

A. Non-transmural model: The infarction was arranged at the inferior wall (including a part of the posterior wall), partly leaving the ordinary cardiac muscle subepicardially around the apex to the lower posterior wall. Its volume was 3.8 cm^3 with an area of 11.0 cm^2 (20% of the total

endocardial area).

B. Transmural model: The infarction was arranged transmurally 2.1 cm upward from the tip of the apex, of which the volume was about 9.1 cm^3 .

IV) Pure-posterior infarction model (Fig. 5)

A. Non-transmural model: The infarction was arranged over one-half to two-thirds of wall thickness in the subendocardial side at the proper pure posterior region. Its volume was 4.7 cm^3 with an area of 6.3 cm^2 on the endocardial surface (13% of the total endocardial area).

B. Transmural model: The infarct of 8.3 cm^3 was arranged transmurally at the same sub-endocardial site as that of IV-A model.

RESULTS

I) Anterior infarction (Fig. 2)

Early stage of QRS complex

In both non- and transmural infarctions, the

Fig.4. Sequential maps in normal (A), inferior non-transmural (B) and transmural (C) infarctions.

In B, the lower part in the back was altered to a negative field in the early stage. In the late stage, it was displaced by a positive one in B. This late displacement was not observed in C. The potential distribution in B was the reverse of those in A and C.

With the aV_F lead on, an $rsr's'r$ pattern with high frequency notches was observed in B. In C, late r waves were not.

upper portion of the left anterior chest was displaced by a minimum negative potential, which showed the location of the lowest potential. Consequently, the positive potential distributed in the lower half of the chest had a maximum, which indicated the region of highest potential and shifted to the left and downward as compared to the normal case. Furthermore, its magnitude was diminished.

Middle stage

The positive area at this stage extended from the left lateral region to the lower half of the chest. The maximum was located on the left midaxillary line. The negative area was found over the upper portion of the right anterior chest. This distribution of positive and negative potentials at this stage was not altered remarkably in contrast to a normal situation. The negative potential at the upper portion of the left anterior chest continued until the middle stage. We called it the "abnormal Q area".

The alterations of maps at the early and mid-

dle stages were not different between non- and transmural infarctions.

Late stage

At this stage in non-transmural infarction, the positive area extended into the upper portion of the left anterior chest, where "abnormal Q area" had appeared at the early stage because of anterior subendocardial involvement. Furthermore, the position of a maximum localized in the anterior positive area exactly coincided with that of the minimum at the early stage. While, in transmural infarction, the upper portion of the left anterior chest remained negative at this stage, resulting in "abnormal Q area" throughout the period of QRS complex. The duration of QRS complex in non-transmural infarction was prolonged by 15% as compared to the norm. However, in a transmural one, the QRS complex was not prolonged.

In lead V3 where QRS complex showed an RS pattern in the control, rsR' and rS patterns were observed in non- and transmural infarctions,

Fig.5. Sequential maps in normal (A), non-transmural (B) and transmural (C) infarctions at the posterior wall.

In the early stage, in B, the whole back was occupied by a minimum negative potential. Correspondingly, the amplitude of positive potential over the anterior chest was increased, yielding denser isopotential lines there.

In the late stage, the back was displaced by a positive potential in both B and C. The early and late maps were similar.

With the V1 lead on, an increase in R wave was observed in B and C.

respectively.

II) Lateral infarction (Fig. 3)

Early stage

The upper portion of the left lateral chest around the left shoulder was displaced by a negative potential at this stage. The positive area protruded into the negative area at the left anterior chest, accordingly, its expansion was reduced as compared with the norm.

Middle stage

The positive area extended over the left half of the chest and the right lower portion. The negative area was located around the right shoulder. The distribution of both potentials resembled that of a normal model except that their magnitudes in both non- and transmural infarctions were lower, yielding sparse isopotential lines on the map at this stage.

These findings at both stages were observed in non-transmural as well as transmural infarctions.

Late stage

The positive area extended over the left half of the chest. There were two differences be-

tween non- and transmural infarctions; (1) The magnitude of the positivity was higher in non-transmural infarction, yielding a maximum in the positive area. (2) The duration of QRS complex was prolonged remarkably by 60% in non-transmural infarction. No prolongation was observed in a transmural one.

In lead aVL where QRS complex showed a qR pattern in the normal subject, a QR pattern with wider and deeper Q wave was observed in both non- and transmural infarctions.

III) Inferior infarction (Fig. 4)

Early stage

The lower portion of the back was replaced by a negative potential. While the whole anterior chest was occupied by a positive potential. The high voltage area present, characterized as dense isopotential lines on the map, was shifted to the upper portion. These findings on the early maps were observed in both non- and transmural infarctions.

Late stage

At this stage in non-transmural infarction, the

Fig. 6. Horizontal cross-sections of the left ventricle showing propagation process in normal (A), anterior (B) and lateral (C) non-transmural infarctions.

Note the denser isochronal lines at the subepicardial side outside of the inner lesion (shaded area) in B and C. These findings indicate a delayed excitation there.

lower half of the chest was displaced by a positive potential. The distribution was such that the positive potential was in the lower half of the chest and the negative was in the upper half—completely opposite to those found in normal and transmural infarction. The duration of QRS complex was prolonged by about 15% only in non-transmural infarction.

In lead aVF where QRS complex showed an Rs pattern, an rsr's'r" pattern with high frequency notches and an rS pattern were observed in non- and transmural infarctions, respectively.

IV) Pure posterior infarction (Fig. 5)

Early stage

The whole back was displaced by a minimum negative potential. The positive area extended over the whole anterior chest. As a reciprocal change, the positive potential in the upper portion of the left anterior chest increased in amplitude, resulting in dense isopotential lines shown on the map. These changes on the map were observed in both infarctions.

Late stage

In both non- and transmural infarctions, the back, where negative potential was present in the early stage, was displaced by a positive one. The whole anterior chest had a negative potential. The difference between non- and transmural infarctions was minor, only in that the negativity was more intense than in the former. The duration of the QRS complex was prolonged by 30% in non-transmural infarction contrasted to 15% in the transmural one.

In lead V1 where QRS complex showed an rS

pattern in the control, the r wave became taller resulting in an RS pattern in both non- and transmural infarctions.

DISCUSSION

The changes during the early stage of QRS complex

Each associated body surface area with the sites of non-transmural infarctions was occupied by a negative potential, that is an "abnormal Q area". None of the non-transmural infarction models set up in this study had any Purkinje fibers in the subepicardial layer outside the inner lesion. However, when some Purkinje fibers were there, the time during which the negative potential appeared over the associated body surface was short, not resulting in the so-called "abnormal Q area". Accordingly, the appearance of an "abnormal Q area" in non-transmural infarction may depend upon the degree of impairment of the Purkinje fibers.

The appearance of "abnormal Q areas" on the body surface, except for the right anterior chest and the upper portion of the back which were normally negative at the beginning of QRS complex, is useful in diagnosing the localization of non-transmural infarctions as well as transmural ones.¹⁵⁻¹⁷

The changes during the late stage of QRS complex

Generally in non-transmural infarction, excitation through the intact subepicardial layer progressed slowly by muscle to muscle conduction, because Purkinje fibers with fast conductivity

were injured. Excitation of that intact subepicardial region was delayed until the late stage of QRS complex (Fig. 6). The negative body surface area which was due to infarction at the early stage was displaced by a positive potential at this stage. This late displacement by a positive potential is compatible with "late R wave" on the conventional ECG. It is most typical in the anterior wall infarction, where the position of a maximum on the map in the late positive area coincided with the place where the minimum had appeared initially. This phenomenon was called "peri-infarction block" by First et al.¹⁹ and is thought to be a characteristic of non-transmural infarction, although it might occur in the transmural one as well. In addition, the duration of QRS complex with each non-transmural infarct was prolonged in our simulation experiments as they reported. This delayed excitation of the intact subepicardial region, if infarctions were transmural in the anterior and the inferior ventricular walls, did not occur. Accordingly, the displacement of the initial negative area as a change of infarction by a positive potential at this time was not observed and the associated area with the location of the lesion was negative throughout the period of QRS complex. Thus, it is easy to make a differential diagnosis between non-transmural and transmural infarctions. However, in spite of transmural infarcts at the lateral and the pure posterior walls, the displacement of the "abnormal Q area" by a positive potential was observed on the late maps, leaving it difficult to differentiate from non-transmural ones.

Clinical cases with anterior non-transmural infarct were reported^{9,20} on whose map there was an initial negative and a late positive potential in the left anterior chest. On the contrary, Yamada et al.²¹ produced non-transmural infarctions by ligation of the left coronary artery in dogs. As a result, they did not reveal "late R wave" on the maps. The reasons for this discrepancy are considered as follows; i) Anatomical differences between hearts and the positioning of electrodes at the chest wall, that is, the difference of "electrical field" between a man and a dog²² ii) In our heart model, Purkinje fibers invaded over one-fourth to one-half of the total wall thickness in the subendocardial side, while they might invade deeper toward the subepicardial layer as reported by Sodi-Pallares²³ iii) Pathologically, most of non-transmural infarctions were occupying ring-like lesions. Otherwise, if necrosis is scattered, a part of the Pur-

kinje fibers and ordinary cardiac muscles are left intact in the infarcted mass. iv) There may be an ischemic zone inside of necrosis²⁴

Although there are some problems, it is expected that body surface isopotential maps will become clinically available and will permit a viable differential diagnosis between non-transmural and transmural infarctions.

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