Body Surface Isopotential Maps

Clinical Application to the Diagnosis of Myocardial Infarction

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Summary

This paper is a review of recent work relating body surface iso-
potential maps to the detection of the site and extent of myocardial
infarction in cases which are either indetectable or difficult to diagnose
through the use of standard 12 lead ECGs. According to the difference
of the site and extent of myocardial infarction, the characteristic maps
are obtained. Through the use of body surface isopotential maps, the
significant clinical information may be obtained in a number of cases,
and we can do better with mapping than without it in the evaluation of
patients with myocardial infarction.

Additional Indexing Words:
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For the clinical medical doctor, the diagnosis of myocardial infarction
continues to be an important matter. Through the works1)-16) of many
investigators, this problem has been resolved to a degree, using the standard
12 lead ECG or VCG. However, several points such as the detection of the
extent of myocardial infarction and precise detection of right ventricular in-
farction and high posterior infarction still remain difficult problems.

Recently, body surface isopotential maps (maps) have been introduced
as a method of clinical examination. Maps, as the name implies, provide a
presentation of body surface potential distributions over the entire trunk
surface. About 90 years ago, such maps were imagined by Waller17) and in
the 1950’s, Nahum, Mauro, Sikand, and coworkers18)-20) obtained maps ac-
tually recorded from a large number of lead points on the body surface and
in the 1960’s, Taccardi21),22) gained a sequence of maps in men and dogs
covering the entire ventricular depolarization process. They stated that maps
have some significant information not present in the conventional ECG.
However, the system for recording and displaying maps was too complicated

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to become widespread. This difficulty is becoming less significant with the use of computers to process the large amount of body surface electrocardiographic data. Thus, the study of this field has greatly progressed. Consequently, it has become clear that the area of positive and negative potentials is apparently associated with major wave fronts within the heart. Noting the distribution of the positive and negative areas in each stage and the transformation of the areas, we can estimate the cardiac electrical phenomena. The locations and potentials of maxima and minima, the movement of maxima and minima also provide us with considerable informations about cardiac wave fronts. That is, it has been shown that maps are useful method to detect abnormal cardiac electrical phenomena precisely and that maps contain several advantages over conventional ECG.

In this review, based on our experimental and clinical study of myocardial infarction, we wish to stress that myocardial infarction, even cases which are difficult to diagnose by conventional ECG, can be detected easily through the use of maps.

**Materials and Methods**

Clinical materials:

Through the use of maps, we have examined more than 100 myocardial infarction male patients (ages 37 to 67, average 55.6) the diagnosis of which were confirmed by acute phase clinical data, ECG, and VCG. In this review, we selected the patients who show only single vessel disease, that is, their obstruction sites (obstruction means 90% or more than 90% obstruction) are limited to only one main coronary artery and there are no obstruction in the other two main coronary arteries. This selection was done so that the relationship between map patterns and the location or extent of the myocardial infarction could be clearly understood. Therefore, only 24 patients whose single vessel disease was certified by cineangiography are taken up in this study. In all the cases maps were recorded within 3 to 12 months from the occurrence of myocardial infarction attack and within 1 to 7 months from the cineangiography.

Ultimately, we want to establish the relationship between autopsy findings and features of maps. Since all cases survived, it was not possible to obtain such correlations. Therefore, as the second best plan, the features of myocardial infarction patients' maps are explained by experimental study and cineangiographic findings.

For control, maps were obtained from 20 apparently healthy persons (ages 22 to 40) as well.

Experimental study:

Adult mongrel dogs were anesthetized and myocardial infarction was caused by ligation of the anterior descending artery, right coronary artery, or a branch of the circumflex artery. All the cases had 2 series of maps recorded before and after experimental myocardial infarction. Within 1 to 4 weeks after infarction, each heart was isolated and cut perpendicular to the long axis of the left ventricle.
into 1 to 2 cm thick slices for direct observation, and the extent and location of infarction were also confirmed by histological study.

Epicardial sequence of the ventricular depolarization; according to the method reported previously, the epicardial sequence of the ventricular depolarization was obtained from 3 healthy dogs after maps had been recorded.

Recording and displaying maps:

The procedure and displaying maps have been reported in detail previously. To sum up, each map was based on the record of the unipolar lead ECGs obtained through the use of paste-filled disposable electrodes of 1.1 cm diameter (clinical study), or needle electrodes (experimental study) attached to 85 lead points (59 on the anterior and 26 on the dorsal) (Fig. 1) in a supine position. In experimental study, each dog was anesthetized and artificially respirated.

It is indeed desirable that ECGs from all 85 lead points are recorded simultaneously. Almost all other investigators do so. However, our mapping system has not been able to progress 85 ECGs simultaneously. Therefore, as mentioned above, 2 lead points with the same time reference are recorded at a time and this procedure is repeated 43 times until all 85 lead ECGs are recorded. Even in this method the reproducibility is good enough.

It is important to determine how many lead points are necessary to obtain a map. There is no agreement among the investigators about this problem. That is, Flowers et al obtained maps from 142 lead points and Spach et al 150 to 250 points, and Taccardi et al 250 points. In general, maybe if there are more lead points, we have more information. On the other hand, if there are fewer lead points, we have more convenience for clinical use. In our laboratory, formerly we obtained maps from 121 lead points. Now we use usually 85 lead points because there are no significant difference between 121 lead points maps (Fig. 2A) and 85 lead points maps (Fig. 2B). We infer from our previous studies that 85 lead points maps are sufficient for clinical use. But we have no data concerning the minimum number of lead points which are necessary to obtain a map without loss of significant information.
RESULTS AND DISCUSSION

To understand the change of map pattern caused by infarction easily, we describe the normal map pattern.

*Normal human maps:*

Fig. 3 shows a typical series of normal human maps. To illustrate the map, the map was cut and separated along the right mid-axillary line on the thoracic surface and was fanned open.

The shaded area illustrates the positive zone, and the white area the negative zone. Each solid line illustrates an equipotential line drawn at an interval of 0.4 mV, and the broken line illustrates the potential of Wilson's central terminal as a zero potential. We assumed Spach and coworkers'
study that Wilson’s central terminal potential may be treated as the zero line.

In the early stage (15 msec), the whole anterior chest surface is occupied by a positive area and the dorsal a negative area. A maximum exists on the center of the anterior chest surface and a minimum on the upper dorsal.

In the early middle stage (27 msec), a negative area appears on the right upper anterior chest surface, and a minimum moves to right upper region. A maximum, which has already existed near the center of the anterior surface, remains its position. Niche like equipotential lines irregularity are observed. A positive area covers right lower anterior chest surface and left anterior and left axillary surface.

In the late middle stage (39 msec), a negative area of the anterior chest surface is more enlarged. A minimum exists near the center of the anterior surface. A positive area covers left axillary surface and dorsal surface. A maximum moves a little to the left side.

In the late stage (54 msec), the whole anterior surface is occupied by a negative area and the dorsal by a positive area, a minimum exists on the center of the anterior chest and a maximum on the dorsal.

Standard 12 lead ECGs of this case are presented in Fig. 4.

In order to explain why these maps are obtained, we have used canine cardiac studies in which both maps and epicardial activation sequence studies...
were taken from the same dog.

*Normal canine maps and epicardial activation process:*

Fig. 5 shows a series of normal canine maps (left side) and epicardial activation data (right side), respectively. For illustration of the epicardial activation sequence, the epicardial area through which the activation has passed is shown as a gray area within the heart schema.

In the early stage (7.5 msec), the epicardial activation is not yet observed. In this stage, the activation front probably spreads as a whole towards the anterior. Thus, in the map reflecting the ventricular activation at this instant, a positive area covers nearly the whole anterior chest surface, and a negative the dorsal surface.

In the early middle stage (12 msec), there occurs a breakthrough of the wave front to the epicardium of right ventricular free wall. In the map of this stage, a negative area appears on the right anterior surface. Niche like equipotential lines irregularity are observed. (This irregularity was observed in every case of pre-infarction map between 9 to 13.5 msec after the onset of ventricular depolarization and it might be induced by the breakthrough of the electrical wave front to the epicardium.) A positive area covers the right lower and left anterior surface, and axillary region, and it expands into the lower dorsal area.

In the late middle stage (18 msec), the epicardial excitation is more spread and left ventricular anterior wall has almost excited. In map of this stage a negative area occupies nearly whole of the anterior surface and a minimum exists near the center of the anterior surface. A positive area covers the left axillary region and over half of the dorsal surface. A maximum exists left axillary region.

In the late stage (27 msec), the epicardial excitation of the ventricular anterior wall has almost completed. The excitation mainly lies in posterior wall. In the map of this stage, the whole anterior chest surface becomes negative, and the dorsal positive. A maximum exists on the dorsal surface and minimum on the center of the anterior chest surface.

As mentioned above, referring to the ventricular activation process, we can understand the map pattern easily.

Human map patterns and canine ones are different to each other in details, as there are some anatomical differences, for example, the electrical impulse from the right ventricle has greater influence on the state of the right anterior surface in the dog than in man. And, since human ventricular activation time is about twice that of the dog. However, essentially we find that human maps and canine maps resemble each other. Because the human ventricular activation process resembles essentially that of the canine process,
comparing Durrer's human heart study with Scher and Young's canine heart study, this resemblance is easily understood.

To detect the extent of myocardial infarction through the use of maps:
Among the 15 cases in which the obstruction site was found in the LAD (left anterior descending artery), there were 2 different map patterns (Groups A and B).

Maps of Group A:
Five cases belong to this group. Fig. 6 shows a series of typical Group A maps.
In the early stage (15 msec), a negative area has already occupied nearly the whole of the anterior chest surface and a minimum is also located on the center of the anterior chest surface. Meanwhile, a positive area has occupied the whole dorsal surface and a maximum is located on the dorsal.
In the early (27 msec) and late (39 msec) middle stage, a positive area does not appear on the left anterior surface and left axillary surface. A

Fig. 6
Fig. 6. Typical Group A maps; the case in which the obstruction site was found in the proximal portion of LAD.

Fig. 7
Fig. 7. The standard 12 lead ECGs in the case of Fig. 6.
maximum is not found on the left anterior surface. A negative area continues to occupy the whole of the anterior surface.

In the late stage (54 msec), a negative area occupies the whole of the anterior surface and a positive area, the dorsal surface. A minimum is located on the center of the anterior surface and a maximum the dorsal surface. The map pattern of the late stage is similar to that of normal.

In summary, the characteristics of the Group A maps are as follows; a positive area never appeared on the left anterior and axillary surface, and a maximum never existed there throughout the ventricular depolarization.

It is common cineangiographic findings of this group that the obstruction site is located in the proximal portion of the LAD.

Standard 12 lead ECGs of this case are presented in Fig. 7.

Referring to the cineangiographic findings, we ligated the canine coro-

Fig. 8

Fig. 8. Typical proximal group maps; the case in which proximal portion of LAD was ligated.

Fig. 9

Fig. 9. Typical Group B maps; the case in which the obstruction site was found in the distal portion of LAD.
nary artery near the orifice of the LAD (proximal group). Typical maps of this group are shown in Fig. 8.

We found that the characteristics of these maps are essentially equal to that of Group A maps.

Maps of Group B:

Ten cases belong to this group. Fig. 9 shows a series of typical Group B maps.

In the early stage (15 msec), a negative area occupies the left anterior chest surface, but the area is not so large as in Group A. A minimum exists on the anterior surface and a maximum on the left axillary surface. A part of the dorsal area has been already covered by a positive area.

In the early middle stage (27 msec), a negative area which covers the anterior surface is larger than the normal one. But, unlike Group A, a positive area covers the left axillary surface and a maximum exists there.

In the late middle stage (39 msec), the map pattern is not so different from the normal one, that is, a negative area covers anterior chest surface and a positive area occupies left axillary region and dorsal region. A minimum exists near the center of the anterior surface and a maximum on the left axillary region.

In the late stage (54 msec), as with Group A, the map pattern is similar to the normal one.

In summary, the characteristics of Group B maps are as follows; in the early stage, a negative area appeared on the anterior chest surface. However, unlike Group A, it did not cover all the anterior surface, and in the middle stage, a positive area appeared on the left axillary surface.

It is the common cineangiographic finding of this group that the obstruction site is located in the distal portion of the LAD, excluding the diagonal branch.

Standard 12 lead ECGs of this case are presented in Fig. 10.

Referring to the cineangiographic finding, we ligated the distal portion (after the base of the diagonal branch: distal group) of the coronary artery. Typical maps of this group are shown in Fig. 11. We found the characteristics of these maps are essentially equal to that of Group B maps.

We believed that the map pattern differences were due to the differences of extent of myocardial infarction. Since in the large infarction (proximal group and Group A), most of the anterior and lateral wall are infarcted and can not activate, there is hardly any excitation in the anterolateral wall. In the maps, this abnormality is expressed by the absence of a positive area on the left anterior and axillary surface and lack of a maximum there. Meanwhile, in the small infarction (distal group and Group B), since some of left
anterior wall and lateral wall and apical region are free from infarction, the loss of electromotive force is not so complete and a positive area occupied a part of left anterior surface and a maximum existed there.

Because, as a whole, left anterior wall is activated rather early and in the late stage, the excitation mainly exists only in the posterior wall, the greatest differences of map pattern are observed in the early and middle stage, in the late stage, no significant differences are observed.

The difference of electrical abnormality of the left ventricular anterior and lateral wall is well expressed in the maps. But it is unknown that what degree of difference the maps can obtain, for maps are not the cardiac electrical phenomena itself but is a surface manifestation of it. However, the difference in the extent of infarction on the scale of the data presented in this chapter can be deduced through the maps and therefore it will be useful and, being noninvasive, convenient procedure for clinical examination.
The diagnosis of right ventricular infarction or high posterior infarction remains a difficult problem. We are greatly interested in whether there are consistent characteristic map patterns associated with these infarction or not.

**Detection of right ventricular infarction:**

There are common features of map pattern in which the obstruction site was observed near the orifice of right coronary artery. It may be true that right coronary artery obstruction does not always mean the true right ventricular infarction. However, the obstruction of near the orifice of right coronary is always associated with some involvement of the right ventricle. Five cases belong to this group. Fig. 12 shows typical maps of this group.

In the early stage (15 msec), unlike the normal maps it is worth noticing that the rather larger area of right anterior and axillary surface is occupied not by a positive but by a negative area. A minimum is also located there.

In the early middle stage (27 msec), a negative area occupies most of

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**Fig. 12**

Fig. 12. Typical human maps in which the obstruction site was observed in the right coronary artery.

**Fig. 13**

Fig. 13. The standard 12 lead ECGs in the case of Fig. 12.
the right anterior surface. But the difference is not so remarkable as in the early stage.

In the late middle stage (39 msec) and late stage (54 msec), there are no remarkable difference comparing the normal map with this presented case. Standard 12 lead ECGs of this case are presented in Fig. 13.

We can obtain a similar map experimentally by the ligation of the canine right coronary artery. Fig. 14 shows typical maps of the experimental case.

There are some differences in map patterns which are probably due to the anatomical difference between dog and man, but essentially both maps (Figs. 12 and 14) have common characteristics. That is, in the early stage, a considerable portion of the anterior chest surface is occupied by a negative area and a minimum exists there.

Fig. 14 Fig. 15

Fig. 14. Typical canine maps in which the right coronary artery was ligated.

Fig. 15. Typical human maps in which obstruction site was observed in a branch of the circumflex artery.
This characteristic map pattern must be due to the loss of electromotive force which had existed in the right ventricular free wall, and this electrical abnormality of right ventricular free wall is probably projected to the right anterior chest surface.

In conclusion, maps have unrivalled diagnostic ability compared with conventional ECG, which have only one lead point on the right anterior surface. It has been believed that the right ventricular infarction is rather rare, but if maps had wider use for examination, the incidence of right ventricular infarction might be increased. Without maps, misdiagnosis is likely to occur and therefore suitable management of right ventricular infarction patients are severely hindered.

Detection of high posterior infarction:

Four patients were diagnosed as high posterior infarction by acute phase complaints, physical examination, and serum enzyme (GOT, GPT, CPK) elevation.

Fig. 15 shows a series of high posterior infarction maps.

It may be said that maps of this group are trivial and shows poor change, that is to say, they are non-dynamic. However, one with experience in maps can see an abnormality clearly. That is, in the early stage (15 msec), nearly the whole of the anterior surface is occupied by a positive area and the dorsal by a negative area. This map pattern itself does not indicate any abnormality, however a positive area expands into the dorsal surface so slowly that, in the early (27 msec) and late middle stage (39 msec), a large area of the dorsal surface is still covered by a negative area. One can see that white region occupies most of the dorsal surface.

In the late stage (54 msec), the map pattern itself is similar to normal, but we are impressed by the fact that the positive potentials of the dorsal surface are lower than normal.

In the cineangiographic findings, the obstruction is observed in a branch of the circumflex artery. Standard 12 lead ECGs of this case are presented in Fig. 16.

A branch of the circumflex artery was selected and ligated to induce high posterior infarction. Fig. 17 shows typical maps of this experimental high posterior infarction. As in the clinical case, this experimental high posterior infarction map shows almost no difference between infarction and normal in the early stage (7.5 msec). In the early (13.5 msec) and late middle stage (18 msec), however, most of the area of the dorsal surface has been occupied by a negative area instead of a positive area after infarction. The time when more than half of the dorsal surface changed from negative to positive was 16±4.2 msec (Mean±SD) before infarction, meanwhile it
Fig. 16. The standard 12 lead ECGs in the case of Fig. 15. In this case, lateral wall probably infarcted, too.

Fig. 17. Typical canine maps in which a branch of the circumflex artery was ligated.

was $22 \pm 4.9$ msec after infarction. This delay proved to be significant.

In the late stage (27 msec), the map pattern is similar to normal, but we found that the positive potentials of the dorsal surface were significantly lower than normal.

These map patterns are explained as follows.

Since a considerable part of the posterior free wall is infarcted, the electromotive force which exists in the posterior wall is smaller than that of pre-infarction, the approach of the wave fronts towards the dorsal surface is so impaired that expansion rate of the positive area on the dorsal surface is very slow and positive potentials of the dorsal surface are decreased. In general, it is at the late middle and late stage of the ventricular depolarization that excitation of the posterior wall is greatest and therefore it is at these stages that difference in map pattern is most obvious.

The reason why diagnosis of high posterior infarction is difficult lies in
these points; 1. there is no lead point on the dorsal surface in the standard 12 lead ECGs; 2. the dorsal surface is at a greater distance from the heart than the anterior surface, so the ECG potential recorded on the dorsal surface is reduced and its deflection is usually very small; 3. Q waves, that is, minus deflection of early QRS appear even in recordings of normal hearts, making the determination of abnormal Q difficult; and so on. Maps have many lead points on the dorsal surface, too and obtain the electrical abnormality which is projected to the body surface as an area, not as a point. From the change of transformation of the positive and negative areas, we can infer the electrical abnormality of the posterior wall exactly as well as the anterior wall and right ventricular wall.

As mentioned above, maps are powerful tool in the diagnosis of myocardial infarction. Even with the use of computers, the recording of ECGs from many lead points and displaying of maps remains a difficult problem, but this difficulty leads us to the correct diagnosis, for few lead points provide us poor information. It is true that it is complicated to use maps for routine clinical examination, however, maps have strong advantage, that is, they are non-invasive. There are patients who are suspected of infarction, but conventional ECG does not prove the abnormality clearly. In these cases, if we do map examination, we can determine whether infarction has occurred or not. Furthermore, we suggest that the obstruction site may be ascertained by maps. Of course, in this point, we have to perform map examination on more patients and study whether a relationship between map pattern and double or triple vessel disease can be recognized or not.

Maps are made by electrical events within the heart which are projected to the body surface. Maps are not made by electrical events within the heart itself. It may be said that maps are like a shadowgraph. Even taking this limitation of maps into consideration, we can expect that it is useful and sometimes indispensable for some patients to have map examinations.

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