Experimental Basis and Clinical Application of Body Surface Isopotential Map**

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It is important in the diagnosis of cardiac disease to obtain information on the electrical event of the heart. Twelve lead electrocardiogram (ECG) and Frank lead vectorcardiogram (VCG) have been used to accomplish this for decades. However, these methods have few lead points and consequently electrical information from some important body surface region may be ignored. To overcome this drawback, a system has been devised in which the isopotential lines are drawn at each instant of the cardiac cycle based on electrocardiograms recorded from a large number of lead points on the body surface. This new method is known as body surface isopotential maps (MAP). This overview summarizes the clinical and experimental studies with the MAP method performed in our institute.

MAPs were recorded using a mapping system developed in our laboratory. Eighty-seven unipolar electrocardiograms were recorded simultaneously with reference to Wilson's central terminal. These electrocardiographic data were digitized by A/D converter, processed by microcomputer, and MAPs were displayed on a CRT together with ECG and VCG. The basic principles of construction of isopotential lines for the shaded region in the upper portion of Figure 1 are shown in the middle portion of the figure. On the left are 9 electrocardiograms recorded from the region. In order to make MAPs at a given instant of the cardiac cycle, potential values at the instant were calculated for each lead point, and these values

were plotted in digital form (middle). Based on the digital data, isopotential lines are at an obtained interval (right). These procedures are performed for all the areas of the chest surface to obtain MAPs at a given instant as shown in the lower portion.

Figure 2 shows MAPs of a normal subject in

Fig. 1. Principles of construction of body surface isopotential maps.

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Fig. 2. Body surface isopotential maps of a normal subject.

early, mid and late phases of QRS. The shaded area represents the positive potential and white area the negative one. The highest positive potential (maximum) is shown by plus (+) sign, and the lowest negative potential (minimum), by a

minus (-) sign. The activation front in the ventricle is schematically shown based on Durrer's study on the right.

In the early phase, most of the anterior chest is covered by positive potential with a maximum and the back by negative potential with a minimum. This potential distribution represents the activation front proceeding anteriorly in the interventricular septum. In the mid phase, a small portion of the negative potential protrudes into the positive area in the precordium, creating a minimum. The protrusion and minimum is called "niche" and "breakthrough minimum," respectively. This phenomenon is considered to indicate the arrival of the activation front on the epicardial surface of the right ventricle. Corresponding to the last portion to be activated in the ventricle, that is, the posterobasal portion of the left ven-

Fig. 3. Body surface isopotential maps of right bundle branch block.

Fig. 4. Body surface isopotential maps of WPW syndrome.

tricle, positive potentials appeared on the back in the late phase.

In a clinical study of our laboratory, MAPs of 28 patients with complete and incomplete right bundle branch block could be divided into 3 types (Figure 3). In Type I, the MAP pattern was near normal in the early phase of QRS. At 40 msec, a "niche" appeared over the left precordium. It was more to the left and appeared considerably later than in the normal MAP. In the late phase, a positive area widely covered the entire right anterior chest. This MAP pattern indicates significantly delayed activation throughout the right ventricle. Similar MAP findings were observed after complete interruption of main right bundle in dogs. In type II, the "niche" appeared at 40 msec on the left anterior chest in the mid phase, and positive potential was observed in the late phase mainly on the right upper anterior chest. Experimental incision of the false tendon of the right bundle branch showed similar MAP findings. Type III was very close to the normal except for a positive potential with a maximum on the anterior upper chest in the late phase. This indicated that Type III has its activation delay restricted to the outflow tract of the right ventricle.

MAPs were recorded from 21 patients with WPW syndrome, and classified into 3 types (Figure 4). In Type I, the positive area covered the anterior chest and the negative area covered the posterior chest during the entire QRS phase, suggesting

that the location of early excitation was at the posterior aspect of the left ventricle. In Type II, the positive area covered the left chest and the negative area covered the right chest during the entire QRS phase, suggesting that the early activation site was at the lateral wall of the right ventricle. Type III showed a map pattern similar to Type II in the early and mid phases of QRS. In the late phase, however, the positive area covered the upper part of the anterior chest. These findings indicated that the region of early activation was located in the vicinity of the ventricular septum within the posterior aspects of right ventricle. Our experimental studies revealed that preexcitation of these estimated early activation sites showed map findings similar to the respective clinical cases.

MAPs were examined in 54 patients with old myocardial infarction. They had an evident history of the infarction and showed evident ECG findings of the infarction. Figure 5 shows MAPs of anterior, inferior and posterior infarctions for the early, mid and late phases of QRS. Anterior infarction was characterized by a widespread and deep abnormal negativity observed on the anterior chest during early and mid phases. In inferior infarction, abnormal negative area was observed on the lower chest during early and mid phases. Posterior infarction was characterized by an abnormal negativity on the back during mid and late phases.

In order to detect a significant surface potential

Fig. 5. Body surface isopotential maps of anterior, inferior and posterior myocardial infarctions.

Fig. 6. Difference maps of anterior, inferior and posterior myocardial infarctions.

Fig. 7. Body surface isopotential maps of anterior myocardial infarction undetectable by 12 lead electrocardiograms.

defect due to myocardial infarction, difference MAPs were made by subtracting MAPs of normal subjects from MAPs of patients for each instant of the cardiac cycle (Figure 6). The shaded area in the difference MAPs represents the area of surface potential defect due to infarction. The "difference" was observed on the anterior chest during early to mid phases in anterior infarction, on the inferior chest during the same phases in inferior infarction, and on the back during mid to late phases in posterior infarction. The location of the surface potential defect corresponded well to the location of the infarction in the heart. The time at which the potential defect appeared corresponded well to the time at which the myocardium infarcted originally activated before

infarction. These findings suggested that the location and extent of myocardial infarction could be detected by inspection of serial difference MAPs.

MAPs were recorded in 53 patients who had an evident history of infarction but showed no ECG findings of the infarction. Maps of anterior infarction undetectable by ECG were characterized by an abnormal minimum located on the anterior chest in the early phase of QRS (Figure 7). Anterior infarction undetectable by ECG was distinguished most clearly from normal subjects by the position of the minimum at 15 msec (Figure 8). Twenty-four of 30 patients with infarction showed the minimum on the anterior chest, while 29 of 30 normal subjects did so on the back. On the

Fig. 8. Position of a minimum at 15 msec in anterior myocardial infarction undetectable by 12 lead electrocardiograms and normal subjects.

other hand, MAPs of old inferior infarction undetectable by ECG were characterized by an abnormal minimum located on the lower chest in the early phase. These findings suggest that MAPs can diagnose the presence or absence of old myocardial infarction more accurately than

by ECG or VCG.

ST mapping with 30 precordial matrix electrodes has been used in 17 patients for clinical assessment of the area, size, and prognosis of acute myocardial infarction. An algebraic sum in mV of the ST elevation obtained from all the electrodes was defined as Σ ST. The number of electrodes where the ST elevation exceeds a certain voltage was defined as NST. Σ ST and NST correlated well with the size of myocardial ischemia detected by ^{201}Tl myocardial scintigram with a correlation coefficient of 0.66 and 0.60, respectively. Effects of drugs on Σ ST and NST were examined in experimental and clinical studies. Experimental study demonstrated that propranolol reduced the area of ST elevation (NST), whereas isoproterenol increased it. Clinical studies revealed that administration of nitroglycerin reduced Σ ST as well as pressure rate product. These findings suggested that ST mapping was available for the assessment of ischemic size and selection of drug treatment in acute myocardial infarction.

Table 1 summarizes our clinical applications of MAP. This paper demonstrated its clinical usefulness and experimental basis for ischemic heart disease, conduction disturbances and WPW syndrome. However, there are many other cardiac

Table 1. Clinical application of body surface isopotential mapping

1. ISCHEMIC HEART DISEASE	
Old myocardial infarction	: Location and extent, Presence or absence
Acute myocardial infarction	: Size of ischemic myocardium
Angina pectoris (exercise test)	: Extent of ischemic myocardium, Time course
2. CONDUCTION DISTURBANCE	
Right bundle branch block	: Differential diagnosis of central and peripheral block
Hemiblock	: Conduction of left anterior or posterior division
3. HYPERTROPHY	
Ventricular hypertrophy	: Degree of hypertrophy
Hypertrophic cardiomyopathy	: Location of hypertrophy
4. WPW SYNDROME	
Location of accessory pathway	
5. ARRHYTHMIA	
Origin of ventricular arrhythmia	
Prediction of arrhythmia leading to sudden death	

Table 2. Prospects and problems of body surface isopotential mapping

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1. Number and location of lead points
 2. Diagnostic criteria
 3. Automated diagnosis
 4. New lead system
 5. Low potential
 6. Inverse problem
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abnormalities to which MAPs can be applied. MAPs can be used to diagnose the location and severity of hypertrophy in hypertrophic cardiomyopathy and to detect the origin of ventricular

premature beat or ventricular tachycardia.

Table 2 indicates the prospects and problems concerning MAP. Although controversy has arisen as to the number and location of lead points, widespread clinical use of MAP requires a more convenient, limited lead system. Broad-based collection of data serving as the map base is expected to result in the determination of diagnostic MAP criteria for various heart diseases and successful automated MAP diagnosis. Specific diagnostic MAP findings may contribute to the establishment of a new lead system different from ECG or VCG. A large amount of MAP spatial data may permit detection of low potential in the heart including His bundle potential, and to obtain detailed information on electrical activity in the heart by solving the inverse problem.