Body Surface Mapping During Percutaneous Transluminal Coronary Angioplasty

QRS Changes Indicating Regional Myocardial Conduction Delay

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Using a radiotransparent electrode array, body surface maps (BSMs) were constructed based on simultaneous recordings from 62 leads on the entire thorax before, during, and after balloon inflation during percutaneous transluminal coronary angioplasty (PTCA). Twenty-five patients were studied, and 30 angioplasties were performed; 20 patients had one-vessel disease, and five patients had two-vessel disease. In total, 15 dilations in the left anterior descending artery (LAD), seven in the right coronary artery (RCA), and eight in the left circumflex artery (LCx) were studied. For each patient, the BSM and the QRS integral map before, during, and after the inflation was compared by subtraction of recordings “during-minus-before” inflation and “before-minus-after” inflation. The subtraction was performed on the results of the QRS integral maps. The conclusions derived from the inspection of the BSMs and the difference maps show specific changes in the QRS complex during ischemia related to the corresponding ischemic segment in 21 of 25 patients in the three groups. An area of positive potentials remained present on the BSM during dilation, indicating a depolarization wave front. For the LAD group, positive potentials were seen on the anterior thorax and, for the RCA group, on the lower part of the thorax. By subtraction analysis, these changes were extracted and presented as difference maps. For the LCx group, the BSM revealed no changes in pattern but the difference map showed a difference vector pointing in an anteroposterior direction. A regional myocardial conduction delay was hypothesized as the most likely cause for the results. (Circulation 1990;81:840–849)

Controversy exists about the changes in the QRS complex occurring during ischemia.1–4 The analysis of changes in the QRS complex during ischemia predominantly regards the R wave and both a decrease and increase in R wave amplitude have been described.1–4 Intramyocardial conduction delay and changes in ventricular cavity size have been associated with the cause of changes in R wave amplitude.

Body surface mapping (BSM) provides more surface electrocardiographic data than the standard electrocardiogram (ECG) leads and can be used to study the ischemic changes occurring during inflation of the balloon catheter in a percutaneous transluminal coronary angioplasty (PTCA) procedure.5,6 Electrocardiographic techniques have been used to examine the changes in the ST segment5–11 as well as the R wave13 during PTCA. In this study, the QRS changes recorded on the body surface during PTCA were analyzed with regard to temporal, voltage magnitude and spatial information content. On the basis of our results, we postulate that specific changes in the QRS complex occur on the body surface and show regional correlations with the ischemic segment in the heart.

Methods

Table 1 shows the clinical characteristics of the patients that underwent elective PTCA and were used in this study. None of the patients had a previous myocardial infarction, bundle branch block, or ventricular hypertrophy. All patients had a normal 12-lead ECG in rest before the angioplasty procedure. The PTCA procedure was performed accord-
An Electrode Grid reference location for the electrode spacing of the chest. The image the thorax that are applied vertically on the front and back of the thorax (Figure 1). The minimal vertical interelectrode spacing is 33 mm; the horizontal spacing varied with the size of the patient's chest. Lead V₅ served as reference location for the application of the straps. An irregular grid was chosen, of which 42 leads are based on the two 32-lead sets introduced by Lux et al.¹⁸ These lead configurations are derived from a 192-lead grid. The remaining 20 leads were positioned according to optimal lead sets from other investigators¹⁹–²² and include V₁ to V₆.

**Electrode Grid**

The electrodes are radiotransparent,¹⁶,¹⁷ which allows application without interfering with the x-ray image of the chest. The radiotransparent electrode array consists of a set of 14 flexible electrode straps that are applied vertically on the front and back of the thorax (Figure 1). The minimal vertical interelectrode spacing is 33 mm; the horizontal spacing varied with the size of the patient's chest. Lead V₅ served as reference location for the application of the straps. An irregular grid was chosen, of which 42 leads are based on the two 32-lead sets introduced by Lux et al.¹⁸ These lead configurations are derived from a 192-lead grid. The remaining 20 leads were positioned according to optimal lead sets from other investigators¹⁹–²² and include V₁ to V₆.

**Figure 1.** Diagrams showing location of 62 electrodes on thorax.

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**TABLE 1. Characteristics of the Patient Group**

<table>
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<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age (yr)</th>
<th>Location of stenosis</th>
<th>Proximal/distal</th>
<th>Collat</th>
<th>Duration of dilation</th>
<th>Percentage narrowing</th>
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<td>p</td>
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</table>

LAD, left anterior descending coronary artery; LCx, left circumflex artery; RCA, right coronary artery.
The sixth column refers to absence or presence of angiographically proven collaterals.
Data Acquisition

With a portable BSM unit, 62 unipolar ECGs were simultaneously recorded using a sampling frequency of 500 Hz. Wilson central terminal was used as reference. The equipment consisted of an Apple IIe computer with an eight-bit analog-to-digital converter. For each recording, the input sensitivity could be adjusted, ranging from ±0.6 mV to ±5.0 mV. Sixty-four high-quality monolithic alternating current (AC) amplifiers were used to amplify the electrocardiographic signals. The international safety standards were met by isolating the whole system from the main power supply by means of an isolation transformer. The internal memory of the computer was expanded to 1 MB, which allowed for 16 successive recordings of 1.5 seconds that were stored on floppy disks.

Baseline adjustment was done afterward by choosing a point before the P wave and a point after the T wave in one reference channel as the time instant of the zero potential for all the 62 channels. Signals of bad quality, that is, signals with baseline drifting or with a high noise content were rejected and replaced by estimated values based on surrounding leads. In 66% of the recordings used in this study, no leads had to be rejected, and the recordings in which more than three leads had to be rejected were not included in this study.

Data Analysis

Data were transferred to a PDP 11/73 minicomputer (Digital Equipment) equipped with an electrostatic plotter. First, the 62 irregular array was transformed into a regular 192 array and, by linear interpolation, the data were depicted as BSMs. On the BSM, equal positive potentials were connected by isopotential lines, negative potentials by dashed isopotential lines, and the zero potential by a dotted line. Variable incremental steps between the isopotential lines were automatically chosen to remain below a maximum of seven isopotential lines between the potential extrema and the zero potential. The BSM sequence before, during, and after dilation throughout the QRS complex was visually interpreted for each case.

From the PDP 11/73, the data were transferred to a VAX 11/750 computer on which quantitative analysis was performed. For all 62 leads, the integrals of the QRS complex were computed and stored in the irregular 62-point matrix. From this matrix, a schematic representation of the thorax was drawn in which the values of the QRS integrals were displayed by isointegral lines using the same technique. Thus, three QRS integral maps were obtained before, during, and after dilation for each PTCA procedure. These matrices were further used for a subtraction analysis. The matrices of the 62 integral values from the recordings “before” and “after” dilation were subtracted from the integral values “during” dilation, and the subtraction of the integrals from “after-minus-before” dilation were computed to serve as a control. The subtraction of the two matrices of 62 QRS integrals results in one matrix of 62 difference values that are depicted as a difference map in the same way the integral map and the BSM are computed. For each dilation, a difference map “during-minus-before,” “during-minus-after,” and “before-minus-after” dilation was calculated. Next, a mean QRS integral map and a mean difference map from during-minus-before and before-minus-after dilation was computed by summation of the matrices with integral values or difference values divided by the number of subjects for the left anterior descending coronary artery (LAD), the right coronary artery (RCA), and the left circumflex artery (LCx) groups. The matrix derived of averaged values was then depicted as a mean map. To establish the consistency of each individual difference map with the mean difference map of the LAD, RCA, and LCx groups, a further analysis was performed. Three independent experienced observers classified the during-minus-before difference map of each dilation into one of the three groups by comparing the individual results with the three mean difference maps of during-minus-before dilation. A similar procedure was performed with a computer algorithm. The algorithm used to determine the correlation coefficient is described by Mirvis. For each of the individual difference maps, the correlation coefficient with the three mean difference maps was calculated. In Figure 2, these various steps are summarized. The first stage consisted of visual inspection of BSMs in which the changes in pattern during dilation were compared with before and after dilation. The next stage consisted of a reduction of temporal information by computation of the QRS integral maps, followed by a comparison of voltage magnitude information in the

![Figure 2. Schematic sequence in which analysis of data was performed.](http://circ.ahajournals.org/)
QRS integral maps before, during, and after dilation by the subtraction analysis.

The last stage of the analysis was the classification test. The offset of the QRS complex was visually determined and an 80-msec interval, from ST20 to ST100, was used to calculate the ST integral and presented with the same method as the QRS integral. The mean ST integral maps for each of the three groups were calculated before and during ischemia.

Noise

The interpretation of the subtracted integral maps is hampered by the noise introduced into the recording procedure and the analysis techniques. The use of the PTCA as a clinical model for ischemia offers the advantage of minimizing the physiological noise. All patients had tidal volume respiration, which has a very small influence on the surface potentials.27 Because all patients remained in the supine position without motion artifacts, throughout the procedure, a beat-to-beat analysis could be performed. The QRS duration did not alter in 80% of the subjects, which is in agreement with recent findings of Selvester et al.28 In five subjects, a small change in QRS duration occurred (less than 3 msec). The durations of the QRS complexes before, during, and after dilation for these five patients were taken equally long to perform the subtraction analysis. The electrode grid remained in the same position throughout the procedure, facilitating the subtraction analysis. The onset and offset of the QRS complexes before, during, and after dilation were visually determined on the BSM. Within the limits of our sampling frequency, a possible error of 2 m sec at the onset and 2 m sec at the offset of the QRS complex and an error caused by the occasional noise might be introduced. This error, however, is negligible because the integral of the QRS complex is primarily determined by the part after the onset and before the offset.

Shielding of all cables and a driven right leg circuit29 suppressed the influence of the MAINS frequency interference in the recordings. Thus, the total noise of preamplifiers and electrodes, and interference remained in the order of one bit (27 µV top-top). This noise and the quantisation noise is calculated to be below 0.4 mVmssec for the integral maps and 0.6 mVmssec for the difference maps (The number of samples is approximately 40 for each recording.). These noise figures are calculated, ignoring the influence of the preprocessing, that is, the baseline definition. Taking this into consideration, the noise level of the difference maps is estimated to be 2 mVmssec. The carbon electrodes16 have a negligible noise of less than 5 µVRms in the frequency bandwidth of interest. Baseline stability improved within 2 minutes after electrode application on the patient’s chest and remained stable throughout the procedure.

The interpretation of the individual results of the subtraction of integral maps was performed to differentiate between artifact and physiological information. In the group of difference maps before-minus-after dilation, an irregular pattern was noted in the majority of the cases. The impression of an irregular pattern is the fact that the observer can track down the individual electrode locations used in our grid. The signals of most electrodes is in the order of the estimated noise values (2 mVmssec). In contrast, the during-minus-before subtraction integral patterns reveal a group of electrodes in which the signal has changed, giving the observer the impression of a pattern. Whether a pattern was information or artifact was judged in the last instance by the observers.

Results

Body Surface Maps

In Figure 3, three examples of sequences of BSMs of a dilation of the LAD (left panel), the RCA (middle panel), and the LCx (right panel) are shown. Each pair of maps has been selected on corresponding time instants that are indicated in the V1 lead by each sequence. In the LAD group, the major difference in pattern occurs at the end of the QRS complex. An area of positive potentials of a larger magnitude is present in the fourth BSM of the sequence during dilation. At the fifth BSM, the signal of the anterior thorax is not negative before dilation, whereas a positive area remains present during dilation. The voltage magnitude of this small area of positivity decreases during the terminal part of the QRS complex, reflecting the last stage of depolarization of the anterior part of the left thorax. The BSMs after dilation are identical with the ones obtained before dilation, indicating that the change found during ischemia is reversible. In the third BSM of the RCA example (Figure 3, middle panel), a larger area of positive potentials in the lower part of the thorax is present during dilation. This is more clear in the fourth BSM. The area of positive potentials of decreasing magnitude at the lower right side of the back reflects depolarization of the inferior wall. The reversibility of the process was confirmed by the BSM sequence after dilation, which revealed no differences with the BSM before dilation. In the example of the LCx group (Figure 3, right panel), no clear change in pattern was observed in this subject nor in the other subjects of this group. The magnitude in negative potentials at the later part of the QRS complex on the anterior thorax, however, is larger in the maps during dilation. Localization of the ischemic area is, therefore, not possible on the basis of changes in pattern but in altered voltage magnitudes of BSMs on similar time instances during depolarization before and during ischemia. The number of patients who showed either a change in pattern or a change in voltage magnitude is reflected by the results of the classification test in Table 2.

Integral Maps

In Figure 4, the mean QRS integral maps from before and during dilation are represented for the 15 LAD, the seven RCA, and the eight LCx patients. In
the LAD group and the RCA group, a distinct change in pattern occurs on the lower anterior and posterior thorax. This region displays more negativity in the LAD group and more positivity in the RCA group during dilation. In the LCx group, the patterns remain unchanged. The most pronounced difference is the increased magnitude of positivity in the LAD group and negativity in the RCA and LCx groups.

In Figure 5, the mean integral maps of the ST segment are shown for the three groups before and during dilation.

**Difference Maps**

The mean difference maps derived from the subtraction of the QRS integrals before-minus-after dilation and during-minus-before dilation for each of the three groups are shown in Figure 6. In the first row, the mean QRS difference maps before-minus-after dilation are depicted. Each group features an irregular pattern with low values. In contrast, the mean QRS difference maps during-minus-before dilation display a clear pattern with values considerably higher than the values of the
control. The patterns of the LAD and the RCA group means are consistent with the changes in patterns of the individual subjects shown in Figure 3. The mean difference pattern of the LCx group is not found in the sequential change of Figure 3. Therefore, the mean difference map of the LCx group results from a change in voltage magnitude as is demonstrated in the integral map of Figure 4. Interpretation by observers of BSMs or integral maps of the LCx group is difficult. The difference map features the change in voltage magnitude during ischemia and presents this change as an image of the ischemic region, facilitating the interpretation.

**Classification Test**

To validate the mean difference maps with regard to each individual difference map, a classification test was performed. The QRS integral difference map of each patient was classified into one of the three mean difference maps by three independent observers and by using a computer algorithm. The results indicate that an accurate classification is possible in the LAD group and the RCA and LCx groups. With inferior ischemia, either by occlusion of the RCA or the LCx, however, a discrimination between the two dilation sites proves to be difficult.

Four dilations in patients with angiographically proven collaterals are excluded from the results shown in Table 2. Two patients (patients 8 and 11)

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**TABLE 2. Results of Classification Test**

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<tr>
<th>QRS</th>
<th>LAD (n=14)</th>
<th>RCA (n=7)</th>
<th>LCx (n=5)</th>
<th>RCA+LCx (n=12)</th>
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<tbody>
<tr>
<td>Three observers</td>
<td>41/42</td>
<td>15/21</td>
<td>9/15</td>
<td>35/36</td>
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<td>Computer</td>
<td>14/14</td>
<td>5/7</td>
<td>3/5</td>
<td>12/12</td>
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</table>

For each group, number of accurate classifications and total number of observations made are shown for observers and computer. In last column, RCA and LCx groups have been combined into one group. Patients 12, 21, and 24 were excluded. LAD, left anterior descending coronary artery; RCA, right coronary artery; LCx, left circumflex artery.
showed signs of ischemia even with collaterals and were not excluded. In one patient (patient 12), a totally occluded LAD with angiographically proven collaterals and a stenosis in the LCx showed no change in pattern during occlusion of the LAD and the typical LAD pattern during occlusion of the LCx. This case was consistently misclassified by all the observers. Furthermore, two of the eight patients (patients 21 and 23) in the LCx group had collateral blood supply. No changes were observed in the BSMs, and consequently, the subtraction patterns revealed no sign of ischemia. These two cases with patient 12 were excluded from the classification results.

**Discussion**

In recent years, many investigators have recognized the PTCA procedure as an excellent model for the study of acute ischemia. The techniques that have been used for this purpose are predominantly echocardiography and electrocardiography. In these studies, it seemed that the first sign of ischemia after balloon inflation was wall motion abnormalities, followed by ST segment changes several seconds later, and angina pectoris as the last sign. The number of leads in these studies, however, was limited, and the changes in the QRS complex during ischemia were not taken into consideration.

In this study, body surface mapping was used to examine the changes in the QRS complex on the surface ECG during ischemia. The advantage of BSM methods over the standard ECG procedure are the extensive techniques available for quantitative analysis. Instead of regarding the changes in R wave amplitude, which is a practical landmark in the scalar ECG, a more extensive analysis of the recorded data is possible. The changes in voltage magnitude and temporal and spatial information can be regarded in combination with each other or separately.

Our data show that specific changes in BSM pattern in the QRS complex as well as in the ST segment occur during anterior and inferior ischemia and return to normal after reperfusion. This reversible change in BSM pattern of the QRS complex can be understood when a slowing of conduction in the ischemic segment is hypothesized. The basis for this
hypothesis is found in the sequence of the BSMs that, in contrast to the integral and difference maps, still contains the temporal information. The leads overlying the precordial thorax (in anterior ischemia) and the lower part of the thorax (in inferior ischemia) show positive potentials during occlusion at an instant during the depolarization when these areas have already become negative before or after the ischemia. Because these positive potentials are decreasing in voltage magnitude until the ST segment is reached, they originate from a depolarization wave front. With repolarization forces, an increase in voltage magnitude and, with an injury current, a constant voltage magnitude is to be expected. These specific body surface areas correspond with the ischemic areas in the heart. Thus, even with a relatively short period of ischemia, we find in most of the studied cases that depolarization of the anterior or inferoposterior part of the heart takes longer during ischemia than before or after ischemia. Consequently, we believe that the subtraction patterns reflect this slowing of conduction in the ischemic segment. One can consider whether the injury current present in the ST segment influences the changes we found in the QRS complex. Although the ST integrals of Figure 5 have a similar dipolar pattern as the subtraction results of the QRS complex, this does not entirely explain the areas on the thorax with decreasing voltage magnitudes. When ST elevations are recorded using AC amplifiers, no distinction can be made between "true" ST elevation caused by systolic injury currents, TQ depression caused by diastolic injury currents, or a combination of both. If, in AC recordings, there is no difference in the level of TQ and ST segments, however, the presence of a diastolic injury current can be excluded.33

Several authors6,13,34–36 have studied the R wave changes during the occlusion of a coronary artery. A biphasic change, that is, a decrease of R wave amplitude in the first phase of ischemia followed by an increase of R wave amplitude in the later stages of ischemia, was found. The cause of this biphasic change is attributed to local accumulation of extracellular potassium, enhancing the conduction velocity in the first minute of occlusion with slowing of conduction caused by deterioration at several minutes of ischemia. There seems to be a large variability in the moment at which the R wave starts to increase, probably caused by the absence or presence of collaterals.13 The recordings analyzed in this study are at 40–60 seconds of balloon inflation, and profound alterations in the QRS complex were seen in the majority of the cases. Because only recordings at maximal ischemia have been used, the biphasic change is not taken into consideration in this study. With regard to the discrepancy in time of occurrence of the regional conduction delay in the present study and the time found in animal experiments, where a decrease in conduction velocity only occurs at 2–3 minutes of ischemia,37 it should be noted that the occlusion in animal experiments is sudden, whereas during PTCA, the occlusion is gradual. Insertion of the guiding wire and, later, the uninflated balloon catheter in an already narrowed vessel causes a further reduction of lumen diameter. Animal experiments have shown that the increase of concentration of extracellular potassium starts at a higher level when occlusion was preceded by a period of low flow.38

Additionally, the patients with angiographically proven collaterals and no obvious ischemia did not show this phenomenon. In the patient with an occluded LAD and a stenosis in the LCx showing anterior ischemia during a dilation of the LCx, a plausible explanation might be found in a steal effect from collateral blood supply of the RCA to the inferior wall, and consequent anterior ischemia,39,40 as well as the obvious cut off from collateral blood supply from the occluded LCx artery.

The data from the subtraction analysis of the recordings during-minus-before occlusion show a distinct pattern for anterior and inferior ischemia. Although the temporal information is lost by this technique, these results accentuate the change in the overall depolarization sequence. The difference consists of an anteriorly and leftward-directed difference vector for anterior ischemia and an inferiorly directed vector for inferior ischemia. This finding is confirmed by a study from Barnhill et al.36 In their study, the QRS complexes during angina pectoris attacks by five patients with Prinzmetal angina were subtracted from the QRS complexes at rest, resulting in similarly oriented difference vectors for both anterior and inferior ischemia. Further investigation in dogs showed that these difference vectors could be attributed to a conduction delay in the ischemic segment.

Although a discrimination between ischemia caused by occlusion of the RCA and occlusion of the LCx proved to be difficult, the mean difference map showed a subtle difference. In the RCA group, the mean difference map shows negativity on the left upper side of the thorax and positivity on the lower right side of the thorax. The difference vector, therefore, points from the left shoulder area to the right lower flank. In the LCx group, the entire anterior thorax is negative with positivity on the back and left lower flank, indicating a difference vector from the anterior thorax pointing to the left side of the back. The direction of both difference vectors suggests a relation between the respective ischemic segments. Although these difference vectors point clearly in a different direction, the two-dimensional representation of the thorax causes a loss of this three-dimensional phenomenon. The patterns of the QRS subtraction before-minus-after occlusion have, for the three groups, a low value. We believe that these patterns reflect the total noise introduced into the experimental protocol. That these patterns contain physiological information about the status of the heart seems unlikely. The clear difference in QRS pattern of the before-minus-after and the during-
minus-before analysis with the corresponding values indicates a reversible change reflecting the regional ischemia.

The mean ST integral maps, during dilation, show a dipolar pattern with vectors similarly directed to the ischemic area.

Clinical Implication

The results of this study show that the QRS complex contains information about the presence and location of regional ischemia. To extract this information, recordings before, during, and after balloon inflation were needed. The corresponding clinical setting in which recordings before, during, and after regional ischemia are analyzed is the exercise ECG. The results of this study, however, cannot be extrapolated entirely to exercise ECG because the ischemia induced by exercise often is of subendocardial nature, whereas the ischemia induced by sudden occlusion of a main branch of a coronary artery is transmural. The transmural ischemia we have studied causes a change in voltage amplitude and a change in temporal information within the QRS complex. We have shown that both changes are spatially related to the area of ischemia and that the physiological explanation probably can be found in a conduction delay of the ischemic segment. The analysis of the QRS complex during exercise ECG comprises the change in voltage magnitude and, specifically, the R wave.1–4 Our data reveal no change in total QRS duration but a temporal change occurring within the QRS complex. Furthermore, profound changes in voltage magnitude are seen at the later stages of depolarization. Assuming that changes in the QRS complex found in exercise-induced ischemia have the same physiological basis, more information about the presence and location of ischemia can be extracted from the QRS complexes recorded during exercise ECG. A more extensive use of voltage magnitude changes and temporal changes within the QRS complex might lead to a better diagnostic performance of the exercise ECG.

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References


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