Signal-Averaged Body Surface Mapping for the Assessment of Low-Amplitude Potentials

Relation between Ventricular Depolarization and Repolarization in Normal Subjects

Takehiko SHIBATA, M.D., Isao KUBOTA, M.D., Kozue IKEDA, M.D., Michiyasu YAMAKI, M.D., Kanji HANASHIMA, M.D., Kai TSUIKI, M.D., and Shoji YASUI, M.D.*

SUMMARY
To examine the relation between ventricular depolarization and repolarization, body surface isopotential maps at the end of the QRS complex were studied in 32 normal subjects using a signal-averaged body surface mapping system. The number of beats averaged was 96–154 (mean 126.2). In this study, there were 8 types of isopotential map patterns at the end of the QRS complex. Mean±SD of QRS duration, appearance time of repolarization, and disappearance time of depolarization were 82.0±8.7 msec, 71.8±10.5 msec, and 79.7±9.4 msec, respectively. Time duration of overlapping depolarization and repolarization was 8.6±6.4 msec. The early repolarization was widely distributed on the left anterior chest and the upper sternal region. These results demonstrated the difference between the appearance time of repolarization and the disappearance time of depolarization for each lead. We concluded that it is difficult to evaluate ECG waves in the terminal portion of the QRS complex with the dipolar theory only.

Additional Indexing Words:
Signal-averaging Body surface mapping Ventricular depolarization Ventricular repolarization

RECENTLY, signal-averaged electrocardiograms (ECGs) have been used for the detection of late potentials.1)–4) On the other hand, body surface isopotential mapping is well known as a method to detect regional elec-
We thought that the introduction of the signal-averaging technique would enhance the utility of body surface isopotential maps. In this report, we developed an 87-lead signal-averaged body surface low-amplitude isopotential mapping system and studied the body surface distributions of low-amplitude potentials at the end of the QRS complex in normal subjects. In addition, the information obtained between ventricular depolarization and repolarization was studied in detail.

Methods

Subjects:
Thirty-two subjects, ages 18–56 years (28 males and 4 females, mean 27.8 years), were studied. Clinically, all had normal hearts as defined by medical history, physical examination, standard 12-lead electrocardiogram, and echocardiography. None of the subjects was taking medication at the time of recording.

Recording-methods:
Body surface mapping was performed with 87-unipolar lead body surface electrocardiography (VCM-3000 system: Fukuda Denshi Corporation, Tokyo) for 8 sec. The sampling data for 8 sec were recorded on a 1 MB floppy disk. This procedure was repeated 20 times. The VCM-3000 system consisted of amplifiers that had a frequency response of between 0.05–500 Hz, an analog-to-digital (A/D) converter (12 bit samples, 5 μV/bit), and a sampling interval of 1 millisecond. For the purpose of recording the entire QRS complex by a 12-bit A/D converter, the resolution of our system was adjusted to 5 μV (range ±10 mV). Electrocardiographic signals were recorded from 87 disk electrodes placed on the anterior (59 electrodes) and posterior (28 electrodes) thoracic surfaces (Fig. 1). All potentials were measured with Wilson's central terminal. Subjects were kept relaxed in the supine position. Their basal heart rates varied from subject to subject during recording. They ranged from 51–82 beats/min (mean 64.3 beats/min). Data recorded on 20 floppy disks were transferred to a 16-bit personal computer (PC-9801 VX4: NEC, Tokyo) and stored on a hard disk unit. The stored data were copied on a 40 MB magnetic tape and later analyzed. Analyzed data were printed by a laser printer.

Data analysis:
Data were analyzed with a PC-9801 VX4. We selected a template beat at a precordial lead (lead G4) for the processing of signal-averaging and
Fig. 1. Electrode sites on the body surface. Eighty-seven lead points were arranged lattice-like (13×7 matrix), except for 4 leads in both midaxillary lines, and covered the entire thoracic surface. Eighty-seven electrodes were placed along 9 columns (B-H) on the anterior chest and 4 columns (J-M) on the back. Columns A, E, and I were positioned on the right midaxillary, midsternal, and left midaxillary lines, respectively. Columns B-D and F-H were evenly spaced between columns A-E and E-I, respectively. Column J was located so as to make the distance between columns I and J equal to that between columns H and I. Column M was located so as to make the distance between columns M and A equal to that between columns A and B. Columns K and L were evenly spaced between columns J and M. Lead points E4 and E6 were located on the second and fifth intercostal space, respectively. Dots indicate the location of the standard V1 to V6 leads.

all subsequent beats were tested against the template. A 75% point of the amplitude of maximal dV/dt (V: potential) in the template beat (X) and that in a successive beat (X') were selected as the approximate alignment point. We moved X' from (X−10) msec to (X+9) msec (total 20 points). Then, 20 correlation coefficients between the template beat and a subsequent beat were calculated using 160 pairs of potential data from (X−75) msec to (X+84) msec. Time alignment and signal-averaging were performed when the highest correlation coefficient between X and X' was attained. A beat was excluded from the averaging when the highest correlation coefficient was less than 0.990, or when the coupling interval of the beat changed more than 20% compared with that of the template beat.

**Noise levels by signal-averaging:**

Signal-averaging was used to increase the signal-to-noise ratio. Figure 2 shows the effect of signal-averaging on the improvement of signal-to-noise ratio. The upper panel shows an electrocardiogram on the left anterior chest. The middle panel shows a 10-beat signal-averaged potential and the lower panel shows a 100-beat signal-averaged potential. If signals are averaged at about 100 beats, peak-to-peak noise levels will be less than 5 µV. In the present study, 96–154 (mean 126.2) beats were averaged and
Fig. 2. Effect of signal-averaging on the improvement of signal-to-noise (S/N) ratio. An electrocardiogram on the left anterior chest is shown. In our system, the S/N ratio was greatly improved when 100 beats were averaged.

the peak-to-peak noise level of signal-averaged beats was less than 5 μV in all subjects.

Isopotential maps:

In each subject, a signal-averaged beat was used to construct isopotential maps. Onsets and offsets of the QRS complex were manually determined from the 87-lead root-mean-square (RMS) voltage. In all waveforms, the selected baseline was UP interval. Signal-averaged low-amplitude isopotential maps were constructed at 1 msec intervals from (J point−30) msec to (J point+30) msec. The maxima and minima were indicated by large + signs and — signs, respectively. Zero-potential lines were drawn by broken lines. We studied the patterns of the maps, the appearance time and position of early ventricular repolarization, the disappearance time and position of ventricular depolarization, and the overlapping time in the terminal portion of the QRS complex.

According to the criteria of Taccardi, we defined potentials to ven-
tricular depolarization as those which develop during the QRS complex and then disappear before or with the onset of the ST segment, and assigned potentials to ventricular repolarization as those which evolve late in the QRS complex, and continue in the ST segment.

**Results**

Thirty-two subjects were classified into 8 types (types A through H) of body surface isopotential maps. Types A, B, C, and D were previously reported by Spach et al.\(^{10}\) and types E, F, G, and H were found in our study. In the first type (type A) (Fig. 3), at 72 msec after the onset of the QRS complex, there was one minimum on the front and one maximum on the back. At 74 msec, the extra-maximum appeared on the left anterior chest. At 76 msec, the depolarization maximum disappeared. At 94 msec, the maximum moved to the middle chest and the minimum moved to the back. It was considered that the first maximum was ventricular depolarization and the second showed the appearance of early ventricular repolarization. Type A was observed in 8 of 32 subjects.

![Fig. 3. Type A.](image)

![Fig. 4. Type B.](image)
In the second pattern (type B) (Fig. 4), at 45 msec, there were two maxima (on the upper sternum and the back) and one minimum (on the middle chest). At 47 msec, the maximum on the back disappeared and the extra-maximum (ventricular repolarization) on the left anterior chest appeared. At 67 msec, the maximum was in contact with the maximum of the upper sternum and the minimum moved to the back. At 69 msec, the maximum on the upper sternal region disappeared. Type B was observed in 5 of 32 subjects.

Figure 5 shows the changing patterns of type C. In the upper sternal region, there was one minimum and one maximum (ventricular depolarization) at 72 msec. At 76 msec, the positive potential (ventricular repolarization) after the appearance of the less negative region, appeared on the left anterior region from the high left axilla. In the next stage, the maximum on the upper sternal region disappeared and the minimum moved downward. Type C was observed in 4 of 32 subjects.

In type D (Fig. 6), there were two maxima (on the upper sternum and back) and one minimum (on the anterior chest) at 63 msec. The maximum stayed on the upper sternum. The extra-maximum appeared on the upper...
sternum and shifted to the left anterior chest at 83 msec. The maximum on the back disappeared. At 85–87 msec, the positive maximum on the upper sternum disappeared and the minimum moved to the back. Type D was observed in 8 of 32 subjects.

In this study, we added 4 new types (types E, F, G, and H) as follows. In type E (Fig. 7), there was one minimum on the front and two maxima (on the upper sternum and the back) as in type D during the first phase. In the next phase, the positive potential of repolarization appeared on the left anterior chest and the maximum on the back disappeared. In the last phase, the minimum on the anterior chest moved to the back. This pattern occurred in 2 subjects.

In types F, G, and H, it was impossible to distinguish ventricular depolarization from repolarization in the positive maximum. In type F (Fig. 8), first, there was one minimum (on the upper sternum) and one maximum (on the left anterior chest). In the next phase, the maximum continued to stay on the left anterior chest and the negative potentials moved backward. This type was seen in 3 subjects. In type G (Fig. 9) which occurred in 1 subject, there was one maximum (on the upper sternum) and one minimum

---

**Fig. 7. Type E.**

**Fig. 8. Type F.**
(on the middle chest). The maximum shifted to the left anterior chest from the upper sternal region. The negative potentials moved backward. In the final pattern (type H) (Fig. 10), there were two minima (on the middle chest and the back) and two maxima (on the upper sternum and the left anterior chest) of ventricular depolarizations. In the next stage, two maxima (probably, depolarizations) were fused on the upper sternal region and the minimum on the middle chest disappeared. Then, at 105 msec the maximum shifted to the left anterior chest from the upper sternal region. Type H occurred in 1 subject.

In all subjects, the mean±SD duration of the QRS was 82.0±8.7 msec. The mean±SD of the appearance time of ventricular repolarization was 71.8±10.5 msec from the onset of the QRS complex. In 5 subjects, it was im-

**Fig. 9. Type G.**

**Fig. 10. Type H.**

Figs. 3–10. Changing patterns of isopotential maps at the end of the QRS complex in types A-H. Electrocardiographic waves of a precordial lead are shown to the left. Times indicate those from the onset of the QRS complex. The maxima and minima are indicated by large (+) signs and (−) signs, respectively. Zero-potential lines are drawn by broken lines. Arrows indicate that the repolarization potentials shifted to the left anterior chest from the torso (type B), high left axilla (type C), upper sternum (type D), and middle back (type E). See text for details.
Fig. 11. Sites of the appearance of the early ventricular repolarization. The early ventricular repolarization was widely distributed on the left anterior chest and upper sternal region.

possible to determine the appearance time. Thereafter, in 23 of 27 subjects (85%), the appearance time was ahead of the J point and in 4 subjects (15%), it was after the J point. Figure 11 shows the sites of appearance of the early ventricular repolarization. The appearance sites of the early repolarization were widely distributed on the left anterior chest (around G4) and the upper sternal region. The mean±SD of the disappearance times of ventricular depolarization was 79.7±9.4 msec. In all subjects, the disappearance time of ventricular depolarization did not exceed an absolute value of ±20 msec at the end of the QRS complex. The last excitation position was the upper sternal region or the upper back region. The overlapping time between the appearance time of ventricular repolarization and the disappearance time of ventricular depolarization was 8.6±6.4 msec.

DISCUSSION

There have been several reports about body surface isopotential maps at the end of the QRS complex in normal human subjects. However, each investigator has used a system with a variable resolution. Spach et al have reported on a system which emphasized the accurate depiction of low-level potentials less than 250 μV and the peak-to-peak noise was 15–20 μV. Our system had a resolution of 5 μV and the peak-to-peak noise level was less than 5 μV.

Mirvis used 20-sec sampling data for averaging to construct isopotential maps during atrial excitation and recovery and Green et al used a signal-averaging beat of 5–15 beats to evaluate low-level potentials during QRS complex. In the present study, a mean of 126.2 beats were averaged.

Spach et al have classified low level isopotential map patterns at the
end of the QRS complex into 4 types in normal subjects. However, we classified them into 8 types, including the 4 types reported by Spach et al. In types F, G, and H, we could not accurately distinguish ventricular depolarization from repolarization. It was considered that the appearance position of ventricular repolarization was close to the disappearance position of depolarization.

In the previous report described by Spach et al, with increasing age there was a decrease in the duration of overlap of excitation and repolarization. In the present study, the time duration of overlapping excitation and repolarizing potentials was $8.6 \pm 6.4$ msec and the mean age of our subjects was 27.8 years. In subjects 20-29 years old, our result for overlapping time was almost consistent with that of Spach et al.

Widman et al have reported that in $18/40$ (45%) subjects the body surface manifestation of repolarization was seen an average of $9.4 \pm 4.8$ msec before the end of the QRS complex. In our study, this was seen in 23 of 27 subjects (85%), at an average of $8.2 \pm 8.0$ msec prior to the end of the QRS complex.

The early repolarization was widely distributed on the left anterior chest and upper sternal region (Fig. 11). The excitation waves propagate along the myocardial fibers. However, ventricular repolarization is not synchronous in all the fibers and consequently some of the fibers are in a more advanced stage of recovery than their neighbors. Therefore, there are variations in the appearance patterns of the early repolarization.

In conclusion, this study detailed body surface isopotential map patterns in normal human subjects with overlap between the end of ventricular depolarization and the onset of ventricular repolarization using our signal-averaged body surface mapping system. In this system, there was abundant evidence that the body surface distribution of low level potentials provides more information concerning localized cardiac activity than standard 12-lead electrocardiograms and the conventional mapping system. These results suggested the difference between the appearance time of repolarization and the disappearance time of depolarization for each lead. We concluded that it is difficult to evaluate electrocardiographic waves at the end of the QRS complex with the dipolar theory only.

Acknowledgments

We would like to thank Mr. Nobuyuki Kitagawa for his skillful technical assistance.
REFERENCES


