The Expression of Normal Ventricular Repolarization in the Body Surface Distribution of T Potentials

J. A. Abildskov, M.D., Mary Jo Burgess, M.D., Robert L. Lux, Ph.D., Roland Wyatt, B.S., and G. Michael Vincent, M.D.

SUMMARY Isopotential maps from 120 normal subjects were obtained from 192 simultaneously recorded electrocardiographic leads. Maps were plotted at 1 msec intervals during the QRS and 5 msec intervals during the ST-T deflection. Repetition of QRS features was evident during all but the first few msec of the initial half of serial T maps. This suggests similarities of the normal sequence of ventricular excitation and recovery. Such similarities have been demonstrated by direct studies but are not evident from other electrocardiographic examinations. Serial maps during later portions of the T wave showed decreasing intensity of potentials with little change of body surface locations. This also correlates with an established feature of ventricular repolarization, namely that potential difference boundaries with stable locations are widely distributed during part of that process.

Findings suggest isopotential maps show features of ventricular recovery not apparent from less extensive examinations.

THE SEQUENCE OF VENTRICULAR ACTIVATION has been defined in considerable detail using the rapid deflection in bipolar electrograms to determine local excitation time. 1, 2 That information furnishes the most satisfactory explanation of the normal QRS complex and its alteration by disease. There is no index of local ventricular repolarization comparable to that available for excitation. Information concerning the normal repolarization sequence has been based on measurement of refractory periods, 3 recovery of excitability following particular activation sequences, 4, 5 suction potential recordings, 6 and most recently by unipolar potential patterns from multiple epicardial and intramural electrograms. 7, 8 These data have provided a reasonably detailed description of the normal repolarization sequence in mammalian hearts. The present study was carried out to examine the degree to which features of ventricular repolarization established by direct studies are evidenced by the body surface potential distribution during the T wave of normal human subjects.

Methods

Electrocardiograms were recorded from 192 body surface sites in 120 normal subjects in the age range of 20 to 65 years. None of these subjects had evidence of heart disease by medical history or physical examination. All had normal 12 lead electrocardiograms including QRS duration less than 0.10 seconds, frontal plane QRS axes within the range of 0 to 90°, and upright T waves in leads I, II, and V3 and V6. Data from all 192 leads were recorded simultaneously on magnetic tape using multiplexing circuitry designed for this purpose. 9 The recording system had a bandwidth of 0.05 to 500 Hz (−3 dB) for each lead. Signals were demultiplexed, computer processed, and copies of body surface isopotential maps obtained at 1 msec intervals during the QRS and 5 msec intervals during the ST-T deflection. Isopotential maps were recorded on 16 mm film and each frame viewed for qualitative description. The location of poles, contour of isopotential lines, and time sequence of changes of these features were noted. Features of the maps during the ST-T deflection were compared to those during the QRS complex.

In addition, a two-dimensional map display of correlation coefficients relating instantaneous QRS and T map frames was developed (see Appendix). The values of given points in these plots were the correlation coefficients between QRS and T maps at the instants defined by the points. Display of the plot as isocorrelation lines showed quantitative information pertaining to similarities in temporal sequences of QRS and T potential distributions.

Results

Repetition of features from QRS maps in the serial T wave maps was evident by both qualitative and quantitative analysis. QRS features repeated in T maps included body surface location of poles, contour of isopotential lines, and changes of these features with time. As shown in the upper left map of figure 1, the normal QRS isopotential patterns characteristically began with a maximum on the left anterior chest wall. At this time, the left posterior and right posterolateral thoracic surfaces exhibited diffuse low level negative potentials. As seen in the map in the lower left panel of figure 1, a distinct negative pole then formed on the upper right thorax. Subsequently the negative pole moved anteriorly toward the maximum pole. During earliest portions of the T wave, a positive pole was present on the anterior surface of the left thorax. Negative potentials, however, were of low intensity and located on the upper right and posterior thorax rather than over the entire posterior and right lateral thorax as was true of the negative potentials during the earliest parts of the QRS (see upper right panel of fig. 1). Map features of approximately the next 40 msec of the QRS were repeated in T maps. An anterior positive pole was present in both and a negative pole on the right upper thorax was present in both (see lower panels of fig. 1).

The time course of movement of the negative pole toward
the anterior chest was also a feature common to the QRS and T and is illustrated in figure 2. Serial maps during the QRS complex and approximately the initial one half of the T wave were both dynamic with changing body surface location of maxima and minima. The similarity of these dynamic changes was especially evident when the maps were viewed as motion pictures but is also evident from the maps illustrated in figures 1 and 2. In figure 1, panel B, an early moment during the T wave shows low level negative potentials on the upper back and upper right and anterior thorax.
Panel D of that figure illustrating a later instant during the T wave shows increased amplitude of negative potentials in the same region and extension of negative potentials to more inferior and anterior locations. The maximum illustrated in this panel is also displaced slightly leftward with respect to that at an earlier instant. In figure 2, panel B, still further leftward displacement of the maximum is present.

After the serial potential patterns just described, the time sequence of QRS and T maps differed markedly. In the case of the QRS, the anterior negative pole continued to increase in intensity and to move toward the left. During this time, the QRS positive pole decreased in intensity and moved posteriorly. Terminal portions of the QRS were characterized by patterns with an anterior negative pole and diffuse low level positivity of the posterior and lateral chest walls. In contrast to the QRS potential patterns, those during approximately the last half of the T wave remained largely stable. Both the anterior positive pole and the diffuse upper thorax negative potentials decreased in intensity gradually with little change of their body surface location. The differences in the time sequence of potential patterns during mid and later portions of the QRS and T are shown in figure 3.

A two-dimensional display of the correlation coefficients of T maps to QRS maps was developed to obtain quantitative evidence of the repetition of QRS potential patterns in T potential distributions. For purposes of explanation, a correlation coefficient map of the QRS to itself is shown in the top panel of figure 4. Regions where high positive correlation exists, as in the regions around the diagonal of the plot, correspond to times along both axes when map frames are similar. Regions where high negative correlation exists correspond to times when map frames have similar contour patterns but of opposite polarity. Low correlations (|ρ| < 0.4) are indicative of dissimilar map frames. Information pertaining to map pattern dynamics is also available from the display. Regions where isocorrelation line density is great indicate intervals of time when the map patterns are changing. Conversely, areas of low line density (plateaus) show intervals of time when map patterns are stable. Furthermore, perfectly vertical or horizontal isocorrelation lines would exist at times when map patterns along one axis are static.

Implicit in plotting the correlation of the QRS to itself is a line of perfect correlation along the 45° axis of the plot. In the plot of QRS to QRS shown, there is a plateau high correlation surrounding the 45° axis of the plot. The width of this plateau varies throughout the time course of the QRS. The areas of high correlation where line density is sparse represent portions of the QRS where the distribution of potentials is relatively stable and the areas of high correlation where line density is great represent portions of the QRS where the distribution of potentials is changing rapidly. The middle panel of figure 4 shows a plot of the cor-

![Figure 3](http://circ.ahajournals.org/)

**Figure 3.** Isopotential maps of late portions of QRS (panels A and C) and late portions of T (panels B and D) demonstrating dissimilarity of QRS and T patterns.
Correlation coefficients of potential patterns during the QRS and T. The plot indicates that over most of the T there exists an increasingly strong positive correlation of the T wave with early portions of the QRS. Mid portions of the QRS complex had little correlation with T potential patterns and late portions of the QRS had a negative correlation to the T wave. The positive correlation of T waves to early portions of the QRS complex is related to the repetition of QRS features in the T maps. The periods during which little correlation of QRS and T maps existed corresponded to intervals during which the potential distributions of QRS maps were changing rapidly, while those of the T were stable. The negative correlation of T potentials with late portions of the QRS reflects a period during which both QRS and T map patterns were relatively stable but an anterior positive pole existed in the T maps while that feature of early QRS maps had been replaced by an anterior negative pole. The shape of the contours indicates that both QRS and T potential distributions are changing although the predominant vertical tendency of the lines points to the greater degree of dynamics of the QRS as compared to that of the T. To further document the fact that the T was not stable, the correlation coefficient plot of the T versus itself is shown in the bottom panel of figure 4. The large plateau indicates a non-dynamic map sequence compared to the QRS vs QRS plot. However, the existing slopes in the correlation coefficient surface can be accounted for only by a changing T map sequence.

Discussion

Both similarities and differences in activation and recovery sequences have been demonstrated by direct studies on experimental animals. The findings of the study reported here indicate that analysis of T potential distributions and their relation to patterns of QRS distribution provide more information concerning ventricular recovery properties than is available in the 12 lead electrocardiographic examination in routine clinical use.

If ventricular recovery sequence was exactly the same as activation sequence, the two processes would have the same spatial and temporal distribution of potential patterns, but the polarity of the two patterns would be reversed. If the sequence of recovery were the inverse of the sequence of activation, the polarity of poles in the QRS and T would be the same, but QRS features would appear in T map frames in reverse sequence.

In the maps of normal T potential distribution, QRS features were repeated in the T maps with the same polarity and with the same spatial and temporal sequence. This suggests that there are both similarities and differences in activation and recovery sequence. The fact that the location of positive and negative poles during the T is similar to their location during early and mid portions of the QRS indicates that recovery sequence does not exactly follow excitation sequence, and is analogous to the concordance of polarity of the QRS and T waves in standard electrocardiographic leads. The physiologic basis for the polarity of poles being the same in T map frames as during the QRS is most likely due to longer repolarization times in the endocardium as compared to the epicardium. Refractory periods have been shown to be longer at endocardial than at epicardial sites.

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Recovery of excitability after normal ventricular excitation is also later at the endocardial than at the epicardial layer. Longer intrinsic recovery times near the endocardium are also suggested by distribution of recovery potentials determined from multiple epicardial and intramural electrograms.

The repetition of QRS potential distribution in T map frames indicates that although the sequence of excitation and recovery may differ on the endocardial-epicardial axis there are also some similarities between the two processes. The most likely physiologic basis for the repetition of QRS potential distribution patterns in T map frames is the similarity in excitation and recovery sequences on the epicardial surface, previously demonstrated by direct experimental studies. The similarity of this feature of excitation and recovery observable in maps of potential distributions has no counterpart in single electrocardiographic leads, or in routinely used 12 lead electrocardiographic examinations.

The stable pattern of maps during later portions of the T wave also correlates well with established physiologic features of ventricular repolarization. In contrast to excitation, which occurs rapidly in individual cells and small segments of ventricular muscle, repolarization requires a longer time for completion. This means boundaries of potential difference between areas at different stages of recovery become distributed in all or most of the ventricular mass and have relatively stable locations during part of the repolarization process. This distributed character of repolarization, in contrast to the localized and changing cardiac location of excitation, could be anticipated to result in the more stable pattern of maps during portions of the T than during the QRS deflection that was observed.

Although this study concerned the body surface distribution of T potentials of normal subjects, the findings have significance with respect to the recognition of repolarization abnormalities. Predictions of body surface potential patterns to be expected with particular abnormalities will require direct confirmation but the basis of these predictions is implicit in the physiology of normal repolarization and its expression in surface potential patterns.

Failure of serial T maps to repeat features of normal QRS maps would be expected with such abnormalities of repolarization at the epicardial level as reduced recovery time near the ventricular base or prolonged recovery time near the apex. These situations would change the normal apex-to-base sequence of recovery which is the most likely physiologic basis of repetition of QRS features in the T. In contrast, prolonged basal recovery or reduced apical recovery time at the epicardium would not be expected to alter the repetition of normal QRS features in serial T maps since the repetition has the physiologic basis of apex-to-base recovery sequence and this would still occur with the abnormalities named. All of the epicardial abnormalities named would be expected to alter polarity or amplitude of T potential poles since the normal epicardial to endocardial gradient of recovery would be altered.

The stable pattern of T maps during the later portions of normal T waves provides particular promise for improved cardiac diagnosis. This feature of T maps is based on repolarization occurring simultaneously in many cardiac locations. Against this background of relatively homo-
Anterior Infarctional Changes Occurring during Mid and Late Ventricular Activation Detectable by Surface Mapping Techniques

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SUMMARY This is a comparative body surface potential map study of 26 patients with a recent acute anterior myocardial infarction. The mean plus or minus two standard deviations (± 2 SD) for the voltage distribution was established at 5 msec intervals throughout the cardiac cycle in normal subjects and in a recent study. Zones in which a patient's potential distribution fell outside the normal range were analyzed as to location, duration and intensity of the expected time course of ventricular activation. Only four patients had departures from the normal distribution confined to the time zone. Twenty patients had abnormalities in the Q wave. One patient had not only Q wave abnormalities, but also had areas of both positivity and negativity exceeding ± 2 SD, which occurred well after 30 msec. Two patients with clear documented diagnostic evidence for Q waves during the first few days of hospitalization had these findings by the date of body surface mapping. They did retain, however, departures in the left ventricular activation sequence. The diagnostic abnormalities occurring between 30 to 60 msec after onset of ventricular activation. These changes occurring in the mid and late time zones of the activation sequence are not detectable by conventional electrocardiography or vectorcardiography, yet present a strikingly apparent finding by this technique of analysis and display.

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Received March 12, 1976; revision accepted July 6, 1976.

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_Circulation_. 1976;54:901-906
doi: 10.1161/01.CIR.54.6.901

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1976 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

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