Isopotential Body Surface Mapping in Subjects of All Ages: Emphasis on Low-Level Potentials with Analysis of the Method

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SUMMARY In this report we consider the body surface distribution of low-level potentials, particularly those of the U wave, the PR segment, and the ST segment in normal subjects. The long-term objective of this report is to study low-level body surface potentials, but adequate methods are required for that. Therefore, the primary emphasis in this report is artifacts that may occur in these potential distributions. Although receiving a secondary emphasis, the findings show that accurate low-level potential distributions can be recorded and they have interesting and distinctive features. The results present two features that are important in the construction of isopotential maps: 1) The normal distribution of the surface potentials characteristic of these signals, especially the U wave and the PR segment, and 2) the influence of subtracting or not subtracting these patterns in the process of constructing body surface maps for the entire cardiac cycle. An isopotential map method is described for identifying U waves. A reciprocal relationship was found between the normal body surface patterns of the U wave and the PR segment. If the period of the U wave was unfortunately chosen as the time for zero potential reference for all electrodes, the maps subsequently made for QRS and the ST segment were distorted on the anterior chest by an element of potential representing currents of terminal ventricular repolarization during overlapping T and U waves. The implication of these results is significant in that the timing of the reference baseline is critical in determining the pattern of the depicted map. These considerations are especially important in the interpretation of precordial and body surface maps recorded during exercise. The false patterns found can be quite misleading, since the baseline artifacts produce regional changes that may simulate patterns of abnormalities.

IN THIS PAPER we consider the mapping of body surface potential distributions that primarily contain potentials under 250 \( \mu \)V, particularly those that occur during the U wave, the PR segment, and the ST segment. Implicit in the mapping of low-level potential distributions during these intervals is the assumption that the methods used are precise enough to ensure that accurate patterns are depicted in the maps. Our idea was that knowledge of the locations and shapes of the U waves and the PR segments over the entire torso in normal subjects would enhance the reliability of the spatial interpretation of these deflections which, with the ST segment, are particularly sensitive to error and are most difficult to depict accurately in the form of potential distributions.

Our interest in low-level potentials was stimulated by the results of experiments with simultaneously measured epicardial and body surface potential distributions in intact chimpanzees and dogs. These experiments showed that many of the important electrical events in the heart were reflected on the body surface in the low-level patterns rather than in the maxima and minima, the two features that have thus far received major emphasis in characterizing surface maps. Taccardi and associates have observed in patients with coronary artery disease that diagnostic information was conveyed by small signals of 40–100 \( \mu \)V. Thus, it is becoming more important for clinical interpretation and for quantitative inverse calculations that artifact in the surface maps be identified carefully in order to represent the low-level potential gradients as accurately as possible. This is particularly appropriate, since methods are improving, the use of maps is increasing, and total body surface maps are becoming easy to record in clinical practice with limited lead systems that use 24–32 leads. Study of both the methods used to construct the potential distributions and the patterns that result during the ST segment is especially pertinent in view of the widespread use of various electrocardiographic methods for detecting and evaluating ventricular ischemia in patients at rest and during exercise.

Information about the spatial distribution of U wave and PR segment deflections should provide a way to evaluate the differences that result in ST-segment maps when different zero voltage baselines are used to construct the maps. For example, when the heart rate is greater than 110 beats/min, the P wave is usually superimposed upon the preceding U wave, and error is introduced in the analysis of the PR segment and ST segment. When this occurs in standard ECGs, the physician automatically selects the baseline for the interpretation. However, to construct body surface maps it is not clear how to make baseline selections in all of the waveforms recorded from the total torso and what effects would be produced in the maps by different baseline selections in the original wave-
forms. Depending on the shape of these waveforms and their body surface locations, the apparent surface map patterns will be altered from the real ones, particularly the low-level potential distributions.

In this report we describe the procedures that we used for the construction of body surface maps that contain primarily low-level potentials. U wave and PR segment potential distributions are presented, with the validity of the patterns checked by inspection of the waveforms that were used to construct the maps. Knowledge of U wave and PR segment deflections and their locations provided a way to detect and determine false patterns that can occur in ST-segment maps, especially when surface maps are recorded at rapid heart rates.

The ultimate goal of this work, the study of low-level potentials, requires adequate methods. Therefore, we focus primarily on artifacts that can occur in these potential distributions. In addition, even though a secondary emphasis, the results show that accurate low-level potential distributions can be obtained, and these have interesting and distinctive features during the U wave, PR segment, and the ST segment.

Methods

Clinical Information (Normal Subjects)

More than 400 subjects with and without heart disease, ages ranging from 1 day to 60 years, have had body surface maps recorded in our laboratory over the past 4 years. From this group, we initially reviewed the data of 105 normal subjects and selected 66 subjects between the ages of 1–60 years that were considered to have waveforms and surface maps sufficiently free of artifact to undergo detailed analysis for this study. At least 10 subjects represented each decade between 1–60 years. There were 59 males and 7 females. The women were 40–60 years old. The major criterion for normality was a history of good health. Subjects who had a history of suspected or known coronary artery disease, hypertension or peripheral vascular disease were excluded. None of the subjects underwent coronary arteriography. All of those older than 22 years had had a routine physical examination within 1 year and had a documented blood pressure not exceeding 140 mm Hg systolic and 90 mm Hg diastolic, a normal chest x-ray, and a normal routine ECG. The maps were recorded with the subjects in the supine position and while they were relaxed; some were asleep. For each subject there was little change in heart rate during the recording; however, these basal heart rates varied from subject to subject. They ranged from 65–100 beats/min in the children younger than 6 years and from 53–80 beats/min in those older than 6 years. Inspection of the waveforms and the maps showed all subjects to have normal P waves (i.e., no atrial ectopic foci) and a QRS duration that was within normal limits for age.

Protocol for Direct Digital Recording of the Waveforms

Waveforms were recorded from 150 positions on the chest and upper abdomen with a lead array composed of 15 straps with 10 electrodes each (fig. 1A). All potentials were measured with reference to Wilson’s central terminal. The straps were similar to those designed by Taccardi with recessed electrodes to which regular electrode paste was added. Double sticky tape was used to hold the straps against the skin. Placement of the electrodes required 5–10 minutes. With this method of electrode application, an electrode occasionally dislodged from the skin. The use of a wide elastic bandage to swathe the patient by encircling the entire electrode array to compress the electrodes against the skin eliminated that problem (fig. 1B). In most subjects the swathing seemed to have a relaxing effect, and we thought it resulted in a decrease of muscle tremor.

The 150-point electrode array was connected to a switching box (figs. 1B and C) which allowed the technician to select one of two modes of recording the maps. First, all subjects had waveforms recorded from the 150 positions by recording subsets of 20 waveforms simultaneously along with a time reference trace. This method required combining data recorded during different heart cycles to construct the maps. Initially, we used a respiratory plethysmograph to monitor respiration to ensure that different beats undergoing analysis were those selected for the same instant of the respiratory cycle. Monitoring the time reference electrocardiographic trace was at least as accurate an index of the respiratory effects on the waveforms as the plethysmograph. Therefore, we relied on changes in shape and amplitude of the time reference waveform as an indicator of the similarity of cardiac potentials recorded during different heart beats. Also, the time interval extending from the onset of the P wave through the U wave was so long (600–960 msec) that it was impossible to maintain a perfectly constant body surface-to-heart geometric relationship throughout one heart beat. Attempts to minimize variations with held inspiration or fixed expiration failed since the waveforms changed while the chest position was fixed, presumably due to cardiac volume changes secondary to the absence of respiratory movement.

In 10 subjects, after the resting maps were recorded with the 150-electrode array, a second type of map recording was used to study the effect of an increase in heart rate on the waveforms. This recording used a limited lead system reported by Barr et al. and Warren et al. The method has been described in detail. It consists of construction of the 150-point maps from the waveforms recorded at 24 positions. The computation of the potentials at 150 positions from the original 24 waveforms was achieved for about 2000 maps within 10–15 minutes. The calculation used a predetermined set of coefficients which did not vary from subject to subject. Comparison of the maps constructed from 24 leads and those recorded from the 150 electrodes has shown excellent agreement for QRS and ST-T waves. Since all of the 24 leads were sampled simultaneously, this method was used to record maps at different heart rates after mild sit-up exercises in these subjects.

The recording ensemble (fig. 1C) consisted of 24 AC
amplifiers that had a flat frequency response between 0.1-30,000 Hz. The analog signals from each amplifier were sampled in real time at a rate of 1000 samples/sec (12-bit samples) in patients older than 2 years of age and at a rate of 1667 samples/sec in infants. Previous analysis by Barr and Spach\(^8\) showed that 1000 samples/sec were adequate to reconstruct the waveforms of adult patients after myocardial infarction, a feature consistent with the frequency spectrum analysis of Langer and Geselowitz.\(^19\)

The design of the computer system has been described in detail by Barr et al.\(^20\) It allowed triggering from QRS to initiate sampling just before the P wave. Sampling was continuous for 1800-3600 msec (block of data). This interval encompassed two to four heart beats, and several blocks of data were recorded for each subset of waveforms in the following manner. The computer stored the data and displayed the waveforms immediately on a Tektronix 4002 display unit.\(^21\) Once it was established that each waveform was as free as possible of artifact, the waveforms were recorded permanently on digital tape. The recording of the waveforms with the 24-lead system required 2-10 minutes, and to record from 150 positions required 12-30 minutes. A major component of the recording time was occupied by on-line editing of the waveforms, discarding those that were not acceptable, and redisplaying new waveforms to obtain the best quality possible for permanently recording them on digital tape.

**Protocol for Constructing the Isopotential Maps from the Waveforms**

The recording procedure described above was performed by a separate program that did not incor-
porate data analysis capability. During data acquisition, the investigator or technician concentrated full attention on assuring that the waveforms being measured were as free as possible of artifact. In addition, the program for data processing was designed to check the waveforms for artifact by displaying them in detail; then the program converted the measurements into potential distributions.

The original blocks of data were redisplayed in the exact format of the original display, automatically photographed on 16-mm film (fig. 1D), and converted into hard copy for detailed editing. A typical group of 20 waveforms, along with the time reference trace, is shown in figure 2A. First, the original waveforms were scrutinized for interference due to electrical noise, and many were expanded for a better estimate of this artifact. As shown in figure 2B, the peak-to-peak noise was 15–20 \( \mu V \), and these waveforms were considered
Figure 3. Baseline adjustment and time alignment of original waveforms. The waveforms in A were considered to have linear baseline drift. The baseline times in B were picked for purposes of this illustration during the PR segment and at the end of the T wave as indicated by the vertical lines (v). Linear interpolation of each potential value was performed between the first and second baseline times, and the waveforms were redisplayed in the corrected format (C). In D the time reference waveform is indicated by the asterisk for trace 13, and the time alignment instant is indicated by the vertical line (z) at the baseline crossing of the intrinsicoid deflection of QRS.

quite acceptable. Second, a search was made for baseline shifts in the waveforms. When there was no detectable baseline drift or if mild drift was present that was judged to be linear from one beat to the next, the waveforms were selected for the map. However, when non-linear drift was present, such as the positive arc in trace 8 of figure 2A, the waveforms were discarded.

The procedure used for baseline selection for a single heart beat is illustrated in figure 3. One waveform was used to select two baseline times, one just before the onset of the P wave and the other just after the U wave (UP interval baseline), to include maps for P-QRS-T-U-wave analysis. The two baseline times were automatically applied to all of the waveforms in the block of data. The computer program applied linear interpolation to adjust each potential value from the first baseline time instant to the second one, and the corrected waveforms were displayed (figs. 3B and C). The zero time instant was selected as follows: The operator selected any time instant during the upstroke of the R wave of the time reference tracing. The zero time instant then was selected by the computer as the zero baseline crossing of the QRS intrinsicoid deflection (fig. 3D), and the common reference trace was used for time alignment purposes. The computer program then stored each adjusted waveform at its designated map position.

Display of the Isopotential Maps

The computer program automatically displayed the body surface maps, drew the contour lines, and photographed each map for subsequent instant-by-instant and motion analysis. The usual procedure was to display maps for every other millisecond during the P wave and the PR segment, every millisecond during QRS, and every fourth millisecond throughout the ST-T-U wave. The isopotential lines (fig. 4) were drawn according to a logarithmic scale to represent progressively the following values in microvolts: 40, 60, 100, 150, 250, 400, 600, 1000, 1500, 2500, 4000, 6000 and 10,000. The use of a log separation was considered beneficial, since it accentuated the details of the low-level potential changes, without loss of detail near the extrema (fig. 4). Additionally, the potential value at each recording point was displayed in tens of microvolts (e.g., 14 = 140 μV). We found that by displaying the potential value at each location the ability to judge errors in the individual maps, as well as to
A. 18 YEARS

\[ \begin{align*} 
& - -40 \mu V \\
& + 140 \mu V \\
\end{align*} \]

B. 39 YEARS

\[ \begin{align*} 
& + 60 \mu V \\
& - -10 \mu V \\
\end{align*} \]

**Figure 4.** U-wave body surface potential distributions in two normal subjects. The time instant of each map is indicated by the vertical mark in the associated waveform beneath each map. The heart rate in the 18-year-old subject (A) was 90 beats/min, and the baseline choice was in the UP interval. In the 39-year-old subject (B) the heart rate was 70 beats/min, and the baseline choice was the same. The values of the contour lines are indicated in \( \mu V \) by the oval lines below each map, and the values of the maxima and minima are noted above each map.

gain confidence in the accuracy of the depicted distributions, was considerably enhanced compared with viewing the contour lines alone. However, these individual values became very difficult to see when the size of the surface map was reduced, e.g., in publications,\(^1\)\(^4\) although the numbers still served as an indicator of the electrode positions.

Finally, we studied the ST segment in the form of the average body surface potential distribution during the first 50 msec of this interval. This was done by first inspecting the individual QRS maps (not the waveforms) to select the final instant of excitation. The potentials at each of the 150 positions then were averaged over the next 50 msec, and a single ST-segment average body surface map was displayed, i.e., an ST(0-50 msec) map.

**Evaluation of the Recording Methods**

The noise in the typical digitized waveforms was in the range of peak-to-peak values of 15 to 20 \( \mu V \) (fig. 2B). The on-line visual editing of the waveforms was particularly helpful in keeping undesirable high frequency noise to a minimum in the final waveforms stored on digital tape. When it occurred, the cause...
often could be found and eliminated immediately; e.g., amplifier noise, muscle tremor, 60-Hz interference, or a loose electrode contact. Some initial subjects were omitted for analysis because errors appeared in their QRS maps, rather than in the ST-T-wave maps, due to the time alignment of reference waveforms with variable QRS shapes. Again, editing during the recording was important to ensure that similar shapes of the reference waveforms were present. This problem was eliminated with the simultaneous recording of all waveforms with the 24-lead system.

The problem that we found most difficult to resolve was non-linear baseline drift in the original waveforms (fig. 2A, trace 8). When present, the drift often occurred simultaneously in multiple leads, but with varying polarity. We were unable to make adjustments for this artifact, and we avoided it only by constant attention during the recording procedure so that undesirable waveforms could be discarded, new waveforms displayed and, if acceptable, saved. A final limitation was the inability to select an adequate baseline when the rate was so rapid that the UP interval disappeared in some leads, i.e., heart currents visibly affected the body surface potentials all the time. Inspection of all of the waveforms was important, since in some subjects only a few waveforms showed superimposed U and P waves.

Results

U Wave and PR Segment:
Waveform Shape and Location

Often it was difficult to recognize the polarity of the U wave and the PR segment in some leads. To facilitate determining whether a deflection was positive or negative, the waveforms of all 150 positions were expanded as shown in figure 2B. The analysis procedure consisted of visual inspection of three to four heartbeats in each of these waveforms. The maximum and minimum amplitudes of the U wave and the PR segment were measured manually, and they showed good agreement with the potential values depicted in the final maps. In all 66 subjects the basal heart rate was low enough so that all waveforms were judged to have a definite UP interval (TP interval for small children) for the baseline choice. This feature met the requirement of an “ideal” baseline, which is based on the assumption that it is chosen when and where there are no detectable effects in the potentials from cardiac currents.

U Waves

In many waveforms the U waves were difficult to recognize, as has been emphasized by Watanabe. Slight shifts in the baseline were especially troublesome at the end of the T wave, and only by viewing consecutive waveforms after baseline adjustment did we consider some 15–20 μV deflections to be U waves rather than artifact, and vice versa. Also, we had to resolve the question frequently as to whether a deflection should be identified with the T wave or with the U wave.

We first tried to apply the following long-standing textbook criteria for typical U waves for standard ECGs as summarized by Lepeschkin: 1) To identify a questionable notched wave, the time relation of the notch to the adjusted QT interval provides evidence as to whether the deflection is pure T wave or U wave; 2) ordinarily, the U wave is well separated from the T wave and the TU junction is at the baseline; and 3) if the QT interval is prolonged or the U wave appears early, the T and U waves may show partial fusion and the TU junction becomes elevated.

It was difficult to apply these criteria to all of the waveforms used to construct the body surface maps. For example, we could not define a time relation between notches and the adjusted QT interval that provided adequate evidence of whether the deflection was a pure T wave or U wave. Also, numerous electrocardiographers told us that they thought the T and U waves were not separate in most normal adults. Therefore, we used the maps to identify the instant that we considered to represent the transition from the T wave to the U wave. This was done by viewing the maps sequentially during the time when the T-wave voltage was decreasing. When the T wave spatial maximum decreased to less than 250 μV, any increase or sudden stability in the value of the maximum, which always was located on the anterior torso, was the time judged to be the time of transition between the T wave and the U wave. This time coincided with the notches of typical U waves and TU fusion waves in the original waveforms.

This method allowed identification of U waves, as illustrated by the waveforms shown in figure 5, I. Deflections that were suggestive of U waves, such as the one shown at position A for subject 1, were considered to be notched T waves and unrelated to the U wave if they occurred at a time when prominent voltage still existed in the T wave at other body surface locations. For example, subject 1 had a definite U wave at position B; the deflection at position A of the same subject was not considered a U wave because it occurred during the T wave at position B. This method obviated the necessity for using indirect criteria based on time intervals adjusted to heart rate. It allowed rapid and easy direct identification of questionable U-wave deflections based on the subject's own waveforms. The advantage provided by this method of inspecting the maps was that it provided an unambiguous criterion that was easily met for defining U wave potentials.

U waves were not present in any of the 11 children younger than 8 years of age, and U waves were present in all of the 55 subjects 8–60 years of age. This latter group underwent the following analysis: When the waveforms from all of the locations were reviewed, most subjects had easily identifiable U waves in several leads on the anterior torso, and in most subjects there was elevation of the TU junction in some of these leads. Only in 11 of the 55 subjects did we find a definite well-separated T and U wave with the TU junction at the baseline in all leads. We concluded that most normal subjects have U waves of the type
previously described as partial fusion of T and U waves. The frequent presence of fusion was confirmed by the maps which showed only slight changes in the pattern of the positive potentials on the anterior chest as merging occurred during the time of the downstroke of the T wave and upstroke of the U wave.

A composite map of the locations of the positive and negative U waves for the group of subjects 8–60 years is shown in figure 5, II (top). The composite map represents all of the subjects, and it was constructed in the following way: The polarity of the U wave was marked at each of the 150 locations in each subject. The locations that had U wave deflections with an absolute value less than ±15 μV were left blank. The composite map was made by overlay markings of the locations of the positive and negative U waves for all subjects. The resulting map shows the locations of the positive and negative deflections for the total group rather than for an individual. The locations of the maximal and minimal U wave voltages were plotted in the same way to produce a composite picture of the locations of the extrema of the U wave (indicated by the areas containing the plus and minus signs).

Positive U waves occurred within a broad vertical area on the anterior torso and left side (fig. 5, II, top). The maximum voltage varied from 30–140 μV, and the location where it occurred varied within a circumscribed area near the heart (darkly stippled area).
Lepeschkin noted from analysis of the precordial leads of normal subjects that the maximum voltage of the U wave was shifted several inches to the right of the location of the maximum of the T wave. To further clarify this spatial relationship, we analyzed all of the T waves and plotted the location of the maximum T-wave voltage and that of the U wave in each subject. For the total group, the locations of the T-wave maximum voltage were confined to the same area of the U-wave maximum voltage (fig. 5, II, bottom). In most subjects the specific locations were the same. However, in 17 subjects there was a slight shift from the location of the maximum during the T wave to that of the maximum during the U wave. These shifts in the location of the T wave maximum to that of the U wave occurred primarily in a rightward direction as described by Lepeschkin, but some occurred in a vertical direction. The area where both the T-wave and the U-wave maximum voltage occurred included the locations where precordial leads V3 to V6 are placed, as noted for normal U waves by Nahum and Nuland, but this area extended considerably above and below the line of placement of these leads. It was difficult to be certain of the precise location where the maximum amplitude occurred, because the amplitudes were often similar at adjacent points during the U wave and the T wave. However, within the total region of positive U waves, the amplitude at specific locations varied considerably from subject to subject (fig. 5, I).

Body surface potential distributions at the time of the peak voltage of the U wave are shown for two subjects in figure 4. Note the regular shapes of the positive isopotential lines on the anterior torso in both subjects. There was a well-defined area of negative poten-
tials on the right upper chest in the 18-year-old subject (fig. 4A). However, in the 39-year-old subject (fig. 4B) the negative potentials were vaguely defined, a feature of negative U-wave potentials noted by Naham and Nuland in their normal U-wave body surface maps, the only other U-wave maps that we have seen.

Negative U waves were found in 10 subjects. There was little overlap in the locations where either negative or positive U waves occurred in the composite map (fig. 5, II, top). The negative deflections were located on the right upper chest, and the negative peaks varied from $-20 \mu V$ to $-50 \mu V$. The negative U-wave deflections are illustrated for two subjects in figure 6, I, at position A. Note that in the 21-year-old subject (no. 2) the negative peak at the right shoulder occurred 30 msec after the positive U-wave peak on the anterior precordium at position B, a phenomenon similar to the T-wave events in the epicardial potential distributions of intact chimpanzees.

**PR Segment**

Figure 6 shows examples of typical waveforms (I) and composite maps (II) of the locations of the positive and negative PR segments for the total group. The composite maps of the locations of the positive and negative PR segments were made with the same method used to construct the previously described U-wave composite maps. The composite maps in figure 6, II represent all of the subjects — the group of 11 children younger than 8 years at the top and the 55 subjects 8–60 years at the bottom.

The positive deflections occurred on the right upper chest (stippled area) and the negative deflections were on the anterior torso and left side (slashed area). The clear areas represent regions for the total group where the absolute magnitude of the PR segment deflections was less than 15 $\mu V$. In the younger children the values of the maxima (15–30 $\mu V$) and the area within which they were located were smaller than in the older group (fig. 6, II). Also, the area within which the peak negative values occurred was smaller and extended more superiorly on the anterior chest in the younger group, while the absolute values of the minima (–40 to –60 $\mu V$) exceeded most of those of the older group (–30 to –60 $\mu V$). In the 8–60-year-old group, negative PR segments occurred over a broad area extending more inferiorly and to the left and the minima were located lower on the torso than occurred in the younger children. A small area on the right anterior torso was the only place where either positive or negative deflections occurred among different subjects of the total group (fig. 6, II, bottom). Note that positive PR segments occurred only on the right upper chest, with a few extending under the left clavicle, and negative PR segments occurred only in the broad region shown on the middle and lower torso. However, within each region the amplitude of the deflections at specific locations varied from one subject to another, as shown in figure 6, I. Note that the positive PR segment at the right shoulder (position A) was more prominent in the two adults (subjects 2 and 3) than in the 6-year-old child (subject 1). At position B on the precordium the negative deflection was most prominent in the child. However, there was variation in the total negative area from subject to subject. Only the two adults (fig. 6, I, subjects 2 and 3) had negative PR segment deflections on the lower anterior torso at position C. Subject 2 had a negative PR segment deflection at position B, but subject 3 did not.

**Effect of Increase in Heart Rate on the Waveforms According to Location**

Ten of the normal subjects, 25–36 years old, had waveforms recorded at 80–90 beats/min and after sit-up exercises at slightly higher rates of 100–110 beats/min when there was superposition of the P wave on the preceding U wave. The rates were selected within this range in order to have the least rate effect on the U wave while producing waveforms with and without a definite isoelectric UP interval. A typical result is shown in figure 7, I. The waveforms are shown for positions in three different regions to illustrate the deflections during the U wave and the PR segment in relation to the baseline choices in those areas. At a rate of 90 beats/min the correct baseline was the one in the UP interval (U-P) (fig. 7, I). When the baseline was chosen during the PR segment (P-R) at that rate, the negative U wave and ST-segment potentials at the right shoulder became more negative and the U-wave and ST-segment potentials on the middle and lower anterior torso became more positive.

These regional effects of baseline choice were accentuated when there was superposition of the P wave on the U wave at a rate of 105 beats/min. In the right shoulder region the occurrence of the P wave on the negative U wave produced an apparent "TP interval" (T-P) for baseline choice that resulted in a falsely accentuated positive PR segment and early ST segment (fig. 7, I, position A). Simultaneously at positions B and C a definite isoelectric interval disappeared, and the TP junction baseline (T-P) resulted in lowering of both PR- and ST-segment potentials. That the effects of increased heart rate on the waveforms were regionally determined can be seen from the composite map of the locations of the most positive and negative deflections of both the U wave and the PR segment at basal rates for the group of subjects 8–60 years of age (fig. 7, II). Note that the most positive PR segments occurred in the same area as the negative U waves and the most negative PR segments occurred in the same area as the most positive U waves. Accompanying the composite map of figure 7, II are expanded tracings of the waveforms shown in figure 7, I, for the heart rate of 90 beats/min for the UP interval baseline. The expanded waveforms are shown to allow detailed inspection of the shapes and relative amplitudes of the U waves and PR segments of a typical subject and to suggest the effects of increased heart rate on the waveforms when the P wave became superimposed on the preceding U wave in each of the three areas indicated by the bold arrows.
PR Segment and ST-Segment Maps at Varying Heart Rates and With Different Baselines

We considered the only reasonable times for selection of the reference voltage in the original waveforms to occur during the UP interval (the TP interval in young children) or during the PR segment. An adjustment at the J point or early ST segment did not seem reasonable for the construction of maps, since in most subjects many of the waveforms had ST-segment deflections that were considerably greater than those of the U wave or the PR segment. We compared the PR and the ST-segment potential distributions that were constructed when the same waveforms were used but different baselines were chosen, one during the UP interval and one during the PR segment. This procedure was carried out for the lower heart rates of 80–90 beats/min, when there was an isoelectric UP interval, and then it was repeated at the higher rates for baselines at the TP junction and during the PR segment.

Changes in heart rate affect the amplitude of the U wave, the PR segment, and the ST segment. However, for these observations we assumed that the slightly different heart rates produced little change in the magnitude of the currents arising from the heart since the U wave, the PR segment, and the ST-segment deflections changed little in amplitude during the time the heart rate was slowing and the superposition of the P wave on the U wave was disappearing. Thus, for the 10 subjects studied at slightly different heart rates, we considered the differences in the patterns of the maps to be due primarily to the artifact related to the baseline choices. The changes in the surface maps presented for the 18-year-old subject occurred systematically in the same body surface areas in the other nine subjects studied at different heart rates. The maps shown in figure 8 were constructed from the same waveforms illustrated in figure 7.

PR-Segment and Early QRS Maps

The correct potential distributions are those shown in figure 8, A1, for the UP baseline choice at a rate of 90 beats/min. The PR-segment map shows positive potentials over the right upper chest with negative potentials over the middle and lower anterior torso. The maximum (30 μV) was at the right shoulder and the minimum (−40 μV) was in the left axilla. This distribution was similar to the one shown by Taccardi et
Figure 8. Effect of baseline choice on potential distributions depicted for instants during the PR segment and early QRS at different heart rates. These instantaneous body surface maps were constructed from the same waveforms illustrated in figure 7. I. The correct maps are those shown in (A,1) at a rate of 90 beats/min. They were constructed from the baseline chosen in the U-P interval. When the same waveforms were used to construct the PR segment map of (A,2) all of the potentials were zero because the baseline was chosen at that time. The locations of the precordial leads are shown superimposed on this map to indicate their positions in reference to the total torso representation of the map format. The PR segment and early QRS maps shown in (B) for a heart rate of 105 beats/min were constructed from the baseline chosen at the TP junction. Note the differences in these maps (B) and those of (A,1) when the UP interval was the baseline choice at a rate of 90 beats/min. When the PR segment was chosen as the baseline for the maps at a rate of 105 beats/min, the PR segment and early QRS maps were the same as those shown in (A,2) for the same baseline choice at a rate of 90 beats/min, and they are not presented.
During early QRS, a maximum appeared on the anterior torso in the area of the negative potentials with the continued presence of the atrial repolarization maximum at the right shoulder. This pattern with two maxima, one due to atrial repolarization and the other due to early ventricular excitation, was found during initial QRS in many normal subjects when the maps were constructed from waveforms that had an isoelectric UP interval as the baseline. When the baseline was chosen during the PR segment in all of the same waveforms, the entire PR-segment map was isoelectric at zero potential (fig. 8, A2); during early QRS only the maximum due to ventricular excitation appeared in the map, and the magnitude of the left chest minimum was less than in the previous map. This comparison demonstrated that even at basal rates small but clearly detectable changes are produced in the patterns and in the magnitudes of the potentials in the maps dependent upon the baseline choice.

At more rapid rates, the PR-segment and early QRS maps constructed from the baseline chosen during the PR segment were essentially the same as those shown for the same baseline at the lower rate, and they are not repeated in the figure. However, when the baseline was chosen at the TP junction (i.e., on the U wave), the maps were altered markedly (fig. 8B). Although the overall pattern of the PR-segment map was similar to the correct pattern at the lower heart rate shown in figure 8, A1, the magnitude of the values was markedly different, with artificial increases in the absolute values of the maximum at the right shoulder and of the minimum on the left precordium. Note that these effects in the maps of figure 8B can be explained from the waveforms shown in figure 7, I.

The TP junction baseline choice produced an artificial increase in the PR-segment positive potentials at the right shoulder due to the baseline in the negative U wave in that area, and the accentuation of the negative PR-segment potentials on the left precordium was due to the baseline in the positive U wave there. These baseline artifact effects also were considerable in the early QRS map shown in figure 8B, with a pattern of positive potentials over the entire upper chest and negative potentials on the inferior torso with the QRS maximum positioned immediately above the minimum.

**ST Segment**

ST(0–50 msec) maps are shown in figure 9 for different heart rates and for different baseline choices for the same subject whose waveforms and PR-segment maps were presented in the previous two figures. The correct map is the one shown for the rate of 90 beats/min with the baseline in the UP interval (fig. 9, A2). It is similar to an ST-segment map shown by Taccardi et al. for a normal adult, and it illustrates the major features of normal body surface ST-segment potential distributions. The maximum was located near the heart on the left precordium with an absolute value (210 μV) that was greater than that of the poorly-defined minimum (−30 μV), which was located at a distant area. Note the low-level negative potentials on the anterior inferior torso and the low-level positive potentials on the anterior upper right chest. These low level potentials changed in sign when the same waveforms were used to construct the map with the baseline in the PR segment (fig. 9, A1). Positive potentials were depicted now on the inferior torso anteriorly, and negative potentials were on the upper right chest. The absolute value of the maximum was slightly greater, and both the maximum and minimum shifted slightly in position. Review of the original waveforms (fig. 7, I) provided a ready explanation for these regional differences in the ST(0–50 msec) map. Note that the PR segment was positive at the right shoulder region. Therefore, when it was chosen for the baseline, the associated ST segment was measured at a lower voltage in that region. The negative PR segment on the lower torso was associated with an ST segment that was also negative, but with a value closer to zero. When the negative PR-segment potentials became the zero reference voltage, the negative ST-segment potentials in that area assumed slightly positive values in the map.

At a heart rate of 105 beats/min the ST(0–50 msec) map constructed from a baseline during the PR segment (fig. 9, B1) was almost the same as its counterpart shown in (A,1) for the lower heart rate. The values of the extrema varied by only 10 μV between the two; however, this resulted in a medial shift of the location of the minimum. The significance of this slight shift was difficult to interpret in view of the similarity of the values in that region. When the TP junction was used for the base of the line for the same waveforms at the increased rate, drastic changes occurred in the ST(0–50 msec) map (fig. 9, B2). Prominently positive ST-segment potentials were depicted on the right upper chest and shoulder, and the absolute values of the maximum and minimum changed considerably. These regional ST-segment pattern differences produced by the different baselines at the TP junction and PR segment were due to the following: The marked increase in positive potentials at the right shoulder was due to the TP baseline occurring in negative U waves on the right upper chest (fig. 7, I, position A), and the decrease in the left precordial maximum was due to the zero baseline reference with respect to the positive U waves there (fig. 7, I, position B). Also, the accentuated negative ST-segment potentials on the lower anterior torso were due to choosing the voltage reference with respect to the positive U waves there.

**Discussion**

**U Wave**

It is sometimes stated that in most normal ECGs the T wave and the U wave are separate with the TU junction at the baseline. The results of our studies indicate that most normal people have overlap of the T and U wave to produce TU fusion waves. That is, elevation of the junction of the downstroke of the T wave and onset of the U wave occurred in 44 of 55 normal subjects 8–60 years old. We account for the
difference between these results and the established idea on the basis that the waveforms used for constructing body surface maps provided sampling of all the body surface areas, while previous analyses were limited to waveforms recorded from fewer leads, i.e., the limb and precordial leads. Heart rate, elevated blood pressure, and potassium concentration affect the magnitude of the U wave. Since these subjects had normal blood pressures, and presumably they had normal blood concentrations of electrolytes, it would appear that the TU fusion waves represent the normal state of the heart.

These results showed 1) the spatial distribution of the positive and negative U waves; 2) the similarity of the locations of these parameters of the U wave with those of the PR segment, but of opposite sign; 3) the same locations of the maximum voltage of the T wave and of the U wave; and 4) that most normal subjects had TU fusion waves. These results suggest that there are similarities of the "cardiac electrical generator" (i.e., the spatial distribution of intracellular potentials) during the T wave and the U wave. These results are consistent with the theory of Hoffman and Cranefield that in some way the U wave is related to action potentials of the Purkinje system, as emphasized recently by Watanabe. We would add

**Figure 9.** ST-Segment average maps at varying heart rates and with different baselines. Each ST (0-50 msec) map represents the potential at each location averaged over the first 50 msec of the ST segment. The brackets on the waveforms indicate the 50 msec interval depicted in the associated map. The maps were constructed for the same subject whose waveforms and PR segment maps are shown in figures 7 and 8, respectively. The correct map is the one in (A,2) for the reference zero voltage baseline in the UP interval at a rate of 90 beats/min.
that rapid filling movement of the heart, which occurs
during the U wave, should be considered as a con-
tributing factor to the shape of the TU fusion waves. 
This contribution would be produced by movement of 
the cardiac sources toward the anterior chest leads, 
since the most prominent TU fusion deflections (fig. 5, I) 
were located directly over the heart. If the U wave is 
related to the Purkinje system, movement of those 
endocardial areas located near the anterior torso elec-
trodes would have a much greater effect on the 
recorded U waves than would movement of more dis-
tant endocardial areas. Thereby, movement of the im-
mediately underlying endocardial areas toward the 
anticipate chest surface would have the same effect as 
moving the recording electrodes closer to the electrical 
resources. The result would be a relative increase in the 
voltage at the recording site simultaneous with a slight 
increase, no change, or even a decrease, in the cardiac 
currents.

The U-wave isopotential maps in individual subjects 
(fig. 4) had positive isopotential lines that appeared 
irregular in shape. The U-wave body surface maps of 
Nahum and Nuland demonstrated markedly 
irregular positive isopotential lines during the U wave 
as well as during the T wave. Their results have been 
used to support the thesis that U waves are generated 
by afterpotentials that occur in the areas of the 
papillary muscles. The suggested link between the 
locations of these heart sources and the body surface 
U wave potential distributions is that the maps of 
Nahum and Nuland depicted complex contours in the 
positive equipotential lines at the peak of the U wave, 
pointing to multiple discrete muscle areas forming a 
complex electrical source in the heart. However, our 
results did not demonstrate the marked irregularity of 
the isopotential lines during the U waves or the T 
waves. Our results also differed from theirs in that the 
peak U wave values we found were low-level poten-
tials (30–140 µV). Their U wave maps in normal sub-
jects depicted maxima of 500 µV and 600 µV, values 
considerably greater than those we have encountered 
in the U wave maps of any subject and in excess of 
that found in the T wave maps of some normal sub-
jects.

PR Segment and ST Segment

In our group of subjects, positive PR segments oc-
curred only on the right upper chest and negative PR 
segments occurred only on the middle and lower 
anterior torso. Since atrial activation appeared to be 
normal from analysis of the waveforms and the P 
wave body surface maps, we assumed that atrial 
repolarization also was normal. The locations of the 
positive and negative PR segment deflections of the 
composite map (fig. 6, II) were reflected well in the 
instantaneous low level isopotential maps during the PR 
segment (fig. 8, A1). The clarity with which the 
positive and negative regions were demarcated with 
slight overlap between the two for the total group of 
individual subjects (fig. 6, II) suggests that surface 
maps of the PR segment might enhance the ability to 
detect and evaluate primary atrial repolarization ab-
normalities, as occur in patients with pericarditis.

A common problem in the interpretation of stan-
dard ECGs is presented by a scalar tracing that has a 
slightly negative ST segment, even at basal rates. It is 
often difficult to know if the ST segment is negative 
because of superposition of a negative Ta wave (atrial 
repolarization) on the ST segment or whether 
the negative polarity of the ST segment is primarily the 
result of ventricular currents during early ventricular 
repolarization. We could not define the magnitude of 
the effect of superposition of atrial repolarization 
potentials on the early ST segment in these normal 
subjects. This was primarily because in the ST-
segment maps there was variability from subject to 
subject in the patterns and magnitudes of the positive 
potentials that were due to ventricular repolarization. 
However, the polarity of any possible atrial 
repolarization effects on the ST segment was defined 
clearly on a regional basis (assuming a normal atrial 
repolarization sequence). Positive Ta potentials could 
be superimposed on the ST segment only in the region 
of the right upper chest and negative Ta potentials 
only in the region of the middle and lower anterior tor-
so, the positive and negative PR segments being con-
fined to these regions (fig. 6). In these normal subjects 
the question of false polarity of ST segments occurred 
only on the right upper chest anteriorly (false positive 
ST segment) and on the lower anterior torso (false negative ST segment). The question of false polarity 
of the ST segment did not arise in the anterior precordial region, where the PR segments were most 
negative, because all of the normal subjects had 
prominently positive ST-segment deflections there as 
illustrated in figure 9, A2 for a normal ST-segment 
potential distribution.

Methods of Recording Low-Level Potential Distributions

The construction of accurate isopotential maps is 
based on the requirement that there are no potential 
differences on the body surface at the time the zero 
reference level for the voltage measurements in each 
waveform is chosen. However, some of the conditions 
for which surface maps may be most useful 
diagnostically are those which result in an inability to 
meet this requirement. It is clear that this require-
ment is not fulfilled when either or both of the follow-
ing conditions exist: 1) There is superposition of the P 
wave or QRS on the preceding U or T wave in any 
lead, such as shown in this study or when prolonged 
PR or QT intervals are present, or 2) there is current 
flow from one area of the ventricle to another during 
the resting phase of action potentials, as in ventricu-
lar ischemia.

An apparent solution to the zero voltage baseline 
dilemma for either of the above conditions is to use 
DC coupled amplifiers to identify the zero reference 
voltage. We do not believe that in practice the use of 
DC amplifiers will offer an improvement over AC 
amplifiers (low-frequency response of 0.1 Hz). A 
minimum number of leads, such as 24, is required to
produce high-quality body surface maps in patients, and it has not been shown that electrode contact potentials can be maintained constant simultaneously for multiple electrode sites (or even for one site at this low level). Furthermore, even with DC amplifiers there is no way to identify the zero reference voltage for each lead if the recording system is connected to the patient after either conditions 1) or 2) above exist.

In practice, when condition 1) exists, i.e., superposition of the P wave on the U wave, the results indicate that the PR segment is the best baseline choice because it represents the time of least effect due to heart currents. With the PR segment as the baseline, detectable systematic changes were produced in the ST-segment map patterns, although the alterations were slight in these normal subjects (fig. 9A). The use of the TP junction as a baseline when the heart rate exceeded 105–110 beats/min produced artifically prominent negative ST-segment potentials on the lower torso (fig. 9, B2). This type of artifactual change is especially misleading, since the artifact may simulate the patterns that one is looking for in abnormalities. The baseline artifact occurred over broad regions, rather than producing potential differences over short distances, and was related to the locations and shapes of the U wave and the PR-segment deflections.

The implication of these results is significant in that the timing of the reference baseline is critical in determining the pattern of the depicted maps throughout the cardiac cycle. For example, some maps are recorded when the patient has an elevated heart rate and there is superposition of the P wave on the U wave and terminal T wave. The QRS and ST-segment map patterns that are constructed from a “TP-junction” baseline in these waveforms are distorted on the anterior chest by an element of potential representing currents of terminal ventricular repolarization during overlapping T and U waves. These considerations are especially important in interpreting precordial and body surface maps that are recorded during exercise, as shown in figure 9.

Linear shifts in the waveforms were easily correctable by the linear interpolation method described (fig. 3). However, we had no solution for non-linear drift other than to discard the waveforms at the time of the original recording and record others free of this artifact. Numerous methods have been suggested for removal of artifact due to non-linear baseline drift in ECGs, such as the process recently described by Meyer and Keiser for single-lead analysis of many consecutive heart beats. We approached this problem in a hybrid fashion by mixing human judgment with the computer display of the waveforms to achieve as much precision as possible for a few beats in each of the numerous waveforms used to construct the maps.

If the noise on each lead is independent of the noise on the other leads, the accuracy of maps theoretically can be improved by recording with a larger, rather than a smaller, number of leads. Our experience has been that by continuing to increase the number of leads, a point is reached where the advantage of more leads is offset by the disadvantage of an increased number of waveforms with simultaneous non-linear baseline shifts, which then decrease the accuracy of the map. In this study, the non-linear shifts produced greater voltage artifact than any other type of artifact. We think that in clinical practice the use of surface mapping systems with a limited number of leads, such as 24–32, may result in more accurate potential distributions compared with those constructed from a much larger number of recording sites. However, for the 150 leads used in this study the results were gratifying, since they indicate that under specified clinical recording conditions it is possible to obtain an accurate spatial representation of low-level potentials (20–250 μV) over the entire torso.

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