Effects of age, sex, and body habitus on QRS and ST-T potential maps of 1100 normal subjects

Larry S. Green, M.D., Robert L. Lux, Ph.D., Charles W. Haws, M.D., Roger R. Williams, M.D., Steven C. Hunt, Ph.D., and Mary Jo Burgess, M.D.

ABSTRACT Body surface potential maps provide more detailed regional cardiac electrophysiologic information than the standard electrocardiogram. We performed a large-scale study of a normal population to form a comparison base for evaluation of the clinical utility of this technique. We analyzed body surface maps from 1113 normal subjects from 10 to 80 years old to detail map features as a function of age, sex, and body habitus. Maps were analyzed by visual inspection and by a spatial and temporal data reduction technique that allows statistical comparison of map features. On average, both QRS and ST-T potentials decreased with increasing age. Potential pattern distributions remained constant from 10 to 40 years. Beyond age 40, larger numbers of maps from normal subjects showed depolarization patterns consistent with delayed activation of the left anterior fascicle, despite normal 12-lead electrocardiograms. Only minor QRS potential amplitude and distribution differences were noted when male and female subjects were compared within groups of similar age and body habitus. Male subjects consistently showed greater average T potential amplitudes. Slender body habitus was associated with a more horizontal "zero" potential line. In female subjects over age 40 there were more extensive low-level negative potentials recorded over the precordium during the ST segment than in men. This study defines the range of normal body surface potential maps in a large clinically normal population and provides a basis for qualitative and statistical comparison with map features of patients with disease.

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SUBSTANTIAL EVIDENCE suggests that body surface potential mapping is useful for diagnosing states not detectable by the standard 12-lead electrocardiogram. Clinical studies indicate the technique provides improved recognition of old myocardial infarction, localization of anomalous atrioventricular conduction paths, and recognition of myocardial infarction and left ventricular hypertrophy in the presence of intraventricular conduction block.1-5 In addition, special analyses of body surface QRS isoarea maps show promise for detection of cardiac states that indicate a risk of development of life-threatening ventricular tachyarrhythmias.6-8

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Definitive evaluation of the technique's clinical utility for these and other purposes must include statistical comparisons, and statistical assessment requires definition of the range of potential patterns in a large population of normal subjects. Studies reporting body surface potential patterns in normal subjects have thus far consisted of relatively small groups, largely of those under the age of 50.9-11 There are no large-scale published studies of map features categorized by subject age and in particular no information is available on map features of more than a few subjects over age 50.

In this study we report analyses of body surface potential maps from 1113 normal subjects from 10 to 80 years old. To our knowledge, this represents the largest single library of normal body surface potential maps available. We report details of age-related potential patterns and also the effects of body habitus and gender on body surface potential map patterns. Included in tabular or graphic form are features of body surface potential maps that have been reported as clinically useful as well as other features that there is reason to believe could be useful. The maps were also analyzed by a method of map representation we devel-
Methods

Subject population. Body surface potential maps were obtained from 573 clinically normal male and 540 clinically normal female subjects from 10 to 80 years old who were seen in a screening clinic organized to study the genetics of hypertension. A majority of subjects in the population were either spouses or nonfamily members of individuals believed to be at genetic risk of developing hypertension.

All subjects gave informed consent for body surface mapping and the study protocol had prior approval of the Institution Review Board of the University of Utah. Each subject gave a detailed medical history and underwent physical examination by an internist, a standard 12-lead electrocardiographic study, and determination of serum electrolytes, blood sugar, BUN, cholesterol, triglyceride, and high-density lipoprotein (HDL) levels. All subjects were normotensive with normal fasting blood sugars and normal electrolytes at the time of study. None of these subjects had a history of chest pain or cardiac dysfunction and physical findings in all were normal. All 12-lead electrocardiograms were read as normal by two independent physician-readers.

Body frame size was determined from wrist size at the smallest circumference distal to the styloid process of the radius and height without shoes. Frame sizes and weights in light clothing were then compared with frame and weight standards to classify patients as slender (10 or more pounds less than ideal weight), average (average weight for frame size), moderately overweight (20 to 49 pounds overweight), very overweight (50 to 99 pounds overweight), or extremely overweight (over 100 pounds overweight). Table 1 summarizes the normal subject population by age, sex, and body habitus.

Body surface maps from some normal subjects over 40 years old revealed a pattern similar to maps obtained from patients with known inferior wall myocardial infarction. For this reason, body surface maps from a separate group of 34 patients with documented inferior wall myocardial infarction were used for comparison with a subset of maps from those in the normal population over the age of 40. Inferior wall myocardial infarction was diagnosed by a history of typical acute chest pain, ST segment elevation, appearance of Q waves of 30 msec or more in leads II, III, and aVF of the standard 12-lead electrocardiogram, and typical rise and fall of levels of creatine kinase and lactic dehydrogenase isoenzymes. Body surface maps were obtained 10 days to 3 weeks after infarction at a time when ST segment abnormalities had resolved and patients were hemodynamically stable, with normal electrolytes, and were taking no antiarrhythmic drugs.

Recording techniques. A 32-lead body surface map recording system developed in our laboratory was used to record maps. The 32-lead array was derived from a 192-lead array by a sequential selection technique aimed at reduction of spatial redundancy in sampling body surface potential distributions. All maps in this study were displayed and analyzed as 192-lead maps estimated from recordings obtained with the 32-lead array. Ability of the 32-lead array to estimate 192-lead maps has been tested in a variety of patients with an average root-mean-square (rms) voltage error of only 35 µV and a correlation coefficient of .97. Methods of lead selection and comparison have previously been reported in detail.

A Wilson central terminal served as the reference electrode. All 32 leads were simultaneously recorded at a sampling rate of 1000/sec by multiplexer recording techniques and digitized data were stored on a floppy disk.

### Table 1

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S = slender (10 pounds or more less than ideal weight); A = average weight; M = moderately overweight (+20–40 pounds); V = very overweight (+50–99 pounds); E = extremely overweight (+>100 pounds).

Map processing. An interactive computer program was used to baseline- and gain-adjust the electrocardiograms. Five to fifteen consecutive complexes were averaged after time alignment by cross correlation. In this approach a QRS from a high-amplitude lead was manually delineated from a 5 to 20 sec record with use of the cursor of a graphics terminal. This template waveform of n milliseconds was compared to each n millisecond segment of the original waveform by cross correlation. The resulting sequence of correlation values, one for each millisecond of the record, showed well-defined, sharp peaks when the template matched each QRS, and provided temporal fiducial points in each beat for alignment. Since these data were all from normal subjects who were in sinus rhythm, the purpose of averaging was to reduce beat-to-beat differences since these were not desired in this study. The average was considered a "representative" beat. Data were analyzed at 1 msec intervals and body surface isopotential maps were printed at 5 msec intervals during QRS and at 10 msec intervals during the ST-T wave.

Contour lines of maps were displayed at 0.1 mV increments during QRS and 0.05 mV increments during the ST-T unless otherwise noted. Distribution of maximum R wave voltage, minimum S wave voltage, and average ST segment amplitude over the first 80 msec of the ST segment in each lead were printed for each subject. For each subject, a voltage curve representing the rms voltage at all sites during one complete cardiac electrical cycle was calculated. This allowed accurate determination of QRS, and of ST-T duration taking potentials recorded from the entire thorax into consideration. All maps were individually checked for signal quality. Sites at which signals were noisy or absent were estimated from all other sites with least mean square estimation and only noise free maps were included in the data base.
Body surface maps were also processed by previously reportedly spatial and temporal data reduction techniques that permit accurate statistical representation of each patient map by 216 independent coefficients. Each spatial potential distribution (each map frame in the sequence of frames for each patient) was first mathematically represented along a set of 12 orthonormal basis vectors, each with a dimension of 192. The resulting QRS and ST-T sequences were time normalized to 150 samples each and then mathematically represented along sets of 12 and six orthonormal temporal basis functions, respectively. Justification for time normalization in this group of normal subjects was that differences in QRS or ST-T duration are primarily due to differences in conduction velocities or heart sizes. Time normalization permits comparison of presumably similar physiologic events in different subjects. It has the practical effect of reducing the number of temporal basis functions needed to represent the temporal sequences to a given level of accuracy. Furthermore, time normalization does not affect frequency response since the interpolation functions used are constrained to pass through the original data points. The result is that a map, characterized by at least 100,000 numbers (192 leads x 400 msec), can be represented by 216 parameters. These 216 numbers, along with the 18 temporal and 12 spatial bases, are used to reconstruct the potential at any lead and at any time in the QT interval to within errors of 64 µV rms for QRS and 23 µV rms for the ST-T. The utility of the technique is that maps from many patents and representing a variety of normal classes or different cardiac disease states may be quantitatively compared. Since the parameters are amenable to conventional statistical procedures, it is possible to identify those features that provide discriminating power between pairs of subject classes and to reconstruct maps of those differences. This approach to map analysis is very different from that of the classic inverse in which estimates of cardiac current distributions during depolarization and repolarization are sought. Instead, the technique focuses on map features that are quantifiable and statistically comparable both within and between groups of maps from patients in similar cardiac states.

Maps were grouped by age decade and into groups based on sex and body habitus. Since there were small numbers of subjects in the extremely obese and over age 80 categories, these subjects were not included in the analyses. For each group, average maps were constructed from the average of each of the 216 corresponding coefficients.

To identify those map features that were important in discriminating between each pair of map classes, a new technique of map analysis was developed. Since each patient map was accurately represented by 216 parameters along common spatial and temporal bases, averages and variances of each of the 216 parameters were calculated to represent the "average map" for each class. Since the 216 parameters were independent, features that were significantly different between any pair of map classes could be identified with the simple t statistic at any chosen level of significance. Differences of the n < 216 parameters that were statistically significant (p < .01) were then calculated. Difference maps were reconstructed from the n parameter differences and set of spatial and temporal basis vectors common to all subjects. We have designated this type of map a feature difference map. In this manner, features that were significant in discriminating between two classes of maps could be displayed as a sequence of potential distributions. More importantly, insignificant feature differences were "filtered" from the resulting maps, thus yielding an enhanced display of the important discriminating information. This approach differs from conventional map subtraction techniques in which corresponding frames of time-aligned map sequences are subtracted and express all potential differences, whether significant or not.

**Results**

Examples of map frames from average normal maps of subjects in the age ranges of 10 to 19 years, 20 to 29 years, and 30 to 39 years are shown in figures 1 to 3. Averages were calculated without regard to sex or body habitus since relatively equal distribution of male and female subjects and each body habitus category were represented in each age group. Comparable time frames from early, mid, and late portions of the QRS and ST-T deflection are shown. Features of maps from patients in these age groups were similar to those of previously published maps from normal subjects.

All maps showed negative potentials over the right superior anterior torso and right shoulder during early portions of the QRS and positivity over the left and inferior portions of the torso at this same time. The null line extended from the left superior to the right inferior portion of the torso. During later moments of the QRS, the negative pole migrated inferiorly and to the left and there was diffuse positivity over the posterior torso. Repetition of early QRS features at a lower potential in the T maps was apparent. The physiologic basis and significance of these QRS and ST-T potential patterns have been previously discussed.

Differences resulting from age in maps were exam-

**FIGURE 1.** Selected frames from an average normal map compiled from all subjects aged 10 to 19 years. Isopotential lines are 0.1 mV apart during the QRS and 0.05 mV apart during the T wave. This and all subsequent maps are displayed as a cylinder unrolled from the right midaxillary line. Vertical lines superimposed on an electrocardiogram above each map indicate time during the cardiac cycle. The top of the grid is the level of the clavicles and bottom of the grid is the level of the umbilicus.
defined by constructing feature difference maps. As explained above, these maps are based only on map coefficients that showed significant differences. Maps and coefficients were first calculated in groups defined by subject age only since, as noted above, each age decade group contained comparable numbers of male and female subjects and body habitus types. Figure 4, A, shows a feature difference map obtained by subtracting significant map feature differences of the 20- to 29-year-old group from those of the 10- to 19-year-old group. Three representative frames from early, mid, and late QRS show that the 10- to 19-year-old group on average had a slightly stronger positive pole over the anterior chest during the early QRS. Negative potentials recorded during the mid and late QRS were also slightly more prominent in the 10- to 19-year-old group.

Figure 4, B, illustrates the feature difference map obtained when map feature differences of the 30- to 39-year-old group were subtracted from those of the 20- to 29-year-old group. As was noted in figure 4, A, there was again a small decline in potential amplitude with increasing age and little change in potential distribution. The few isopotential lines in these feature difference maps (one or two for each pole) indicate that differences were small.

Although not illustrated, T potential patterns remained unchanged and potential amplitudes decreased slightly with each succeeding decade for the first four decades. The finding that potential amplitude decreased with age is in agreement with the detailed

**FIGURE 2.** Selected frames from an average normal map compiled from all subjects aged 20 to 29 years. Maps are displayed as described in figure 1.

**FIGURE 3.** Selected frames from an average normal map compiled from all subjects aged 30 to 39 years. Maps are displayed as described in figure 1.

**FIGURE 4.** A. A feature difference map constructed by subtracting statistically different map coefficients in subjects aged 20 to 29 from those in subjects aged 30 to 39. B. A similar feature difference map showing average feature differences between the 20 to 29 and 30 to 39 age groups. Maps are displayed as in figure 1. The small number of isopotential lines indicates differences are small. These two examples show the gradual decrease in absolute value of recorded potentials with advancing age.
studies of standard normal electrocardiograms by Simonson. Subjects were also subgrouped by sex and body habitus type and feature difference maps were constructed between each decade through decade four. Findings were the same as shown in figure 4 and are not illustrated.

In maps from subjects over age 40 a further general decline in body surface potentials was noted with increasing age. In addition, in some subjects over the age of 40, a potential distribution that was apparently age related was noted. Maps were obtained from a total of 372 subjects over 40. A total of 256 subjects or 69% of the group over the age of 40 had map potential distributions similar to those of younger subjects and were designated group I. Selected frames from the early portion of the QRS of an average map of this group are shown in figure 5. In contrast to this pattern, 116 subjects or 31% of subjects over the age of 40 had the potential distribution shown in the average map labeled group II in figure 5. Unlike the QRS maps of younger subjects and those from group I, maps of group II subjects had early negative potentials recorded more diffusely over the right hemithorax and the null line had an early vertical orientation in the midsternal region. Features of the early T potential distribution are not illustrated but in both groups I and II they showed repetition of the early QRS map features in younger subjects. A feature difference map (not shown) constructed to compare average group I and group II maps confirmed these potential distribution differences during the QRS and ST-T.

The mean frontal plane axis calculated from the 12-lead electrocardiograms of subjects in group I was +47.1 ± 22.8° and that from the group II subjects was +22.6 ± 20.7°. Although the difference between these two groups was significant at the $p < .005$ level, there were no other obvious differences between the 12-lead electrocardiograms of the two groups, as read by two independent observers who were unaware of the body surface potential map patterns. The frontal plane axis was normal on all electrocardiograms from subjects in groups I and II.

Since groups I and II contained nearly equal numbers of male and female subjects, gender is not the likely explanation for the different potential patterns. Body habitus may play some role since 42% of group I and 62% of group II were obese. On the other hand, it is unlikely that body habitus is the sole factor responsible for the differences in map patterns since the average map of all obese subjects over 40 years old showed the map pattern of group I rather than group II. Age appeared to be a more important factor since in each age decade after 40 there was a progressive increase in the percentage of subjects with a group II map pattern. For example, 27% of subjects in the 50- to 59-year-old group exhibited early QRS negativity over the entire right hemithorax while 63% of subjects in the 60- to 69-year-old group exhibited this finding. In both these age groups, fewer than 50% of subjects were obese, suggesting further that obesity was not the major factor in the group II pattern.

Because the map pattern displayed for group II subjects over the age of 40 resembled the map pattern seen in patients with inferior wall myocardial infarction, the average map of subjects in group II was compared with an average map from 34 patients with inferior wall myocardial infarction documented by typical history of chest pain, electrocardiographic abnormalities, and results of isoenzyme analysis. An average map from myocardial infarction patients and an average map from group II normal subjects are shown in figure 6. Figure 6, A, shows the first 20 msec of the average QRS potential maps for patients with inferior wall myocardial infarction and figure 6, B, shows comparable average QRS potential maps for the group II normal subjects. Negative potentials in the average inferior infarction map began more inferiorly on the right and crossed the sternal midline early in the QRS. These features were repeated in the T wave maps that are not displayed. These features were not seen in the

**FIGURE 5.** Comparably timed average map frames at 5 msec intervals from early portions of the QRS are shown for two different groups of normal subjects over the age of 40. Group II is distinguished by greater anterior distribution area of negative potentials.
average map obtained from the clinically normal group II. Both the QRS and T differences between group II and the group with inferior infarction that are described were apparent in a feature difference map that is not illustrated. In this particular analysis, none of the maps from normal group II subjects showed an early QRS negative pole that began inferiorly or crossed the sternal midline within the first 20 msec of the QRS. These findings are in agreement with previous studies comparing body surface map findings in patients with left anterior fasicular block and inferior wall myocardial infarction. 21, 22

Differences in maps due to gender were investigated by constructing average maps for male and female subjects in each age group and also by constructing feature difference maps between groups. Average maps were also constructed for group I and group II subjects of each sex over the age of 40. Feature difference maps were not made for separate groups defined by body habitus type because, as shown in table I, the distributions of body habitus type in each age group were quite similar for male and female subjects. Figure 7 shows a feature difference map constructed by subtraction of significantly different map coefficients in the 30- to 39-year-old female group from the coefficients in the 30- to 39-year-old male group. There are few feature differences during the QRS, as illustrated by the small number of isopotential lines, but male subjects showed slightly more prominent potentials. In contrast, as shown to the right in figure 7, men showed much greater amplitude of T waves than women. These differences between sexes were similar for all age decades and were apparent on maps for subjects over age 40 irrespective of classification in group I or II.

In a similar manner, effects of body habitus on maps of normal subjects were evaluated. Average maps for each body habitus class were constructed by decade for each sex and compared visually and by construction of feature difference maps. In female subjects, body habitus had no effects on body surface map features. The effects of body habitus in men is illustrated in the feature difference map shown in figure 8. The map for the 30- to 39-year-old group is illustrated but differences were similar in all age groups. Slender male subjects tended to show increased positive and negative voltage with a horizontal null line as shown in selected QRS map frames. T potentials are not illus-

**FIGURE 6.** A. Sequential time frames of the early QRS at 5 msec intervals on an average map constructed from maps of patients with inferior myocardial infarction. B. Comparable time frames from an average map from group II subjects. Note the more extensive inferior negative potentials in the inferior infarction map, especially in the first three time frames.
Greater in male than female subjects in each age group. In both sexes the absolute value of recorded potentials declined with age, a finding already noted in feature difference maps between age decades. The greatest change occurred between the third and fourth decades. The locations of the peak maximum and peak minimum remained constant without regard to age or sex and they are represented on the map grid in figure 9 by the star and filled circle, respectively.

ST segment potentials in normal subjects were obtained by averaging the potential recorded at each electrode site for 80 msec after the manually selected end of QRS as determined from the rms voltage vs time curve of the entire map. These values for each electrode site were then averaged for all subjects in each age decade by sex. Examples of average ST segment potential maps for decades 2, 4, 5, 6, and 7 for each sex are displayed in figure 10. Isopotential lines in these maps were displayed in 10 μV increments to show the comparatively small positive and negative potentials. In each decade, male subjects had slightly higher precordial positive potentials. Female subjects, on average, tended to have more extensive negative potentials recorded during the first 80 msec of the ST segment, especially after the age of 40. Small negative potentials were recorded over an increasingly larger area of the precordium in women in the fourth and fifth decade. By the seventh decade, the area of negative potentials over the precordium increased in both men and women.

Discussion

This study reports body surface potential data from a large population sample as well as a statistical analysis of the effects of age, sex, and body habitus on map potentials. Unlike previous reports involving body surface maps taken from normal subjects, this study is many times larger with respect to number of subjects. It also includes subjects in every decade from the sec-
ST SEGMENT POTENTIAL

**MALE**

**FEMALE**

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**FIGURE 10.** Average ST potential maps for each sex in the age groups indicated. Average ST maps were constructed by averaging ST potential for 80 msec after the J point and then averaging all sites in each sex and age group. Isopotential lines represent 10 μV increments. Female subjects over the age of 40 tended to have a greater distribution of precordial negative potentials until age 60 when distribution in male and female subjects was similar.

were considerably higher in male than female subjects irrespective of body habitus type. The physiologic basis of the higher T potentials in male than in female subjects when there are only small differences in QRS potentials is not evident.

In male subjects, body habitus type appeared to have significant effects on both absolute potential and potential pattern distribution of QRS and T potentials. Higher potentials and a horizontally oriented null line between positive and negative poles were the major feature in slender male subjects. This is most likely best explained by a thinner chest wall and more vertically oriented heart within the thorax in these individuals. Body habitus in female subjects appears to be an unimportant variable with regard to body surface maps.

Maximum and minimum potentials were greater in male than female subjects in all age and body habitus groups, but the body surface position of these two potentials was consistent for all groups.

On average male subjects had slightly higher potentials during the ST segment than did female subjects. Average maps for women from each decade after the second showed more extensive precordial distribution of small negative ST segment potentials than did average maps for men. The physiologic basis for these two findings is unclear. The possible implications of these findings with regard to exercise testing for ischemic heart disease is also unclear since records were not obtained in any of the subjects in this study during exercise.

Characteristics of maps of many subjects over the age of 40 contained a distinct QRS difference from those of younger subjects. All subjects in this study had detailed medical histories, physical examinations, and laboratory studies that indicated they were normal as judged by standard clinical criteria. The reason for progressive change in body surface potential pattern with age in some subjects is therefore unlikely to be due to clinically significant cardiac disease as judged by usual clinical criteria. Differences in body habitus are unlikely to account completely for the age-related changes in body surface maps, even though obesity is more prevalent in those over age 40 in the population of this study than it is in the population at large.

A recent review of the significance of left-axis deviation of the standard 12-lead electrocardiogram cites a number of studies indicating that the frontal plane axis of the electrocardiogram tends to move to the left with advancing age. The mean axis of the 12-lead electrocardiograms of group II subjects over 40 years old was
significantly more leftward than the axis of the electrocardiograms of group I subjects over the age of 40. It is important to note, however, that none of the subjects in group II had left-axis deviation as currently defined on standard electrocardiogram. This body surface map finding is additional evidence that the regional sensitivity of body surface mapping provides additional physiologic information over that recorded by the standard electrocardiogram. Progressive although variable fibrosis of the intraventricular conduction system with advancing age has been reported.\textsuperscript{24} Although it is not proven by this study, it seems likely that the changes in early QRS map patterns we observed were the result of progressive cardiac fibrosis of the intraventricular conduction system.

Even though evidence supports the concept that a more regionally selective examination is possible with the use of body surface potential maps, wide use of the method has been hampered by lack of a quantitative method of analysis of the large volume of data recorded for each map. In this study of a large normal population we used the rigorous quantitative method of map analysis reported from our laboratory. The method involves both spatial and temporal data reduction and results in expression of individual serial isopotential maps as 216 independent coefficients. Although this method is not directly based on a physiologic model, importantly it makes possible the statistical comparison of map features. Unlike the classical “inverse” approaches that are directed at the use of map data and detailed models of the volume conductor to estimate cardiac current distribution for individual subjects, our approach seeks to identify quantifiable map features that are common in patients from similar diagnostic groups. Although the technique does not directly provide physiologic reasons for map features, it may contribute to a useful inverse solution by identifying those features with discriminating value to which the inverse solution can be applied.

The 216 independent coefficients that describe each map can be used in at least two ways to quantitatively analyze maps. One use described in this study first involves independent grouping of subjects or patients by clinical characteristics or results of other diagnostic testing. Corresponding map coefficients from all subject maps in a particular clinical class are averaged and an average body surface map for that class is constructed and compared with average maps of other groups. Differences between average maps can be displayed by construction of feature difference maps and by subtraction of only those map coefficients that differ to a statistically significant degree. Map differences so identified can then be related to known electrophysiologic differences between classes. A potential limitation of this approach is that all average maps represent data that has been time normalized. Comparisons between average maps of different patient classes therefore carries the possible risk of time-alignment errors. This particular type of error would be least for comparisons made during early portions of the QRS.

A second use of the 216 independent coefficient map descriptors likely to have the greatest clinical utility is the statistical diagnostic classification of individual maps. This technique again initially requires patient classification by independent means. Maps from a small number of patients in each class are used as a “training set” and are compared in terms of map coefficients. Those map coefficients that statistically differentiate each group pair are identical. Maps from additional patients are then statistically compared with each training set and maps are sorted into diagnostic categories. Nonlinear, parametric decision rules based on likelihood ratios are presently being evaluated for this purpose. This technique requires a large normal data base. Accuracy is also limited by the range of diagnostic categories available and the adequacy of each training set.

This study demonstrates the feasibility of a statistically based analysis of body surface maps from a large population sample. The study also reports body surface map features in normal groups defined by age, sex, and body habitus. The clinical utility of the methods of body surface map analysis described can only be determined by comparing a large normal data base of body surface potential maps with maps recorded from a significant number of patients with specific cardiac diseases documented by independent means. Such studies are in progress.

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