Body Surface Distribution of Electrical Potential During Atrial Depolarization and Repolarization

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SUMMARY Limited information is available documenting body surface isopotential distributions during atrial excitation and recovery. To expand the current data base, body surface isopotential maps from 40 normal subjects were examined. Data were acquired at a gain of 10,000 and isopotential distributions constructed at 2-msec intervals from the onset of the P wave to the onset of the QRS complex. During the initial half of the P wave, a left precordial maximum dominated the distribution. Negative potentials existed over the upper back. Subsequently, the maximum migrated to the left; negative potentials moved into precordial areas. Near the end of the P wave, the maximum shifted to the left back as a minimum evolved over the midprecordium. This minimum increased in intensity and remained stationary throughout the PR segment. These patterns are consistent with previously reported epicardial records from canine preparations documenting initial right and then left atrial activation, and repolarization beginning before the end of excitation and enveloping much of the posterior atrial epicardium with low-level positive potential. All distributions had but a single maximum and/or minimum, consistent with a single dipole equivalent cardiac generator.

This study was undertaken to extend the information on instantaneous surface potential patterns during atrial activation and recovery. In addition, the surface correlates of the mean cardiac generator during atrial depolarization were derived.

Methods
Forty subjects, ages 20–34 years old, were studied. All were clinically normal as defined by history, physical examination, chest roentgenography and standard 12-lead ECG. Left atrial size, as defined by M-mode echocardiography, was uniformly within normal limits. Voluntary, informed consent was granted before study.

Electrocardiographic signals were recorded from 150 silver-silver chloride disc electrodes placed on the anterior (100 electrodes) and posterior (50 electrodes) thoracic surfaces. The sampled area extended from the level of the clavicles to the inferior margin of the rib cage. Additional electrodes were placed on the distal extremities to record standard bipolar and unipolar limb leads and to derive the Wilson central terminal potential.

Electrical signals were amplified 10,000 times by a bank of custom-designed, low noise (4 μV, peak-to-peak) differential, capacitor-coupled amplifiers. All thoracic electrode voltages were referenced to the Wilson central terminal potential.

Signals were acquired in five sets. Each set consisted of 30 electrode potentials plus standard leads I, II and III. These three standard leads, recorded with all five sets of electrode grid potentials, verified the stability of recordings over the entire data acquisition period and the correct merging of the data sets. A 20-second epoch of data for each electrode group was digitized on-line at a sampling rate of 500 samples per channel per second.

The five data sets were merged and individual grid electrode wave forms were averaged to form one set of 150 unipolar thoracic wave forms. Time alignment of wave forms in each of the five data sets was ac-
completed using a previously described hardwired QRS trigger.

Beats to be averaged were selected by an automated numerical routine, comparing one PQRST wave form with another and yielding a value or "wave form index" (WFI) of unity if the two were identical. First, one standard bipolar limb lead, usually lead II, was selected for study and referred to as the "monitor channel." Second, the first beat recorded from the monitor channel in the first electrode set was compared with each subsequent cycle in that lead and data set, and wave forms with WFI values of 0.8–1.2 were averaged to form a standard reference beat. Next, all monitor channel ECG complexes from each of the five data sets were numerically compared with the standard reference beat and cycles with WFI values of 0.8–1.2 were identified. Wave forms recorded from each grid electrode during cycles with monitor channel complexes so selected were then averaged. This method served to reduce random noise recorded at the high gain used, and obscured the previously reported respiratory cycle-related variations in wave form morphology.

Adequacy of these methods was assessed in three ways (fig. 1). First, results of the numerical routine comparing each cycle of the monitor channel in each data set with the average reference beat were plotted (fig. 1A). Second, averaged complexes of standard leads I, II and III from each of the five data sets were superimposed upon one another (fig. 1B), verifying successful time alignment and stability of the subject during the recording period. Last, plots of the first selected beat of each of the 150 grid leads, the averaged beat for that lead and the outcome of the numerical technique comparing the first selected beat of that lead to each subsequent cycle were plotted. This permitted assessment of noise levels as well as the validity of the averaging method relying upon a single lead, i.e., the monitor channel.

Isopotential maps were constructed from these 150 averaged unipolar wave forms. Onsets and offsets of the P wave and PR segment were manually determined from plots of root-mean-square (RMS) potential, and maps were drawn at 2-msec intervals from the onset of the P wave to the end of the PR segment. The terminal 50-msec segment of the TP segment served as a baseline. A combined linear-bilinear interpolation routine was used and contour lines were drawn at 10-μV intervals. Isopotential patterns were displayed in composite video form and photographed by a computer-controlled microfilm camera assembly for later examination.

**Results**

All subjects were in normal sinus rhythm. P-wave and PR-segment durations were 97.3 ± 11.8 msec (range 72–115 msec) and 54.7 ± 16.3 msec (range 32–73 msec), respectively.

**P-wave Isopotential Mapping**

Isopotential maps depicting the electrical field during portions of the P wave are presented in figures 2 and 3. Patterns were consistent in all subjects; therefore, data from one representative study will be presented. Figure 2A depicts the potential distribution 4 msec after the onset of the P wave. A low-level maximum is located in the left midprecordial zone and dominates the field. The left anterior chest and the lower right anterior and posterior chest electrodes display positive potentials, while electrodes on the upper back sense negative potentials, without a discrete minimum.

By 16 msec into the P wave (fig. 2B), magnitudes of positive and negative potential increase but the locations of the maximum and the zero potential line remain stationary. Subsequently (24 msec, fig. 2C), the maximum intensifies and either remains fixed in location or shifts slightly to the right to overlie the sternum. Zero potential lines continue to remain stationary.

Next, the zone of maximal positive potential moves leftward (36 msec, fig. 2D) and negative potentials encroach upon the lower right anterior chest. This leftward migration of the maximum and inferior movement of negative potential continues to the peak (fig. 3A) and onto the downslope (fig. 3B) of the P wave. By 68 msec into the P wave the maximum has shifted to overlie the V₆ electrode position; at 80 msec, it is to the left of or posterior to the V₆ site, while

**Figure 1.** (A) An example of one standard reference beat (upper left) and values of the wave form index (WFI) of each set of data (numbered 1–5) to the standard reference beat. Brackets enclose values of the index of 0.8–1.2. Each cross represents one beat. A high degree of uniformity of wave forms is indicated. (B) An overlay of five averaged wave forms from each of the five data sets for each of standard leads I, II and III, indicating uniformity of morphology and accurate time alignment. The scale figure (lower right) corresponds to 200 msec (horizontal) and 1 mV (vertical).
negative potentials now engulf the right chest, both anteriorly and posteriorly. Finally, as the P-wave terminates (fig. 3D), a minimum dominates the anterior chest isopotential pattern.

All isopotential maps studied were characterized by a single maximum and/or a single minimum. Definite "pseudopod" formation, or the simultaneous presence of greater than one maximum or minimum, was never observed.

PR Segment Isopotential Mapping

Potential patterns during the PR segment are presented in figure 4.

The distribution of electrical potential during this phase of atrial recovery is dominated by an anterior minimum, continuous in location and intensity with that observed at the end of the P-wave (fig. 3D). This minimum, observed at a location near that of the
anterior maximum early in the P wave (fig. 2), remains stationary or migrates slightly to the left, while increasing in magnitude until ventricular activation begins. All PR-segment isopotential maps had a single minimum.

**P wave and PR-segment Wave Forms**

Plots of the P-wave and PR-segment wave forms from the anterior (right midaxillary line to left midaxillary line) grid electrodes are presented in figure 5.

Wave form morphologies are as anticipated from the isopotential maps (figs. 2-4). P-wave recordings from sites on the right upper anterior thorax were uniphasically negative, while those on the left precordium and the lower right chest zones were uniphasically positive. Biphasic patterns, with initial positive deflections, were recorded from sites in the upper parasternal zones. The PR segment was negatively displaced relative to the TP segment in all anterior areas except the uppermost anterior thorax.

**P-wave Isoarea Mapping**

To evaluate the mean P-wave forces, isoarea maps22, 23 were constructed. First, areas under the P wave in each of the 150 electrode recordings were calculated using Simpson’s method for numerical integration.24 Areas were determined from the onset of

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**Discussion**

Detailed evaluation of atrial electrical activity is more difficult than is the study of ventricular forces.
First, the magnitude of the generated potentials during the P wave and PR segment are low, requiring high-gain, low-noise recordings, often coupled with signal averaging methods, as well as close attention to baseline selection. A discussion of the problems related to low-level recordings has been offered by Spach et al. Second, much of atrial recovery occurs after the onset of ventricular excitation; the considerably greater magnitude of the QRS complex effectively prohibits direct study of the complete atrial recovery cycle. Only by studying P waves not followed by QRS complexes, as in atrioventricular block, may the entire atrial recovery phase be observed on the body's surface.

Within the bounds set by these limitations, the present study has attempted to enhance the scope of atrial electrocardiography by applying methods of isopotential body surface mapping. The results may be interpreted in terms of the advantages of this technique.

First, one may relate surface electrical phenomena to regional myocardial events. Numerous studies have demonstrated the values and limitations of this application. Most have indicated that single major epicardial sources have direct counterparts on surface tracings, whereas multiple active zones may not project the specific surface patterns characteristic of each effect. This is particularly limiting if the individual sources are weak or widely dispersed. In location and similarly directed in spatial orientation. Nevertheless, one can indeed calculate surface potentials from epicardial ECG data with reasonable accuracy, providing perhaps the best proof of the correspondence of electrical events at the two sites.

Direct human atrial recordings are not available. The isopotential records from canine hearts reported by Spach et al. provide a valuable substitute, after considering possible phyllogenetic and physiologic differences. These data are particularly valuable as the use of isopotential rather than isochrone mapping permitted study of repolarization as well as depolarization. Pertinent findings included: (1) the localization of early, i.e., the first 15-20 msec of the P wave, potential changes to the right atrial epicardium followed by, but overlapping with, left atrial depolarization; (2) the existence and frequent collision of multiple, simultaneously active depolarization wave fronts; (3) the presence of both early repolarization and terminal repolarization potentials at instants near the end of the P wave; and (4) the similarity of the general pattern of spread of excitation and recovery on the atrial surface, proceeding from right atrium to left. These phenomena were later identified in canine body surface isopotential patterns, although recognition of multiple, closely located wave fronts was not possible on the body surface.

The current data suggest that the body surface distribution of potential during atrial activation is characterized by (1) an anterior maximum, initially located over the central anterior chest but later migrating leftward, and (2) negative potentials existing over much of the back initially but migrating anteriorly from the right to form an anterior minimum late in the P wave as the maximum migrates leftward. These distributions are similar to those previously reported, with two exceptions. First, we observed only low-level potentials over the right chest rather than a more clearly defined minimum early in the P wave as described by Taccardi. Second, dual maxima as reported by others were not observed. The sources of these differences are unclear.

The observed distributions may be related to the canine epicardial data. The initial position of the anterior maximum may represent the projection of high-intensity, right atrial wave fronts, generating positive potentials ahead of the presumed initial activation of the left atrium is masked by the continued activity in the more eccentric, anteriorly located right atrium. Finally, as right atrial activation is completed, the maximum migrates leftward and posteriorly to more closely reflect the continued left atrial activation. Anterior negative potentials now reflect the oppositely directed left atrial activation wave fronts.

Atrial repolarization patterns may be similarly approached. One critical difference between repolarization and depolarization epicardial potentials affecting surface projections is the significantly lower intensity of repolarization forces. For example, Spach et al. recorded excitation potentials of 1.4-2.0 mV over the left atrium and repolarization forces of only 0.3-0.4 mV on the right atrium of the dog 60 msec into the P wave. Another difference is the slower spread of repolarization, enveloping larger areas of muscle with similarly charged forces than during activation.

Thus, as repolarization commences over the right atrium while left atrial excitation continues, the weaker recovery potentials have little effect on surface potentials (figs. 3C and D). This is in contrast to canine surface patterns, which show extension of an anterior maximum as repolarization begins. The more eccentric position of the canine heart may be the explanation.

Subsequently, the mass of anatomically posterior atrial tissue is engulfed with positive potential; only small segments of lateral or appendicular left atrium remain negative. This configuration is analogous to a uniformly active electromotive surface that may be modeled as a single dipole located at the geometric center of the bounding rim and directed toward the apex of the shell. In the case of the atria, this dipole is posteriorly oriented, projecting negative potential to the anterior thorax. A discrete posterior maximum is not recorded because of the anteriorly eccentric cardiac position. Similar arguments have been forwarded to explain the dipolar nature of late ventricular activation and ventricular recovery patterns.

Spach et al. and Taccardi et al. have also reported the distribution of PR-segment potentials. The pattern of an anterior minimum described here not only confirms their observations, but also describes the tem-
poral evolution of this surface effect from P-wave potentials.

A second advantage of body surface isopotential mapping, related directly to the wide distribution of electrode placement, is the ability to detect diagnostically critical electrocardiographic information projected to areas not usually sampled. This has been clearly demonstrated, for example, in detection and characterization of the various forms of right ventricular hypertrophy. When viewed in this context, our data may serve as a baseline by which to compare distributions from subjects with the various atrial abnormalities, including both anatomic and electrophysiological aberrations.

One corollary of clinical significance may be derived from the wave forms in figure 5. Morphologies range from uniphasically positive to uniphasically negative along a line extending from right upper to the left lower anterior chest. A zone of biphasic P waves existed in an intermediate position (i.e., in the area of the right upper precordium). This zone is identified on the isoarea maps (fig. 6) as lying near the null line. In this region, which includes the site of standard electrode V1, small deviations in electrode location result in significant changes in morphology, particularly in the depth and duration of the terminal negative deflection. Thus, if lead V1 were recorded from a location more superior or rightward than standard, a P-wave pattern considered diagnostic of left atrial enlargement might be recorded. The role of such errors in reducing the specificity of diagnostic criteria is speculative.

Last, the topographic features of surface isopotential distributions may be examined to determine the presence or absence of features generally considered to reflect a dipolar electrical source or equivalent cardiac generator. These include the occurrence at any instant of a single site of peak positive or negative voltage. In contrast, the demonstration of multiple pairs of extrema is usually considered to signify a non-dipolar generator. This method does have serious limitations compared with quantitative assessment of dipolarity; but until such procedures become widely applicable to irregularly shaped, non-homogeneous volume conductors, isopotential mapping remains a readily available method with considerable historical and theoretical validity.

Data presented here suggest that electrical forces generated during atrial activation and early recovery, and detected on body surface, are consistent with a dipolar electrical generator model. Similar impressions have been offered by Taccardii for atrial activation and by Spach et al. and Taccardi et al. for atrial recovery in human studies. In contrast, considerable segments of ventricular activation have been shown to be inadequately represented by a single dipole.

These findings must be reconciled with (1) multiple wave fronts on the atrial surface of the dog, a situation most compatible with multiple dipolar sources, (2) the observation that multiple pairs of extrema may exist on the canine body surface during the P wave, also suggesting a nondipolar generator, and (3) the human data that reveal multiple extrema during the P wave. That multiple dipole sources may generate overly dipolar surface patterns has been amply demonstrated. Whether or not a nondipolar surface pattern is observed under such conditions depends on the size, geometry and contents of the enclosing volume conductor as well as on the strength, orientation and separation of the sources. It should therefore not be surprising that a similar set of sources would generate differing surface patterns when contained within a canine or a human torso. Final documentation that the effects of the conductor rather than the source cause the interspecies variability must await direct atrial mapping in man. Why others have reported multipolar patterns during the P wave is unclear, but may relate to the problems in registering low-amplitude potentials as the investigators suggest.

Similarly, mean P-wave forces as determined by the area method of Wilson et al. are dipolar. This was demonstrated by isoarea mapping and suggested by the smooth progression of wave forms (fig. 5), from uniphasic negative to positive along a line directed to the left, inferiorly and anteriorly, i.e., the classically described mean P-wave vector. Because of the overlap of atrial recovery and ventricular excitation, we could not evaluate atrial recovery or compute the atrial gradient.

These latter findings relate directly to clinical electrocardiography. First, a dipolar source as observed here may be adequately evaluated by as few as three appropriately chosen leads, as in standard vectorcardiography. If nondipolar, electrocardiographic study of the P wave would require complete exploration of the body surface to glean all available information. Second, the symbolism of a mean P-wave vector, particularly as used in evaluation of atrial dysrhythmias, is supported by the findings of a dipolar mean P-wave cardiac electrical generator.

References

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