Electrical Potential Distribution Surrounding the Atria during Depolarization and Repolarization in the Dog

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ABSTRACT

The potential distribution at the atrial surface during depolarization and repolarization was studied in intact dogs. A preparation was developed by implanting 30 to 40 miniature electrodes permanently on each atrium to record unipolar electrograms in the intact animal. Heart block was created to dissociate atrial and ventricular activity. The electrograms were recorded on magnetic tape and atrial isopotential heart maps produced with the use of a digital computer. The changing potential distribution during excitation indicated the early presence of multiple wave fronts which were related primarily to the crista terminalis, Bachmann's bundle, and a special bundle to the base of the right appendage. The interatrial septum provided a conducting bridge which had an important influence of global atrial excitation, depending on the site of impulse formation. Colliding excitation wave fronts were quite prominent. During terminal atrial excitation, repolarization maxima were present simultaneously with depolarization maxima. Repolarization was characterized by a changing potential distribution which followed the same general pattern as excitation spread; and, furthermore, the earliest areas of excitation were associated with a repolarization maximum and terminal areas of excitation were associated with repolarization minima.

ADDITIONAL KEY WORDS activation wave front atrial electrograms atrial excitation atrial repolarization cardiac electrophysiology interatrial conduction

While considerable information has been accumulated for characterizing the excitation sequence of the ventricles and the resultant body surface potential distribution, similar studies concerning atrial activity have been sparse. Recent advances in recording and data processing techniques (1-3) indicate that it is possible to overcome many of the technical problems of recording the low voltage signal of atrial activity on the body surface to explore more fully the ultimate clinical limitations of detecting atrial abnormalities from surface recordings. Since we cannot hope to rationally understand the ultimate value and limitations of atrial body surface activity until we better understand the nature of electrical activity at the atrial surface, it seemed wise to examine further two separate but closely related electrophysiologic questions. (1) What are the characteristics of the field of potentials surrounding the atrium during depolarization? (2) What is the nature of the atrial potentials generated during repolarization?

The classic work of Lewis and co-workers (4) in the dog initially presented evidence for radial spread of activation of the atria. Eyster and Meek (5) challenged Lewis's work since they found evidence of asymmetrical spread from the SA node area. Puech and co-workers (6), van der Kooi et al. (7), and Sano and Yamagishi (8) demonstrated early elongation

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of initial wave fronts which spread inferiorly from the SA node; however, these investigators concluded that atrial excitation was principally characterized by radial spread. The question of radial versus asymmetrical spread of atrial activation remains unsettled. This is due primarily to the lack of detailed physiologic data to deny or confirm the theory of concentric conduction as related to the total excitation sequence of both atria. Additionally, the nature of repolarization of a mass of myocardial tissue remains at a primitive stage of understanding. To gain insight into this latter problem, we considered the atrium to provide the particular advantage of a two- rather than three-dimensional problem due to the thin chamber walls, although the global geometry of the atrium is somewhat complex (9) because of the appendages and venous connections.

This study was designed to define the nature of the electrical potential distribution at the atrial surface during depolarization and repolarization in the intact dog. Although atrial excitation can be characterized by isochronous time lines to depict the movement of wave fronts over the atria, the time course of repolarization precludes the use of this approach. We have developed an experimental model with numerous electrodes permanently implanted on the atrial surface to record electrograms at each site and from these have constructed isopotential atrial heart maps. We thought that this approach might improve insight into the above questions by approximating normal conditions more closely than can be achieved in the open-chest dog.

**Methods**

**A. PREPARATION OF EXPERIMENTAL MODEL**

Twelve dogs weighing 18 to 25 kg initially underwent right thoracotomy with implantation of 30 to 40 insulated silver-coated copper electrodes, 1 mm in diameter, on the external surface of the right atrium. Each electrode was anchored by fine silk superficial stitch in the atrial wall. No electrodes were implanted internally, e.g., on the atrial septum. Particular attention was given to precise placement of electrodes surrounding the sinus node area, along the taenia terminalis, over the right atrial free wall toward the A-V groove, both surfaces of the right atrial appendage, the proximal superior vena cava, and on and around Bachmann's bundle. In eight animals complete heart block was produced by introducing a small iridectomy scalpel through the lateral area of the right atrium with a single incision across the A-V groove. The insulated wires were then approximated, channeled through the approximated pericardium, looped inside the chest, and brought out superficially for permanent implantation beneath the skin of the neck. Three weeks after recovery from the initial surgery, at second operation, through a left thoracotomy, 25 to 30 similar electrodes were implanted over the left atrial surface. Particular attention was given to placement in the area of Bachmann's bundle superiorly, on both surfaces of the left atrial appendage, the main body of the left atrium laterally and inferiorly, and inferiorly at the junction of the two atria (representing the exterior position of the atrial septum). The wires were looped within the chest and the distal ends

**FIGURE 1**

Lateral roentgenogram of chronic preparation shows the electrode positions over the right atrium (right superior) and left atrium (left inferior). Multiple wires on each atrium were confined to bundles and led out through the pericardium. A large intrathoracic loop was created to minimize tension at the electrode-muscle junction.
implanted beneath the skin of the neck. Due to difficulties in separating the atrium from posterior mediastinal structures, no electrodes were implanted posteriorly between the superior pulmonary venous atrial connections (1-cm² area).

B. DATA ACQUISITION

The animals appeared healthy and were quite active following recovery from the second operation. On the day of study, 5 to 8 weeks postoperatively, x-ray films were taken to confirm the position of electrode sites (Fig. 1). A plastic cube clock calibrated with 1-cm wire markings was positioned adjacent to the chest at the level of the atrium to estimate interelectrode distances from the x-ray film. Three animals had roentgenographic evidence of atelectasis of the right upper lobe, but none had evidence of pleural fluid accumulation.

Each animal was studied under pentobarbital anesthesia (30 mg/kg). The electrodes in the neck were exteriorized and connected to a...
plots; therefore, only when potentials exceeded this value were they included in the drawings depicting the potential distribution.

D. CONSIDERATIONS REGARDING THE METHODS

To gain insight into the characteristics of atrial electrical activity by depicting the field of potentials surrounding the atria, several considerations arose which required study.

1. What is the general relationship between the excitation wave front depicted by instantaneous isochronous time lines to the position of maxima and minima and the field of positive and negative potentials? Van der Kooi and co-workers (7) noted that local excitation as timed with a bipolar lead coincided precisely with the most rapid portion of the intrinsic deflection of atrial unipolar electrograms. To examine this time relationship, four dogs underwent acute atrial excitation studies whereby a bipolar electrode with the terminals 1 mm apart was used to record bipolar and unipolar electrograms simultaneously. The unipolar electrograms recorded during the acute studies in the open-chest dogs and those recorded in the intact animals with chronically implanted electrodes were compared. This showed that for comparable atrial sites the shapes of the P waves were quite similar. These data were displayed oscillographically at a paper speed of 800 mm/sec. The local excitation times were measured both from the bipolar tracings and from the simultaneous unipolar tracings by selecting points during the rapid portion of the intrinsic deflection. Only unipolar tracings which provided a single major intrinsic deflection were used for analysis. The comparison of 60 records indicated that the timing of local excitation by either method was in agreement within ±1.5 msec. On the basis of these results, local excitation times were determined from the unipolar electrograms in four intact animals to construct the activation wave front at several instants of time during atrial excitation for comparison with the distribution of potentials at the same time.

2. Since the implantation of electrodes directly on the atrial surface produces local injury, such effects, if prominent, would produce spurious values in depicting the atrial potential distribution. Three additional animals were studied to evaluate the rapidity of injury current changes. Immediately after suturing the electrodes in position, injury produced positive shifts of the curve during both P (atrial excitation) and T (atrial repolarization). These effects disappeared within 15 minutes to 6 hours. After this time, pacing different sites of the atrium produced alterations in the P and T waves, notably. The unipolar tracings coincided precisely with the most rapid portion of the intrinsic deflection of the atrial unipolar electrograms.
the upper lobes of both lungs in four animals. There was no pericardial or pleural fluid present. A thin layer of connective tissue enveloped the atrial surface and wires. In each heart, the wires were dissected free for final identification of electrode sites. Histologic studies (Masson’s stain) indicated that, except for the overlying connective tissue, the atrial muscle was normal.

3. In dogs with complete heart block, the amplitude of the P waves varied with their phase in the ventricular cycle. P waves during the first 120 msec after QRS onset showed no amplitude change; however, if the P wave occurred during the last 200 msec of the ventricular T wave, the amplitude either remained constant or decreased as much as 21% (it never increased). Another type of amplitude alteration found in three dogs was most prominent on the atrial appendages. If the P wave on the appendages occurred within 200 msec after completion of the ventricular T wave, the largest amplitude throughout the cycle was recorded. With the subsequent two or three atrial beats (without a superimposed ventricular beat) there was gradual decrease, as much as 25%, in the amplitude of the P wave prior to the next ventricular beat. This phenomenon remained reproducible for 5 hours. If the atrial beats were selected within a constant time interval between ventricular beats, the peak-to-peak amplitude showed less than 5% variability. Therefore, to minimize error in the final potential maps in the animals with complete heart block, beats were chosen for analysis within a 70-msec range within the R-R interval.

Results

A. CONFIGURATION OF ELECTROGRAMS

In the earliest and latest areas of excitation, the P and Ta waves were always of opposite polarity regardless of the site where excitation was initiated, as shown in Figure 3. The atrial T waves were predominantly upright over the right atrium and superior left atrium and negative or flat over the inferior left atrium during normal sinus rhythm. During normal sinus rhythm, the P wave was negative with a positive T in the area near the SA node, whereas over the latest area of excitation on the left atrium, the P wave was predominantly upright with a flat or negative T deflection. When the inferior lateral left atrium was paced, the curves were reversed with negative left atrial P and upright T waves over the early excited areas; the upper right atrium, which excited late, demonstrated predominantly positive P waves with negative T deflections.

The shapes of the P waves at various sites during normal sinus rhythm (Fig. 4) were similar in form with those reported by Puech and co-workers (6), van der Kooi et al. (7), and Matsuoka (10). Additionally, there were irregular waveforms with high frequency components at sites along the borders of the atrial appendages. The site on the SA node area where earliest negativity was recorded yielded entirely negative deflections; these frequently were associated with a short and small pre-potential (approximately 20 msec and 70 μv) and high frequency notching during the downstroke (7). Electrograms recorded 1 to 3 cm from the SA node on both
the taenia terminalis and Bachmann's bundle yielded rS waves while adjacent atrial areas demonstrated RS configurations. Over the lower right atrium and lateral left atrium the waveforms were primarily RS. The latest sites of excitation yielded curves that were predominantly Rs.

B. ATRIAL POTENTIALS DURING DEPOLARIZATION AND REPOLARIZATION

The results to be presented of the atrial potential distribution during atrial excitation and repolarization were the same for all dogs for any comparable pacemaker site. Minor variations will be noted where appropriate. Since the results were similar in the different dogs, the findings in a single dog with complete heart block will be used to illustrate excitation and repolarization potential distribution changes when atrial activation is initiated at three different sites: (1) sinus node area (normal spontaneous atrial rhythm), (2) area adjacent to the coronary sinus (attempt to simulate coronary sinus and/or nodal rhythm), and (3) area on inferior-lateral wall of left atrium (attempt to simulate left atrial rhythm).

In the presentation of the results, the term wave front will be used to signify the demarcation line between positive and negative potentials during depolarization. This line frequently separated closely apposed maxima and minima. Additional information concerning the relationship of the wave front as constructed from isochronous time lines and the potential distribution is presented in section C.

Normal Sinus Node Atrial Rhythm (Fig. 5).—Atrial activity was initiated by development of an isolated minimum over the SA node area. The minimum remained localized to a small area over the SA node for 3 to 5 msec and in two dogs it spread over the adjacent area 1 cm laterally on the superior vena cava; then it extended down along the taenia terminalis with the development of maxima and surrounding positive potentials (Fig. 5, top, 9 msec). Thereafter, as the minimum progressed further down along the taenia terminalis, another minimum progressed rapidly toward the left atrium in Bachmann's bundle area; additionally there was a small irregular or branch area ("pseudo-pod") along the anterior base of the right atrial appendage. This resulted in early atrial excitation characterized by a nonuniform, nonsymmetrical spread of activity which appeared more complex than previously described (Fig. 5, top, 24 msec). Note that the distribution of positive and negative potentials indicates wave fronts simultaneously progressing over the upper left atrium, superior right atrium and appendage, inferior right atrium, and also superiorly over the proximal superior vena cava. Additionally, there were considerable potential changes over the right atrium for the first 15 to 20 msec, while no changes could be detected over most of the left atrium.

Excitation of the base of the posterior right
atrial appendage started from Bachmann's bundle area; however, both the posterior and anterior surface of the right atrial appendage excited in a base-to-apex manner. Early excitation of the right atrial free wall appeared as an elongated wavefront along the border of the taenia terminalis (Fig. 5A, 24 msec). The movement of closely apposed maxima and minima toward the A-V groove produced an elongated maximum and positive potential area along the A-V groove with trailing minima and enlarging area of negative potentials (Fig. 5A, 32 msec).

The major portion of left atrium and inferior atrium depolarized after right atrial excitation had been completed. Wave fronts progressed in two directions: superiorly downward from the upper left atrium and inferiorly in a leftward direction across the area adjacent to the coronary sinus (Fig. 5A, 45 msec). These two cases require separate consideration as they progressed toward each other to complete left atrial excitation.

In one case, an excitation maximum continued superiorly on the left atrial appendage at the same time as a larger area of depolarization continued over the lateral wall (Fig. 5B, 56 msec). The sequence of spread on the left atrial appendage varied somewhat, but in all cases the inferior surface of the appendage began to excite near the base next to its right edge with base-to-apex spread. Invasion of the superior surface of the appendage started at the base as a result of the wave front progressing over the superior (posterior) wall of the left atrium. The last area of the appendage to excite was at the tip (Fig. 5B, 56 and 60 msec); in one dog this was the latest area of total atrial excitation, and in the remaining animals, the left atrial appendage excitation maximum continued to within 5 to 10 msec of final atrial depolarization, which occurred on the inferior-lateral wall of the left atrium.

In the second case, with leftward movement of the inferior right atrial maximum-minimum across the coronary sinus area, isolated excitation developed rapidly on the central inferior atrium, as indicated by the central minimum at 56 msec. The minimum can be accounted for by excitation via the interatrial septum. Thereafter, multiple maxima and minima occurred transiently on the inferior atrium, indicating wave fronts moving bilaterally from the central region. Multiple maxima and minima, caused by the converging wave fronts, completed excitation of the area adjacent to the coronary sinus (Fig. 5B, 56 msec).

The final area to depolarize (except in the one dog in which the left appendage excited last) was located along the inferior-lateral left atrial wall, as found by Puech and co-workers (6). This area was invaded by converging wave fronts indicated by the movement of separate minima progressing into the area of the maximum from a left superior direction and from the right (Fig. 5B, 60 msec).

During the final 15 to 20 msec of left atrial excitation, repolarization maxima developed over the right atrial free wall and SA node area. Two types of maxima then existed simultaneously, depolarization maxima over the left atrium and repolarization maxima over the right atrium (Fig. 5B, 60 msec).

Repolarization was characterized by development of positive potentials over the right atrium and negative potentials over the left atrium (70 msec). Thereafter, as the intensity of the maxima increased over the right atrium, positive potentials migrated as pseudopod extensions to the left over the superior and inferior atrial surfaces in a sequence similar to that of excitation spread (80 msec).

The progression of the pseudopods of positive potentials continued (Fig. 5C), following the same general pattern of excitation, while the minimum and surrounding negative potential area persisted throughout over the latest excitation areas of the left atrium. Thus, normal atrial repolarization was characterized by a minimum (sink) over the latest excitation area of the left atrium while a maximum (source) was located over the right atrium. The distance separating repolarization maxima and minima was as great as 3 to 5 cm, while that separating the excitation maxima and minima was too small to be measured.
Atrial potential distribution during depolarization and repolarization with normal sinus rhythm. These data represent the potential distribution in a single animal with complete heart block and are representative of the group. Each instant of time is shown with the potential distribution superimposed on an atrial outline of the type shown in Figure 3. The instant of time from onset is indicated above each heart map; this instant also is indicated by a vertical line superimposed on a right and left atrial electrogram. The right atrial tracing was recorded at the junction of the superior vena cava and the base of the right atrial appendage; the left atrial electrogram was recorded from the inferior-lateral left atrial free wall.

The maxima (highest potentials) are indicated by +, and the area of positive potentials surrounding the maxima is indicated by stippling. The minima (lowest potentials) are indicated by — and the surrounding negative area is indicated by diagonal lines. The demarcation line between positive and negative potentials represents zero voltage in reference to the left leg. The potential values (in millivolts) of the maxima (max) and minima (min) for each map are noted below. (See text for detailed discussion.)

A: At 9 msec, minimum over SA node = —1.8 and maxima = 0.5 to 0.7. At 24 msec, minima = —2.2 to —2.7 and maxima 0.4 to 0.7. At 32 msec, minima = —2.3 to —2.7 and maxima 1.7 to 2.5. At 45 msec, minima at left atrial appendage = —3.4 and —4.6; inferior
accurately by the electrode spacings used (3 to 15 mm).

Atrial Rhythm Initiated in Coronary Sinus Area (Fig. 6).—When excitation was initiated in an area adjacent to the coronary sinus, two wave fronts began early, as shown at 14 msec, with closely apposed maxima and minima moving superiorly over the lower right atrium and left across the inferior atrium. This progression continued over both atria. On the right atrium, the maxima and minima formed a widened front which moved over the free wall superiorly; the other wave front migrated around the inferior portion of the left atrium (30 and 43 msec). While these wave fronts were progressing over the free walls, an excitation minimum suddenly appeared on the middle superior surface of the atria (43 msec). This area was probably excited via the interatrial septum. Subsequently, wave fronts moved bilaterally away from the superior central atrium toward the wave fronts migrating upward over the free wall of each atrium (51 msec).

The remainder of atrial excitation (Fig. 6B, 62 to 87 msec) continued simultaneously in three separate areas—the right and left atrial appendages and the proximal superior vena cava. On the appendages, the wave fronts collapsed while migrating distally to the tip. The latest excitation maximum occurred on the left atrial appendage. The duration of excitation was longer than normal when the coronary sinus area was paced (92 versus 72 msec).

During the final 20 to 25 msec of atrial activation, excitation maxima and minima existed over the atrial appendages and proximal superior vena cava simultaneously with an enlarging repolarization maximum which began over the lower right atrium near the site of pacing. Note the enlarging area of positive potentials of repolarization which began inferiorly on the right atrial free wall and progressed superiorly over the right atrium and laterally over the inferior portion of the atria (Fig. 6B, 62 to 118 msec). The salient feature of the potential distribution after the completion of excitation was the presence of repolarization minima on the three terminal but separate areas of excitation (the two atrial appendages and proximal superior vena cava) while repolarization maxima were present over the right atrial free wall and inferior atrium (118 msec). Again, repolarization maxima and minima were separated more than excitation maxima and minima.

As repolarization continued, the maxima migrated toward the appendages (Fig. 6C). The minima remained stationary over the three areas representing late sites of excitation. Thus repolarization was characterized again by (1) a considerable time interval (25 to 30 msec) when both repolarization maxima and depolarization maxima were simultaneously present, (2) development of a maximum in the area of earliest excitation while minima developed over the latest areas to excite, (3) greater distances separating maxima and minima during repolarization than during normal atrial rhythm, excitation maxima at left atrial appendage = 3.0 and 3.2, inferior atrial maximum = 1.5.

B: Atrial potentials during late excitation and early repolarization during normal sinus rhythm. At 56 msec, the left atrial appendage maximum = 4.3 and minimum = −3.7; the two maxima inferiorly = 1.7 and 1.8, while the multiple minima ranged from −0.6 (coronary sinus) to −3.1 (central atrium) and −2.0 (lateral inferior left atrium). At 80 msec, repolarization maxima on the right atrium = 0.3 and 0.4 and the intervening minimum = −0.3. The excitation maxima on the left atrium = 1.4 and 2.0 and the associated minima = −2.0 to −2.6. At 70 msec, the increased intensity of the stippling surrounding repolarization maxima on the right atrium indicates the areas where values were greater than 0.5. The two right atrial maxima = 0.7 and the left atrial minima = −0.7. At 80 msec, right atrial maxima = 0.7 and left atrial minimum = −0.3.

C: Continued atrial repolarization in normal sinus rhythm. At 100 msec, right atrial maximum = 0.7 and left atrial minimum = −0.3. At 130 msec, right atrial maximum = 0.5 to 0.9; left atrial minimum = −0.3.
Atrial potential distribution with pacemaker site in coronary sinus area. The form of presentation is the same as for Figure 5. The right atrial electrogram was recorded from the lower free wall of the right atrium and the left atrial electrogram was recorded from the left atrial appendage. The initial spike in each tracing indicates the pacing artifact. The circled square pulse indicates the site of the pacing on the external atrium in the area adjacent to the coronary sinus. See text for detailed discussion.

A: At 14 msec, right atrial maximum = 1.4 and minimum = -0.6; maximum and minimum on the mid-inferior atrium = 0.4 and -0.6. At 30 msec, right atrial maximum and minimum = 2.6 and -1.9; the inferior atrial maximum and minimum = 1.3 and -1.2. At 43 msec, note the isolated minimum (-1.7) with accompanying maximum (2.1) superiorly between the appendages. Maxima over the right atrium = 0.7 and 0.8, associated minima = -3.8 and -1.3; the inferior left atrial maximum and minimum = 1.2 and -1.8. At 51 msec, the positive potentials occupying the upper left atrium and right atrium encompass the appendages; the clear area at the tip of each appendage signifies voltage levels less than ±0.2 mv. (noise level). Note the superiorly positioned central minimum (-3.3) with adjacent maximum (2.1) at the base of the left atrial appendage. Right atrial maxima = 1.2 and 1.3 and the associated minima = -1.3 (near A-V groove) and -3.3 (over SA node).
depolarization, and (4) the migration of the maxima toward the minima during terminal repolarization.

Left Atrial Rhythm Originating Inferiorly and Laterally (Fig. 7).—Following start of excitation on the inferior lateral left atrial wall, two wave fronts were indicated by the potential distribution shown at 19 msec, one progressing superiorly toward the left atrial appendage over the left atrial free wall, and the other to the right across the inferior atrium. As the closely apposed maximum-minimum migrated right across the inferior atrium, there also was spread around the base of the left atrial appendage superiorly and inferiorly (33 msec). Subsequently, the potential distribution indicated multiple wave fronts, as shown at 44 msec, with one moving right on the inferior atrium, another moving right on the superior surface between the appendages, and with continuing excitation of the left atrial appendage.

Excitation of the superior surface of the atrium was characterized by movement of the maximum-minimum rightward in the area of Bachmann’s bundle with less rapid invasion of the adjoining atrial superior surface at the base of the right atrial appendage and the intervening area between the two appendages (63 msec). Simultaneously, the wave front on the inferior atrium moved across the coronary sinus area to the lower portion of the right atrial free wall.

The sequence of excitation continued with spread along the taenia terminalis with evidence of multiple wave fronts over the right atrium such that the potential distributions shown at 76 msec and 80 msec (Fig. 7B) indicated the following: (1) activity along the taenia terminalis area with wave fronts progressing toward one another from a superior and inferior direction, (2) wave fronts progressing over the posterior and anterior medial surface of the right atrial appendage, (3) invasion of the right atrial free wall toward the A-V groove from the taenia terminalis, and (4) an additional area of isolated activity over the proximal superior vena cava. Excitation of the right atrium was completed with wave fronts moving over the free wall and appendage in a fashion similar to normal, as shown at 80 and 89 msec; i.e., there were excitation maxima and minima positioned so that the area of the taenia terminalis and Bachmann’s bundle were enveloped by negative potentials while the appendage and free wall were occupied by maxima and positive potentials. The demarcation line moved toward the A-V groove from right to left. The latest areas of excitation were on the left edge of the right atrial appendage and along the upper A-V groove at the base of the appendage (89 msec).

The initial evidence of repolarization (63 msec) was development of positive potentials in the areas on the left atrium which excited early; this occurred considerably before atrial

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B: Completion of atrial excitation and early repolarization. At 62 msec, the proximal (SVC) maximum = 1.0, and the right atrial appendage maxima = 1.7 and 2.2, and the intervening minimum = −2.0. The left atrial maxima = 5.4 and 6.3, and the superiorly positioned minima = −5.7 and −5.8. The inferior left atrial wall minimum = −0.5. Note the onset of repolarization signified by the maximum (0.3) and surrounding positive potentials on the inferior right atrium. At 72 msec, proximal SVC maximum = 0.7; right atrial appendage maximum and minimum = 3.4 and −0.8; left atrial appendage maximum = 3.0; inferior right atrial repolarization maximum = 0.5. At 87 msec, proximal SVC minimum = −0.3; left atrial appendage maximum and minimum = 5.8 and −2.4; right atrial free wall repolarization maximum = 0.9. At 118 msec, proximal SVC minimum = −0.3; right atrial appendage minimum = −0.3; left atrial appendage minimum = −0.8; right atrial maximum = 1.0 and inferior atrial maximum = 0.9.

C: Terminal repolarization. At 157 msec, note the three minima over the latest separate sites of excitation: SVC = −0.3; right atrial appendage = −0.4; and left ventricular appendage = −0.9; right atrial maximum = 0.7 and left atrial maximum = 0.3. At 180 msec, the three minima = −0.3 to −0.5; right atrial appendage maximum = 0.7.
Atrial potentials with pacemaker site on inferior lateral left atrium. The time identification electrograms beneath each map were recorded from the right atrial appendage (RA) and inferior left atrium (LA). The initiating spike represents pacemaker artifact. The circled square indicates the site of pacemaker stimulation. See text for detailed discussion.

A: At 19 msec, inferior atrial maximum = 0.8; left atrial maxima = 0.8 and 0.9; both left atrial minima = -1.0. At 33 msec, inferior atrial maximum and minimum = 1.0 and -3.1; LA appendage inferior maximum and minimum = 0.8 and -1.5; superior LA appendage maximum and minimum = 2.6 and -2.8. At 44 msec, inferior atrial maximum and minimum = 1.2 and -2.1; upper right atrial maximum = 1.4, and LA appendage maximum = 2.7; inferior LA appendage minimum = -5.2 and upper LA appendage minimum = -3.0. At 63 msec, inferior RA maximum and minimum = 3.0 and -2.0; superior RA maxima = 1.5 (medial) and 0.9 (SA node); LA appendage maximum = 2.0 and superiorly positioned minimum = -1.3. Note the area of positive potentials (early repolarization) over the lateral left atrium. The highest value in this area was 0.3.

B: Completion of atrial excitation and early repolarization. At 76 msec, note the progression of the upper and lower areas of negative potentials progressing toward one another over
activation had been completed. As excitation continued on the right atrium, a repolarization maximum developed superiorly at the base of the left atrial appendage while pseudopods of positive potentials progressed superiorly and inferiorly in the same general sequence as followed by excitation. At the completion of excitation, the potential distribution shown at 93 msec was typical in that there were positive potentials over the inferior atrium, left atrium, and in the area of Bachmann's bundle, while negative potentials enveloped the right atrium. As repolarization continued, pseudopods of positive activity migrated over the right atrium from both a superior and inferior direction (Fig. 7C, 108 and 155 msec). Again, during this portion of the repolarization the distances separating maxima and minima were much greater than the closely spaced maxima-minima of excitation. During final repolarization, positive potentials invaded areas adjacent to the minima (197 msec).

C. RELATION BETWEEN ACTIVATION WAVE FRONT (ISOCHRONOUS TIME LINES) AND FIELD OF POTENTIAL DISTRIBUTION

We constructed the isochronous time lines by the conventional method (11) to compare them to the potential distribution present at the same time. For each recording site, local activation was determined from the unipolar electrogram, with the complex curves on the appendages excluded. The derived excitation wave fronts were drawn as isochronous time lines and, independently, the atrial isopotential heart maps were drawn; then the two were compared.

Figure 8 shows two instants during normal sinus atrial rhythm. At 30 msec note that (1) there is good agreement between the position of the wave front (isochronous time line) and the position of positive potentials in the areas toward which the wave front is moving and negative potentials occupy the areas which have already been excited; (2) distant areas over the left atrium yet to be excited remain unperturbed; (3) the maxima (highest potentials) and minima (lowest potentials) occur in close proximity in the vicinity of the excitation wave front.

At 60 msec there are multiple collapsing excitation wave fronts on the left atrium. At this time note that (1) the maxima occur in the areas being invaded (source side of the wave front) and there are closely positioned minima in the areas which have just been excited (sink side of the wave front); it is not clear why the minima were located at particular sites along the wave front and not at others; (2) during late atrial excitation there are not only multiple excitation wave fronts with associated multiple maxima and minima, but simultaneously on the right atrium repolarization maxima are present.

Discussion

A. LIMITATIONS OF THE EXPERIMENTAL MODEL

The following limitations should be noted for this experimental model in the dog.

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Relationship of activation wavefronts as derived by isochronous time lines and the field of potential distribution. Local excitation times were determined from the unipolar electrograms. This shows excitation wave fronts derived from isochronous time lines (above) for two instants of time as compared to the associated potential distribution (below). Note that the area of positive potentials is located in the area toward which the wave front is moving (30 msec); the minima and area of negative potentials occur in areas previously invaded. Note the varied positions of the maxima and minima in relation to the wave front. A comparison at 60 msec indicates (1) collapsing wave fronts on the left atrial appendage and on the inferior lateral left atrial wall with positive potentials occurring in the direction of the movement of the wave front, and (2) the discrepancy between the field of potentials and position of the excitation wavefronts due to the simultaneously present repolarization of atria. Potential values of maxima and minima in millivolts: 30 msec, minima = −2.3 to −2.7, maxima varied from 1.7 to 2.5; 60 msec, depolarization LA maxima = 2.0 and 2.3, LA minima = −1.9 to −2.6; RA repolarization maxima = 0.3 and 0.4.

1. Increasing rate shortens atrial repolarization (12) and thereby alters the T waves recorded from the atrial surface. We performed pacing experiments to vary the rate and found that the atrial T wave remained constant in amplitude and form over a range of 180 to 245 beats/min; with further increase in rate, T wave amplitude increased. During the data-recording process, rate remained constant throughout all studies which involved pacing; however, there was slight variability in rate (maximum of 15 beats/min) throughout the recording sequence for the runs during normal sinus atrial rhythm.

2. Another problem concerns injury currents induced by directly implanted electrodes. We were unable to demonstrate direct injury effects in the chronic preparations; in fact, one of the impressive aspects of the study was the remarkable reproducibility of the P and T curves over long periods of time. Thus, it was our impression that atrial excitation and repolarization may be subject to change when the electrodes are manipulated during acute experiments; however, with the volume conductor intact and during prolonged anesthesia, atrial P and T waves remained remarkably constant on a beat-to-beat basis.

3. Although the number of sampling points was large, and although many of the electrodes were within 3 to 4 mm of one another, this separation still does not allow precise definition of the distances between maxima and minima associated with the propagating atrial excitation wave front. However, since the distances were larger between maxima and minima during repolarization, rather confident estimates are possible during repolarization.

B. CONSIDERATIONS REGARDING HEART ISOPOTENTIAL MAPS

Taccardi and Marchetti, in studies of the isolated turtle heart, demonstrated that multiple maxima and minima adjacent to the ventricular surface were related to separate wave fronts (13). Our studies of the atrium show a similar relationship of changing maxima and minima as related to atrial wave fronts. The significance of the zero isopotential line (with respect to left leg reference) during excitation is that its position closely approximated the distribution of the major wave fronts. However, it should be emphasized that at times when multiple areas of right atrium were being excited, the venous area of the...
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right atrium lateral to the taenia terminalis became excited while in the region of negative potentials (Fig. 5A, 9 and 24 msec).

This study of atrial excitation in the form of isopotential heart maps provides two types of additional information when compared to that presented by depicting excitation in the form of isochronous time lines. (1) Total atrial excitation duration is measured to be slightly longer, since the unipolar tracings provide potential measurements at each site throughout atrial excitation, whereas in isochronous time line maps only one instant in time is used for each recording site. Thus the field maps show potential changes in areas adjacent to very early and late excitation sites. (2) The field maps provide information during the latter portion of atrial excitation of the superimposed repolarization potential changes on those of excitation.

C. FEATURES OF ATRIAL POTENTIAL DISTRIBUTION

A salient observation of the potential distribution during atrial excitation was the establishment of multiple maxima and minima simultaneously present over various portions of the atria, indicating wave fronts progressing in multiple directions. The longstanding question of radial versus nonradial spread has been confined primarily to propagation from the area of the SA node. The changing potential distribution throughout atrial excitation indicates that excitation phenomena of the atria are more complex than can be accounted for by a simple model of purely radial versus nonradial spread. Also, colliding wave fronts were quite prominent, much more so than during ventricular excitation. They presented the major mechanism for terminating atrial excitation. This contrasts to terminal ventricular excitation, which is characterized by uniform wave fronts propagating to the boundary of ventricular muscle. This fundamental difference between atrial and ventricular excitation may be important clinically in accounting for the more frequent development of atrial than ventricular arrhythmias.

Early excitation appeared to be influenced by the prominent muscle bundles contiguous with the SA node; i.e., the crista terminalis, Bachmann’s bundle, and a prominent bundle to the base of the right atrial appendage. The results are in agreement with others that conduction velocity is increased in these bundles (8, 14, 15). The geometry of the atrium is such that with the position of Bachmann’s bundle superiorly and the crista terminalis laterally on the right atrium, early multiple wave fronts normally are established so that the potential distribution is far from symmetrical, as illustrated in Figure 5A, 24 msec. As shown by Sano and Yamagishi (8), rapid conduction down the crista terminalis establishes the major wave front over the right atrial free wall in a direction parallel to this bundle. Wave fronts were similarly aligned along Bachmann’s bundle for invasion of the posterior right atrial appendage and superior interatrial area. For beats initiated over the lateral inferior left atrium, the influence of Bachmann’s bundle and the taenia terminalis was indicated by the sudden progression of excitation minima with surrounding negative potentials over these areas once they had been invaded (Fig. 7, 63 to 76 msec). This resulted in excitation of the major portion of the right atrium in a sequence similar to normal; i.e., from the area of the taenia terminalis toward the A-V groove (right to left), although the initiating focus was on the left atrium.

These data indicate that the interatrial septum provides a conducting bridge which has an important influence on global atrial excitation, depending upon the site of origin of the impulse. These results provide no information as to specialized conduction pathways within the septum (16); however, they show the important influence of the septum on global excitation during normal sinus atrial rhythm and for beats initiated in the area in the coronary sinus region (Figs. 5 and 6). Normally, the inferior atrium was excited via the atrial septum at approximately 18 to 32 msec from onset. The start of excitation of the inferior central portion of the atrium produced colliding wave fronts for the completion of excitation of the coronary sinus area and for the inferior lateral left atrium (Fig. 5B). Also, when the atrial pacemaker was located in the
coronary sinus area, excitation superiorly in the central area between the appendages was initiated via the atrial septum at approximately 30 msec, producing wave fronts spreading bilaterally over the upper atrium.

Repolarization maxima were present during terminal atrial excitation. This perhaps was to be expected considering the time course of repolarization in atrial muscle (12).

A prominent finding was that the potential changes during repolarization followed the same general progression as activation. This contrasts with the results of Irisawa and co-workers (17) in acute studies on the dog right atrium with the use of suction electrodes, and also is different from that found by Pipberger el al. (18) for the dog ventricle in acute studies; both groups found no general pattern of repolarization. Our studies consistently showed that an initial repolarization maximum appeared in the area of earliest excitation. Following this, the major changes during repolarization occurred with extensions by pseudopods, in a sequence similar to that of excitation, from the area of positive potentials into that of the minima and surrounding negative potentials, which were located over areas that were the last to undergo excitation.

The great distances between maxima and minima during repolarization, as compared to excitation, indicate that the nature of repolarization is quite different from that of depolarization (19). Particularly, this finding suggests that the length constant (20) for current flow through atrial myocardial fibers is much greater during repolarization than during depolarization. Furthermore, terminal repolarization was characterized by the maximum becoming more closely associated with the repolarization minimum, suggesting that there is a decrease in the length constant during terminal repolarization.

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