Computer-Assisted Animation of Atrial Tachyarrhythmias Recorded With a 64-Electrode Basket Catheter

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OBJECTIVES
The aim of this study was to assess the value of a new mapping technique based on computer-assisted animation of multielectrode basket catheter (BC) recordings in patients with atrial arrhythmias.

BACKGROUND
The three-dimensional activation patterns of cardiac arrhythmias are not completely understood owing to limitations of conventional mapping techniques.

METHODS
The study included 32 patients with atrial tachycardia (AT) and 38 patients with atrial flutter (AFL). A software program was developed to analyze the activation patterns based on 56 bipolar electrograms recorded with a 64-electrode BC deployed in the right atrium (RA).

RESULTS
The total time needed for the animation of activation patterns of atrial arrhythmias was 5 ± 0.8 min. In 22 patients with right AT, the animated maps revealed that arrhythmia was unifocal in 15 patients, multifocal in 2 patients, polymorphic in 4 patients and reentrant in 1 patient. In 10 patients with left AT, breakthroughs on the right side of the septum (2 in 8 patients and 1 in 2 patients) and a left-to-right activation of the RA were demonstrated. In patients with typical AF, the reentrant excitation was a broad activation front with preferential propagation around the tricuspid annulus. In patients with atypical AFL, the reentry circuit involved one of the venae cavae and a line of block located in the posterior wall.

CONCLUSIONS
The computer-assisted animation of multiple electrograms recorded with a BC is a valuable mapping tool that delineates the three-dimensional activation patterns of various atrial arrhythmias. The technique is appropriate for complex, short-lived or unstable arrhythmias. (J Am Coll Cardiol 1999;34:2051–60) © 1999 by the American College of Cardiology
The atrial rate of 100 to 250 beats/min with at least three different P wave configurations (14). Tachycardias with different but monomorphic configurations observed or induced in the same patient were considered as polymorphic. Typical AFL was based on the following criteria: 1) typical surface ECG appearance; 2) constant beat-to-beat cycle length, polarity, configuration, amplitude and counterclockwise or clockwise activation sequence around tricuspid annulus in endocardial recordings; and 3) entrainment characteristics including manifest entrainment during pacing from the high RA and concealed entrainment during pacing within the inferior vena cava–tricuspid annulus isthmus (16). Atypical AFL was considered reentrant atrial arrhythmias with a flutter-like surface ECG pattern and an endocardial activation sequence and response to attempted entrainment inconsistent with counterclockwise or clockwise AFL (17). If no sharp bipolar electrogram was recorded in the RA earlier than 10 ms before the onset of the ectopic P wave, the arrhythmia was considered to have originated in the left atrium (18).

**Table 1. Patients’ Clinical Characteristics**

<table>
<thead>
<tr>
<th></th>
<th>AT (n = 32)</th>
<th>AFL (n = 38)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>52.8 ± 19 (range 19–78)</td>
<td>61 ± 11.5 (range 27–83)</td>
</tr>
<tr>
<td>Male/female ratio</td>
<td>16:15</td>
<td>22:16</td>
</tr>
<tr>
<td>Arrhythmia duration (years)</td>
<td>2.5 ± 2.2 (1 month to 10 years)</td>
<td>2.5 ± 2.3 (3 months to 7 years)</td>
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<tr>
<td>No. of failed drugs</td>
<td>1–3 (median 2)</td>
<td>1–5 (median 3)</td>
</tr>
<tr>
<td>Structural heart disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>6</td>
<td>11</td>
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<tr>
<td>Dilated cardiomyopathy</td>
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<td>4</td>
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<tr>
<td>Congenital heart disease</td>
<td>—</td>
<td>3</td>
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<td>3</td>
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<td>Hypertrophic cardiomyopathy</td>
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<tr>
<td>Left atrial myxoma</td>
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<td>1</td>
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<tr>
<td>Sarcoidosis</td>
<td>1</td>
<td>—</td>
</tr>
<tr>
<td>Arterial hypertension</td>
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<td>10</td>
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<tr>
<td>No structural heart disease</td>
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<td>4</td>
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Data are presented as mean value ± SD or number of patients. AFL = atrial flutter; AT = atrial tachycardia.

**Electrophysiologic study.** Written informed consent was obtained from all patients according to the protocol approved by the Institutional Review Board of our hospital. Antiarrhythmic drugs were discontinued ≥5 half-lives before the study. Patients were mildly sedated with 1 to 2 mg midazolam administered intravenously.

Before the BC (Constellation, EPT) insertion, a two-dimensional echocardiographic study was performed to determine the diastolic dimensions of the RA. These dimensions were used to select the appropriate BC size. The BC was deployed into the RA through an 11F sheath inserted from the femoral or internal jugular veins (10 patients with AF). The BC was composed of 64 electrodes mounted on eight flexible, self-expanding nitinol splines. The electrodes are equally spaced at 4 or 5 mm apart depending on the size of the BC used (with diameters 48 and 60 mm). Each spline is identified by a letter (from A to H) and each electrode by a number (from 1 to 8). Electric-anatomic relations were determined by the following criteria: 1) fluoroscopically identifiable markers (spline A has one marker and spline B has two markers located near the BC shaft); 2) recording of a large ventricular signal identified the spline(s) located across the tricuspid valve; and 3) recording of the His bundle potential. From 64 electrodes, 56 bipolar electrograms are derived (by combining 1–2, 2–3 until 7–8 electrodes on each spline).

After BC deployment in the RA, conventional catheters are introduced and positioned in the standard positions. A 5F decapolar electrode catheter with an interelectrode distance of 2 mm and 10 mm spacing between the electrodes was positioned in the coronary sinus (CS), with the proximal electrode pair located at the ostium. A quadripolar catheter with 5 mm interelectrode distance was positioned across the tricuspid valve to record His bundle potential. A simultaneous surface 12-lead ECG and bipolar intracardiac electrograms were continuously acquired with a filter bandwidth of 30 to 500 Hz, digitized (1,000 samples/s) and displayed on a high resolution monitor at a speed of 200 mm/s for inspection and review. Data were stored on optical disks for retrieval and off-line analysis. Anticoagulation was performed by bolus administration of 5,000 UI heparin, followed by continuous intravenous heparin infusion at an infusion rate of 1000 UI/h.

If AT or AFL was not present at the beginning of study, induction of arrhythmias was attempted with extrastimuli applied during pacing with cycle lengths of 500 ms and 400 ms or burst pacing from the high RA and CS. To facilitate arrhythmia induction, orciprenalin, at a starting dose of 0.5 μm/min was intravenously infused. The dose was incremented until the heart rate increased by 30%.

**Overview of software program.** The software was designed as a 32-bit MS Windows program that can be run on common office computers.

**Data access.** From the BARD LabSystem, selected sequences up to 10 s of the recorded data are binary exported.
to floppy disks (1.44 MByte). In addition to BC bipolar electrograms, up to 10 reference channels can also be exported.

**Activation recognition.** Pattern recognition or the cross-correlation method was used as an algorithm to mark the activation timing (19). The technique "compares" the signal with a pattern (mathematic sine). The result is a function that matches the signal with the sine pattern. Each time the function is higher than a user-definable percentage of the maximal matching on this channel, the nearest zero crossing is marked as an activation. The algorithm works with a user-definable sensitivity and has a preview function. Manual correction of recognized activations (deleting or moving) is available for every channel.

**Data presentation.** The timing and spread of activation are displayed in several ways: ECG view, tables and animations. In the ECG view the activation timing is marked with a red bar. In the table option, a table (also readable by standard office applications like MS Excel) can be generated by the software that contains the following information for each channel: time of all activations, interval between activations and mean and standard deviations of these intervals. During presentation as an animation, the software runs continuously over a selected period of the ECG. The local activation times were then color-coded, with red indicating the ongoing activation front, pink indicating the earliest activated regions and yellow, green and blue indicating immediately activated regions. Isochronal maps can be presented in either a planar or three-dimensional model, schematically drawn as a sphere.

**Radiofrequency ablation procedure.** In patients with focal AT, the spot of earliest activity was targeted. In patients with AFL, the anatomic approach, with the goal of producing a line of block between the tricuspid annulus and the eustachian ridge/inferior vena cava, was used. The ablation catheters used were RF Marinr (Medtronic, Minneapolis, Minnesota), with a 4-mm tip, or Blazer (EPT, San Jose, California), with an 8-mm tip. Radiofrequency energy was delivered as a nonmodulated radiofrequency current at a preset temperature mode (70°C).

**Statistical analysis.** Data are presented as the percentage or mean value ± SD. For continuous data, the two-tailed Student t test was used to test for statistical difference. Differences were considered significant at p < 0.05.

**RESULTS**

The total time needed for animation of atrial arrhythmias was 5 ± 0.8 min (with a 233-MHz microprocessor personal computer). The BC maintained good electrical contact during the cardiorespiratory cycle and provided good quality and stable multiple electrograms throughout the procedure. Stable electrograms of satisfactory quality were obtained in 49 ± 3 electrode pairs.

**Activation patterns in AT.** The AT originated in the RA in 22 patients (69%) and in the left atrium in 10 patients (31%). Response to electrical stimulation and orciprenaline showed that AT was automatic in 17 patients (6 patients with left AT) and nonautomatic in 15 patients (4 patients with left AT).

Animated maps of the ATs located in the RA showed that the earliest activity emerged from a circumvented region (focus or exit point) in 20 patients. A single focus was found in 15 patients. Polymorphic right AT was found in four patients (three patients had two different sustained tachycardias and one patient had three different tachycardias; two patients had multifocal right AT). In patients with unifocal tachycardia, the impulse originated from a single focus and was propagated radially without evidence of turning back to the site of origin (Fig. 1A). A preferential (anisotropic) conduction in superoinferior aspects of the RA, as compared with transversal aspects, was seen in all patients (Fig. 1B). The mean cycle length for unifocal tachycardias was 344 ± 67 ms. The earliest endocardial electrical activity preceded the beginning of the P wave in the surface ECG by 41 ± 9 ms. The locations of successfully ablated RA foci (including patients with multifocal ATs) were as follows: base of RA appendage in 4 patients, lateral region in 11 patients (high in 4 patients, mid in 4 patients and low in 3 patients), low posterolateral region in 3 patients, mid-posterior in 1 patient, mid-septal in 1 patient and low septal region in 1 patient. In two patients with multifocal AT, the animated maps revealed beat-to-beat variations in the site of the earliest activity, activation sequence and cycle length (Fig. 2). As seen in Figure 2C, even a slight change in the focus site was associated with a significant change in the activation sequence within the RA. In one patient, a macroreentrant AT was repeatedly induced with extrastimuli. Animated maps demonstrated continuous electrical activity throughout the cycle length. In addition, the animated maps delineated the lines of block (manifested as double potentials) and the zones of slow conduction and guided the successful ablation (see Results of ablation).

In patients with left AT, the animated maps of the RA had the following characteristics: 1) two separate breakthroughs of earliest activity arrhythmia on the right side of the interatrial septum in eight patients (Fig. 3); 2) one breakthrough of earliest activity in two patients; and 3) activation of the RA from the breakthroughs in a left-to-right direction in all patients. In eight patients, the electrical activity in the CS preceded the activity in the BC recordings. In two patients with tachycardias located in the region of the right upper pulmonary vein, the breakthroughs on the right side of interatrial septum were recorded before activity in the CS electrograms. The mean cycle length for auto-
Figure 1. A, Simultaneous recordings of the surface ECG, leads I and aVF, and 56 bipolar electrograms from the BC in a patient with focal AT. The first beat is a sinus beat. The next three beats are tachycardia beats. His bundle potential is recorded in electrode pairs F2–3 and F3–4. The asterisks show the earliest spot of activation during sinus rhythm (SR) and AT. Spline A was located in the anterolateral RA, splines B and C in the lateral region, splines D and E in the posterior region and splines G and H across the tricuspid valve. The activation times are marked with red bars. B, Animated maps of the SR (upper panel) and AT (lower panel) beats. Planar and three-dimensional options are shown. During SR the impulse emerged in the high lateral area (spline B1–2) and propagated rapidly down the lateral wall. The complete activation of the RA took 85 ms. During focal AT, the earliest activity emerged in the mid-posterior wall (spline E4–5). The activation sequence of the RA was entirely different from that of SR. The complete activation of the RA took 95 ms.
matic left ATs was 282 ± 28 ms (p = 0.03 compared with tachycardias originating in the RA).

**Activation patterns in typical AFL.** Spontaneous counterclockwise and clockwise AFs were documented in 28 and 6 patients, respectively. During electrical stimulation, seven patients with counterclockwise AFL and three patients with clockwise AFL developed both types of arrhythmia. The cycle lengths of the counterclockwise and clockwise episodes of AFL were 238 ± 22 ms and 243 ± 19 ms, respectively (p = NS). The BC recordings and animated maps showed that the reentrant activation consisted of a broad wave front directed superiorly in the septum and inferiorly in the trabeculated lateral wall with preferential propagation around the tricuspid annulus. An example of the BC recordings and the animated maps of a counterclockwise
Figure 3. Left AT. A, Fluoroscopic views of the BC. Small white circles mark the positions of electrode pairs G1–2 and H7–8 located in the high anteroseptal and low posteroseptal regions. Red letters mark splines located anteriorly. B, Simultaneous recordings of the surface ECG, leads I and aVF, and bipolar electrograms from the BC and CS. Electrode pairs G1–2 and H7–8 (arrows) record the earliest activation in the RA. C, Animated maps of the BC recordings. Left panel, First 15 ms of activation in the RA. Right panel, Complete activation of the RA.
AFL episode is shown in Figure 4. In 13 episodes of clockwise AFL, the animation maps showed that the impulse propagation followed the same route in the reverse direction.

**Activation patterns and location of reentry circuit in atypical AFL.** In all 11 episodes of atypical AFL, spontaneous in 4 patients, during orciprenaline infusion in 3 patients and during interruption attempts of AFL in 4 patients), an undulating activity without a discernible isoelectric segment was recorded on the surface ECG. The mean cycle length of atypical AFL was 183 ± 18 ms (p < 0.001, as compared with the mean cycle lengths for counterclockwise and clockwise AFL). Atypical AFL episodes degenerated in atrial fibrillation in six episodes, stabilized to counterclockwise AFL in two episodes and terminated spontaneously in three episodes. In eight episodes (73%) of atypical AFL originating in the RA, we were able to locate reentry circuits. In five patients, the reentry involved the lower RA traveling around the inferior vena cava. Common to all these reentry circuits were the early breakthroughs at the lower mid portions of the crista terminalis (Fig. 5). Cycle length was slightly higher than the rest of the group (192 ± 10 vs. 175 ± 28 ms, p = NS). The lateral and septal walls were activated in a counterclockwise direction in three patients and in a clockwise direction in two patients. In two patients, the reentry traveled around the superior vena cava and a line of block located in the posterolateral region. The reentrant impulse ran in a counterclockwise direction in both episodes. In one patient, the RA was activated from a figure eight reentry with slow conduction located in the posteroseptal region (Fig. 6). In three patients with highly diseased atria (with low amplitude electrograms and large electrically silent areas), the reentry circuit could not be located.

**Results of ablation.** With a mean of 6 ± 5 radiofrequency deliveries, 21 foci (in 17 patients with right AT) were successfully ablated. In patients with multifocal AT and one patient with three different ATs, ablation was not attempted. In the patient with reentrant AT, a linear lesion in the posterior wall terminated the arrhythmia that was not inducible with electrical stimulation and orciprenaline administration. Patients with left ATs were treated in separate procedures: two patients underwent ablation of tachycardia originating in the right upper pulmonary vein region by the transseptal approach and five patients underwent His bundle ablation and pacemaker insertion owing to coexistence of frequent episodes of atrial fibrillation. The remaining three patients were treated with drugs because of their unwillingness to undergo a repeated procedure. In patients with AFL, ablation terminated the arrhythmia in 32 patients with typical AFL, including two patients with spontaneous atypical AFL due to reentry in the lower RA. No attempts were made to ablate pacing or orciprenaline-induced atypical AFLs. A mean of 16 ± 14 radiofrequency applications was registered.

**DISCUSSION**

The present study demonstrates the animated three-dimensional activation patterns of the RA based on activation times recorded with a BC in patients with AT and AFL. The main features of this new technique are: 1) it provides a complete picture of activation for most of the RA endocardial surface. The degree of resolution is lower than that in experimental mapping systems but appears satisfactory for clinical purposes. 2) The procedure is safe and fast; thus, it is useful for unstable or short-lived arrhythmias. Importantly, the recording of only a single beat can be sufficient to enable analysis of the arrhythmogenic substrate. 3) The color-coded animation images simplify the analysis of multielectrode recordings and help in establishing the relation between activation patterns and anatomic structures.

**Animated mapping of ATs.** Atrial tachycardias account for nearly 15% of supraventricular tachycardias (20) and may occur in the presence or absence of underlying heart disease (21). They have several mechanisms (22) and multiple locations with a clustering propensity in selected regions of the RA (4,23). Different mapping techniques have been proposed to increase the accuracy of the location of automatic foci or anatomic determinants of reentry (5,6). After surgical repair for congenital heart disease, delineation of the proper location of ablation target can be difficult (11).

The animation technique was highly effective in delineating the tachycardia mechanisms and location of culprit anatomic structures. In patients with automatic tachycardias, the site of origin and typical activation pattern for focal rhythms were demonstrated in all patients. This mapping technique, based on animation of multiple bipolar electrograms recorded with a BC, was clearly advantageous in patients with complex arrhythmias. Multiple foci were rapidly located and analyzed with animated maps. Thus, the technique avoids a laborious search for multiple foci in the complex three-dimensional structure of the RA. Despite the limited number of patients with reentrant AT, considering the performance of the procedure in patients with AFL the technique could be equally effective in these atrial arrhythmias.

**Three-dimensional activation patterns in typical AFL.** Previous studies (24–27) have shown that typical AF is a stable macroreentrant arrhythmia with a very specific location of reentry circuit. The findings of this study are in agreement with those other studies. Animated maps demonstrated that reentrant excitation in typical AFL presents a broad wave front in most of the reentry circuit. Points located in close proximity to the tricuspid annulus were in the forefront of the activation wave throughout the annulus perimeter. Our data coincide with those of Kalman et al. (9), who have demonstrated that the tricuspid annulus constitutes a continuous barrier for reentrant wave front in typical AF.
Figure 4. Upper panel, Simultaneous surface ECG leads II and III and BC and CS recordings in a patient with counterclockwise AF. Splines A and B were located in the anterolateral region; spline C in the septal region; spline D in the posterior region; spline E in posterolateral region; and splines F through H in the lateral region of the RA. The BC was inserted from the jugular vein. Complete coverage of the reentry circuit is demonstrated. Lower panel, Animated maps. A, Isthmus conduction. B, First 30 ms of activation in the septal and posterior walls. C, After 100 ms, the activation reaches the roof of the RA. D, Craniodorsal activation of the lateral region. E, Locations of the BC splines versus anatomic structures of the RA. The proximal pole of the BC is located over the inferior vena cava–tricuspid valve isthmus.
Activation patterns in atypical AFL. Atypical AFL is still a largely unknown arrhythmia in terms of mechanisms or clinical significance (17). The findings of our study regarding atypical AFL of RA origin can be summarized as follows: 1) atypical AFLs are a heterogeneous group of arrhythmias that have in common a flutter-like ECG appearance; 2) the majority of the reentry circuits involve one of the venae cava owing to transverse breakthroughs at the level of the crista terminalis; and 3) atypical AF has close relations with atrial fibrillation and can be induced by sympathomimetic drugs. The disputed clinical significance of the arrhythmia and the multiple locations of reentry circuits discourage the application of a standardized ablation therapy. However, if the reentry circuit involves the isthmus, then the ablation strategy could be the same as for typical AFL.

A recent, elegant study by Cheng et al. (28) demonstrated that the lower loop reentry appears to be one of the mechanisms for atypical AFL in the RA. Our data, based on the images of the animated maps in the RA atypical flutters, completely confirm this finding. In another recent study, Gomes et al. (29) successfully terminated atypical AFL by applying radiofrequency energy at the lateral high RA. This location could be consistent with the reentry circuit around the superior vena cava found in two patients in our series.

Study limitations. A recent study from our laboratory demonstrated, with contrast injection in the RA, that the BC does not enable complete coverage of the RA endocardium (30). Regions such as the RA appendage and the inferior vena cava–tricuspid annulus isthmus are incompletely covered by the BC. As a result, foci located in or reentry involving these structures are not covered by the BC. In patients with AFL, satisfactory isthmus coverage (two to four electrode pairs) is achieved by inserting the BC through the jugular veins. The degree of resolution provided by BC bipolar recordings is insufficient to reflect microreentry as a mechanism of AT. The transseptal approach to deploy the BC in the left atrium was not used in this study. Accordingly, in our study we could not address questions related to left atrial tachycardia, especially left atrial flutter.

Conclusions. Computer-assisted animation of multiple electrograms recorded with the BC is a valuable mapping technique that enables the construction of three-dimensional activation patterns of various atrial arrhythmias. The technique is appropriate for unstable, short-lived and complex arrhythmias. The color-coded animation im-

Figure 5. Activation patterns in a patient with atypical AF. Reentry involving the lower RA traveled around the inferior vena cava in a clockwise direction. The cycle length was 186 ms. Splines H and A are located in the lateral region; splines B and C in the posterior region; splines D, E and F in septal region; and spline G in anteroseptal region of the RA. Electrode pairs 7–8 were located in the lower RA. Left panel, Reentrant impulse is divided into two wave fronts: the first one shortcuts in the posterolateral wall (B_{4–5}) and reenters the isthmus region, and the second goes up the lateral wall and down the septal wall. Right panel, By the time the second wave front reaches the low septal region, the first wave front passes through the isthmus and reactivates the lateral wall.

Figure 6. Figure-eight reentry in a patient with atypical AF. The zone of slow conduction is located in the posterior RA (splines E and F). Crowded isochrones alongside splines D and F indicate functional lines of block. A, The exit of impulse from the common pathway. B, Impulse propagation in two loops located in the posterolateral (splines C and D) and septal regions (splines G and H) of the RA and merge of both wavefronts at the level of electrode pairs E_{5–6} and F_{5–6}. C, Activation through the common pathway located in the posterior wall. The cycle length of the AF was 166 ms.
ages simplify the analysis of multielectrode recordings and help in establishing the relation between activation patterns and anatomic structures.

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REFERENCES