High-Density Mapping of Activation Through an Incomplete Isthmus Ablation Line

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Background—Activation mechanisms through gaps in ablation lines and resulting electrograms are poorly understood.

Methods and Results—Eight patients (all men; age, 59±9 years) were studied during a recurrence of typical atrial flutter (cycle length, 233±19 ms) after a previous catheter ablation in the cavotricuspid isthmus. High-density 3-dimensional mapping of the isthmus was performed with the Cordis-Biosense EP Navigation system, and local conduction velocity (CV) was estimated. Maps created with 96±19 points revealed 0.8±0.3-cm gaps of recovered conduction in the ablation line. A broad wave front entered the lateral isthmus with a CV of 1.8±0.7 m/s, halted on the lesion line, and penetrated slowly through the gap with a CV of 0.3±0.1 m/s. Activation then curved and returned antidromically to activate the downstream flank of the line with a CV of 1.1±0.7 m/s. This front fused downstream of the line with slow transverse activation (CV, 0.4±0.3 m/s) parallel to it. The ablation line was demarcated by an incomplete line of convergent double potentials with isoelectric intervals (from 123±34 to 62±16 ms); each potential corresponded to local activation upstream and downstream of the lesions, while the intervening delay was produced by slow conduction through the gap combined with the progressively longer curved pathway of downstream antidromic activation as a function of distance from the gap.

Conclusions—High-density isthmus mapping during recurrent flutter indicates slow conduction through gaps of recovered conduction of varying dimensions in the ablation line followed by a curved front of activation antidromically activating its downstream flank, this detour producing wide double potentials on the line. (Circulation. 1999;99:211-215.)

Key Words: potentials ■ atrial flutter ■ electrophysiology ■ mapping ■ catheter ablation

Radiofrequency (RF) catheter ablation in the inferior vena cava (IVC)–tricuspid annulus–coronary sinus ostium isthmus1–2 combined with confirmation of conduction block during sinus rhythm3–5 has resulted in high success rates for the cure of typical atrial flutter. The recurrence rate, however, ranges from 9% to 12%3–5 because of recovered conducting tissue, which can be identified and selectively ablated by use of local electrogram markers of gaps.6 However, the local mechanisms of activation through an incomplete RF ablation line and the resulting electrograms have not been assessed. In this report, we describe the results of high-density sequential mapping in the isthmus in 8 patients during recurrent typical atrial flutter.

Methods

Eight patients were studied during a recurrence of typical atrial flutter (cycle length, 233±19 ms) after previous ablation in the cavotricuspid isthmus. All were men, with a mean age of 59±9 years. None had structural heart disease.

The previous ablation had been performed 263±361 days previously by sequential point-by-point RF application at sites in the cavotricuspid isthmus, with atrial electrograms centered on the surface ECG flutter wave plateau.7

Electrophysiological Study

The procedure was performed with the patients having given informed consent, off all antiarrhythmic drugs, and after 4 to 6 hours of fasting. Bipolar electrograms were filtered with a band-pass setting of 30 to 500 Hz, amplified at high gains (0.1 mV/cm) and recorded at a paper speed of 100 mm/s.

High-Density Endocardial Isthmus Mapping

A 3-dimensional (3D), sequentially acquired, high-density map of the isthmus was obtained during flutter (to ensure that isthmus conduction could sustain the arrhythmia) with the Cordis-Biosense EP Navigation system before conventional mapping and ablation. The method has been described previously.8 For this study, high-density 3D activation maps limited to the cavotricuspid isthmus and contiguous right atrium (RA) were reconstructed, encompassing the area between the 5 o’clock and the 7 o’clock positions on the tricuspid annulus in the left anterior oblique view. Two 8F bipolar catheters (Cordis-Navistar), each equipped with a distally placed miniature position sensor, were introduced into the RA. Bipolar electrograms were band-pass filtered between 30 and 400 Hz, and tip unipolar electrograms were filtered between 1 and 400 Hz. One catheter was placed in the RA appendage, and the maximum peak of the bipolar RA electrogram was used as a fiducial time reference for signals recorded with the other (mapping) catheter. The local activation time was automatically determined from bipolar electrograms as the maximum negative dV/dt of the signal. The electro-
grams were automatically screened for reproducibility of activation times as well as of the 3D coordinates of the mapping catheter so as to exclude instability of catheter position as well as mechanical perturbations of the underlying rhythm. All electrograms were manually checked, and activation times were corrected if necessary by examination of simultaneously acquired unipolar electrograms. Double potentials (defined by 2 major deflections separated by an isoelectric interval of $\geq 30 \text{ ms}$) were identified and their locations marked on the maps. Because of system software limitations, a single activation time was allotted, based on the rapidity of the unipolar slope, adjacent activation, and the relative amplitude of the bipolar spikes (Figure 1). The interpolation threshold of the color fill-in of the reconstruction was progressively decremented during acquisition to permit a relatively uniform density of mapping points.

A region of local block was defined by a conduction delay between contiguously located points of $\geq 30 \text{ ms}$ produced by an activation detour around the block. The remnant of conducting tissue, the gap, was defined as the narrowest region of orthodromic conduction in the isthmus bounded on both sides by regions of complete block as determined from the activation map or by block on one side and an anatomic edge (either the IVC or the tricuspid annulus) on the other. The gap was measured by modifying the displayed activation color range to begin at the activation time just downstream of the line so that activation upstream and just through the gap was represented homogeneously in red (early activation), contrasting clearly with all surrounding downstream (later) activation. Ablation lesion width was determined as the estimated distance between the mean of 2 points in the same axial plane on either side, i.e., from the upstream flank of double potentials (with a larger-amplitude first potential) to the downstream flank with a larger second potential or a single potential corresponding to the second potential with a minuscule first potential (Figure 1). Linear distances between any 2 given points were determined from the reconstructed map with software based calipers.

Conduction velocity (CV) was estimated as previously described from the ratio of distances between points located in the mainstream of the activation wave front and differences in activation times on the basis of the assumption that atrial myocardium behaves as a 2D structure. Four points forming a quadrilateral within a wave front were selected. The difference of the means of the activation times of the 2 points forming each side of the quadrilateral parallel to the advancing wave front was divided by the estimated distance between the midpoints of these sides ($14.4 \pm 3 \text{ mm}$). This distance varied because of the method of data acquisition used, but points too close together or too far apart were excluded. For double potentials, the activation time of the potential corresponding to the chosen wave

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**Figure 1.** Left, Color-coded high-density isochronal activation zone map of isthmus created by incorporating 105 points in a patient with recurrent flutter. Viewed from above, lateral (RA) and medial (coronary sinus ostium) boundaries are diagonally at bottom and top, respectively. IVC border is to left and superior; tricuspid valve (TV) annulus to right and inferior. During flutter (cycle length, 255 ms), activation enters from lateral edge as a rapidly traversing broad wave front (red and yellow zones; CV, 0.9 m/s) blocks at zone of double potentials (pink circles), producing first spike (81 and 68 ms at IVC and right ventricular edges), passes through a gap (green zone; CV, 0.1 m/s), and returns antidromically downstream (CV, 1.4 m/s) to produce activation at IVC and tricuspid annular edges at 226 and 166 ms, respectively (see electrograms). Latest activation at IVC edge is delayed by another zone of conduction delay (26 ms), which could represent a wave-front curvature effect, anisotropy, and/or an additional region of local block (see text). Right, Schematic of same map with double potential positions marked by black circles and line of block by heavy dashed line. W1 and W2 represent lesion line width measured at 2 locations; their mean was used for analysis. Demarcated gap dimension is also shown. Center, Four double-potential electrograms from sites depicted in diagram on right: from IVC edge (top), from gap border nearer IVC (second electrogram), from gap border nearer TV annulus (third electrogram), and from TV annulus (bottom). For top double-potential electrogram, activation time of first spike at 81 ms with a larger and sharper deflection was chosen to represent local activation; for second electrogram, larger and sharper second spike was chosen; and for remaining 2 with similar spikes, first and second spikes, respectively, were chosen in context of adjoining activation. Convergent configuration of double potentials is evident and more marked for longer segment near IVC.
front was used; in case of fractionated potentials, the maximum negative dV/dt activation time was used for estimating CV. Estimates were obtained (1) upstream of the ablation lesion, (2) through the gap, (3) just downstream of the gap and parallel to the ablation lesion, and (4) downstream farther beyond the gap. The virtual CV across the ablation line—the ratio of lesion width and the interval between upstream and downstream local activation at the edge of the isthmus (tricuspid annulus or IVC edge, whichever was farthest from the gap)—was also estimated.

Ablation

Unipolar RF application androve mapping were performed with a 4-mm thermocouple-equipped tip electrode (Cordis) and an RF generator (Stockert) in a temperature-controlled mode (target, 60°C to 70°C).

Ablation was performed during sustained typical atrial flutter. The isthmus was carefully “scanned” by progressive withdrawal of the ablation catheter from the ventricular margin to the IVC during continuous recording from the distal bipole. Ablation was directed at sites with (1) a single electrogram or (2) a fractionated or “triple” potential straddling the center of the surface ECG flutter wave plateau and concomitantly the isoelectric interval of adjacent double potentials. RF energy was applied at these sites for 60 to 90 seconds without moving the catheter. Success was defined by termination of atrial flutter and confirmation of bidirectional isthmus block.

Continuous variables are presented as their mean±SD.

Results

Anatomic Data

A mean of 96±19 points were used to generate the isthmus maps. The ablation lesion was 0.76±0.3 cm in width and varied from the 5:30 o’clock to the 6:30 o’clock positions. The gap through this line was situated near the tricuspid annulus in 6 patients and near the IVC edge in 3. It was bordered by the IVC in 2 and the tricuspid annulus in 1 and ranged from 1.5 to 0.5 cm in size, with a mean of 0.8±0.3 cm. In one map, 2 gaps close to each other (0.5 and 0.8 cm in size) were observed. In 6 cases, the anatomy of the isthmus in its 2 axes was scaphoid (concave in 2 dimensions); thus, the actual length of the ablation segment (3.1±0.8 cm) was greater than the linear distance between the right ventricle and caval edges (2.3±0.9 cm). In 2 cases, the anatomy was flat. The isthmus was also narrower medially near the ostium of the coronary sinus in 5 patients but nearly the same size in 2 (mean size, 1.9±0.7 cm).

Activation

A broad and homogeneous wave front propagating relatively rapidly (CV, 1.8±0.7 m/s) entered the lateral isthmus, and the reached the lesion line as indicated by halting and slowing of activation, along an incomplete, relatively perpendicular border spanning the isthmus to various extents. Activation penetrated slowly (CV, 0.3±0.1 m/s) through a gap (through 2 gaps in 1 case), then curved to return anterogradely to activate the downstream flanks of the line with a CV of 1.1±0.7 m/s (Figure 2). Activation was completed at the tricuspid annulus and the IVC edges of the lesion line 63±35 and 71±20 ms, respectively, after exiting through the line. The virtual CV across the complete portion of the lesion line was 0.06±0.02 m/s.

Conduction slowing through the gap was most marked for the smaller gaps (lowest velocities of 0.08 m/s and 0.1 m/s for gaps of 0.5 cm and 0.6 cm, respectively) (Figure 1). Slow

propagation parallel to the line (CV, 0.4±0.3 m/s) fused with the curving and antidromically returning wave front. In 6 patients, downstream beyond the gap there was evidence of a local conduction delay (40±9 ms) in activation perpendicular to the ablation lesion: toward the caval edge in 5 (Figure 1) and near the tricuspid annulus in 1. Double potentials were noted in 4 patients 18±3 mm away from the ablation line near the IVC.

Correlation With Electrograms

The timing of the activation front reaching the upstream flank of the line corresponded exactly to that of the first spike of double potentials recorded on the line, and the second corresponded similarly to the antidromically returning wave front downstream of the line (Figure 2). This resulted in a characteristic configuration of electrograms on the line: double potentials with interspike intervals converging on the gap, and conversely, the widest interspike potentials being recorded at the anatomic boundary (tricuspid annulus/IVC) farthest from the gap. The longer the line of complete block, the more evident was the convergent configuration of double potentials (Figure 1).

Ablation Results

Atrial flutter was terminated by 1.2±0.5 RF applications (median, 1). However, a total mean of 5.6±4.8 (median, 4) applications were required locally to eliminate all conduction through the gaps in the isthmus and achieve complete block.

Discussion

Detailed mapping of activation mechanisms after RF application has not been described previously in humans. This report of high-density isthmus mapping verifies and corroborates the mechanism of complex potentials in the presence of an incomplete ablation line during unidirectional transisthmus activation.

Recurrence of typical atrial flutter after previous ablation in the cavotricuspid isthmus may be a result of incomplete ablation or reversibly affected myocardium. Subsequent ablation can be optimized by distinguishing remnant conducting tissue from transmurally ablated areas. Selective targeting of such a “gap” identified by local on-site electrograms represents an effective and parsimonious approach. In this study, in all cases but one, activation within the isthmus passed through a single gap of varying size. Homogeneous upstream activation halting at the lesion line was followed by slow conduction through the gap and a detour in the downstream isthmus to produce an activation delay corresponding to double potential interspike intervals. The double potential intervals converged on the gap, with the widest being farthest from the gap (maximum, 123±34 ms) and the shortest (62±16 ms) at its border. Although conduction slowing was less marked through the larger gaps, typical flutter was sustained by conduction velocities in the isthmus as low as 0.08 m/s. The downstream flank of the ablation line was activated by a complex wave front that passed slowly through the gap along the axis of the isthmus and curved sharply to return anterogradely to the ablation line. Activation also
proceeded slowly parallel to the downstream flank, i.e., perpendicular to the isthmus long axis. In 6 patients, this was accentuated by an additional zone of conduction slowing, perhaps due to the drastically curved wave front, combined in lesser or greater measure with anisotropic conduction properties. Alternatively, an anatomic structure such as the eustachian crest forming a line of block in the 4 patients with double potentials could be responsible for this conduction delay.

No topographic feature or location in the isthmus could be consistently linked to the presence of a gap, which favors other factors such as increased myocardial thickness and/or local intramural cooling by large blood vessels as the cause for conduction recovery at these sites.

Under experimental conditions, double potentials have been shown to result from different mechanisms, the most clinically relevant to creating a complete line being slow conduction versus block. In our study, the documentation of antidromic downstream activation confirmed their role as markers of local block. Wide double potentials separated by isoelectric intervals indicate local block under the recording catheter bipole, but they can be just adjacent to a conducting gap (i.e., at its border) (Figure 1); therefore, full mapping of the isthmus is necessary to identify the gap. Although limited by the size of the roving catheter tip electrode and bipole, the technique of high-density mapping used here illustrates the ability of this system to increase mapping resolution under clinical conditions in a specific region of interest and may be applied to other situations, such as the study of complex potentials in ventricular tissue or from the His-Purkinje system.

References


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