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Substrate Mapping to Detect Abnormal Atrial Endocardium With Slow Conduction in Patients With Atypical Right Atrial Flutter

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OBJECTIVES The purpose of this study was to investigate the relationship between the abnormal substrate and peak negative voltage (PNV) in the right atrium (RA) with atypical flutter.

BACKGROUND The impact of a local abnormally low voltage electrogram on the local activation pattern and velocity of atrial flutter (AFL) remains unclear.

METHODS Twelve patients with clinically documented AFL were included to undergo noncontact mapping of the RA. The atrial substrate was characterized by the: 1) activation mapping; 2) high-density voltage mapping; and 3) conduction velocity along the flutter re-entrant circuit. The normalized PNV (i.e., the relative ratio to the maximal PNV) in each virtual electrode recording was used to produce the voltage maps of the entire chamber. The protected isthmus was bordered by low voltage zones.

RESULTS Atypical AFL of the RA was induced by atrial pacing in 12 patients, including 10 upper loop re-entry and 2 RA free wall re-entry flutter. These protected isthmuses were located near the crista terminalis. The mean width of the protected isthmus was 1.7 ± 0.3 cm and mean voltage at the isthmus was −0.91 ± 0.39 mV. The conduction velocities within these paths were significantly slower than outside the path (0.30 ± 0.18 m/s vs. 1.14 ± 0.41 m/s, respectively; $p = 0.004$). The ratiometric PNV of 37.6% of the maximal PNV had the best cut-off value to predict slow conduction, with a high sensitivity (92.3%) and specificity (85.7%).

CONCLUSIONS Characterization of the RA substrate in terms of the unipolar PNV is an effective predictor of the slow conduction path within the critical isthmus of the re-entrant circuit. (J Am Coll Cardiol 2006;48:492–8) © 2006 by the American College of Cardiology Foundation

The understanding of the relationship between voltage mapping and myocardial disease is based on the investigation of the animal and human infarction models. These studies indicated that regions of a contiguous reduction in the electrogram voltage represented the diseased myocardium (1–3). Our laboratory demonstrated that high-density unipolar voltage mapping offered a unique electrophysiologic description of the atrial substrate in patients with atrial flutter (AFL) (4). However, the impact of the local abnormally low electrogram voltage on the local activation pattern and velocity remains unclear. Therefore, the present study investigated the relationship between the conduction delay and abnormal substrate detected by the high-density noncontact unipolar peak negative voltage (PNV) in the right atrium (RA) with atypical AFL.

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METHODS

Patient population. Between July 2002 and March 2004, 12 patients (6 men, age 66 ± 15 years, range 32 to 80 years) with clinically documented atypical AFL were included in this study. None of the patients had a previous history of an RA atriotomy. Six patients had cardiovascular disease, including 6 with hypertension, 2 with hypertrophic cardiomyopathy, and 1 with coronary artery disease.

Electrophysiologic study. All patients were studied in the postabsorptive nonsedated state after giving written informed consent to the electrophysiologic study and catheter ablation, and the details of the noncontact mapping study were explained to all patients. All antiarrhythmic drugs were discontinued for >5 half-lives. In all patients, three multipolar electrode catheters (Daig Corp., Minnetonka, Minnesota) were positioned, respectively, in the high RA, His-bundle area, and right ventricle via the femoral veins. A 7-F deflectable decapolar catheter with 5 pairs of electrodes separated by 5 mm and an interelectrode spacing of 2 mm (Daig Corp.) was also inserted into the coronary sinus (CS) via the internal jugular vein. The position of the proximal electrode pair at the ostium of the CS was confirmed with a contrast injection. In the patients with AFL, a 7-F 20-pole deflectable Halo catheter with 10 mm paired

Abbreviations and Acronyms

AFL = atrial flutter
CS = coronary sinus
CTI = cavotricuspid isthmus
LAL = low anterolateral
LVZ = low voltage zone
MEA = multielectrode array
NCM = noncontact mapping
PNV = peak negative voltage
RA = right atrium
3D = 3-dimensional

spacing (Cordis-Webster, Baldwin Park, California) was positioned around the tricuspid annulus to simultaneously record the RA activation in the lateral wall and the lower RA isthmus simultaneously. A 9-F sheath placed in the left femoral vein was used to introduce the noncontact mapping catheter. All patients presented into the laboratory in sinus rhythm. The techniques used to induce different types of tachycardias in our laboratory have been described previously (4–11). Conventional multicatheter mapping and noncontact mapping of the RA were performed simultaneously during sinus rhythm, atrial pacing (the low anterolateral [LAL] wall and CS ostium with cycle lengths of 500 and 300 ms), and the tachycardia.

Noncontact mapping (NCM) system. The use of NCM system in our laboratory has been previously described in detail (4–11). In brief; the system consisted of a catheter (9-F) with a multielectrode array (MEA) surrounding a 7.5-ml balloon mounted at the distal end. Raw data detected by the MEA was amplified and digitally transferred to a computer workstation.

The MEA catheter was deployed in the RA over a 0.035-inch guidewire, which was advanced 5 cm into the superior vena cava. The system located the 3-dimensional (3D) position of the electrodes on any desired catheter relative to the MEA using a navigation signal. Navigation provided the means to define a model of the chamber anatomy and to track the position of standard contact catheters within the chamber relative to labeled points of interest, such as anatomic structures or critical zones of conduction. Simultaneous virtual unipolar electrograms were mathematically reconstructed and displayed on the anatomic model, producing isopotential or isochronal color maps. Signals for both electrograms were filtered with a bandwidth of 2 to 300 Hz. Virtual electrograms could also be selected and displayed from any site of interest on the anatomic model using the mouse pointer.

Atrial substrate analysis. The atrial substrate was characterized by the: 1) activation mapping; 2) high-density voltage mapping; and 3) conduction velocity along the flutter re-entrant circuit, using the NCM system (EnSite 3000 System, ESI, St. Paul, Minnesota). The details of these measurements have been previously described (4–11).

ACTIVATION MAPPING. Activation mapping was performed during atypical AFL. During a review of the recorded data during the tachycardia, we began the analysis with a default high-pass filter setting of 2 Hz to preserve the components of the slow conduction on the isopotential map. Color settings were adjusted so that the color range matched 1:1 with the millivolt range of the electrogram deflection of interest. We also interactively placed virtual electrodes on the map's color contours to analyze the corresponding unipolar virtual electrograms. Occasionally, conduction through gaps in a line of block was sufficiently slow that we moved the high-pass filter down to 1.0 to 0.5 Hz. If the atrial electrograms overlapped with the T wave, we delivered ventricular extrastimuli to block 1 ventricular beat and eliminate the associated QRS and T waves. This typically revealed more than 1 full cycle of the re-entrant circuit without far-field interference (8,9).

HIGH-DENSITY VOLTAGE MAPPING. In this study, the mean voltage of the global RA was analyzed from the negative portion of the unipolar electrograms, which were obtained simultaneously from 256 virtual mapping sites equally distributed throughout the RA; off-line software was used to analyze the voltage data (4). The normalized PNV (i.e., the relative ratio to the maximal PNV) in each virtual electrogram was used to produce the voltage maps of the entire chamber (Fig. 1). The protected isthmus was bordered by the low-voltage zones (LVZs). At the border, the convergence of voltage lines was observed (Fig. 2). Once the protected isthmus was found, concealed entrainment pacing was performed to further prove the isthmus in the re-entry circuit. The post pacing interval was defined as the time interval from the last stimulus artifact to the beginning of the first rapid electrogram deflection of the first tachycardia cycle after cessation of the pacing (12).

CONDUCTION VELOCITY. The conduction velocity was also analyzed along the re-entrant circuit of the atypical AFL. The NCM system displayed the Cartesian coordinates relative to the MEA center of points on the virtual endocardium. Therefore, we calculated the distance between two points using the mathematical formula:

$$d = \sqrt{(x_2 - x_1)^2 + (y_2 - y_1)^2 + (z_2 - z_1)^2}$$

where: d = distance

x_1, y_1, z_1 = the Cartesian coordinates at 3D surface point 1
x_2, y_2, z_2 = the Cartesian coordinates at 3D surface point 2

The time it took the wavefront to pass across a distance was determined by observing the propagation of the leading edge of the wavefront on the isopotential map and confirmed by the time interval between the fastest down slope (maximum − dV/dt) of the electrograms at the start and end of the distance measured. The conduction velocity was measured within the path of the protected isthmus and outside of the isthmus, respectively.