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Pacing Mediated Heart Rate Acceleration Improves Catheter Stability and Enhances Markers for Lesion Delivery in Human Atria During Atrial Fibrillation Ablation

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ABSTRACT

OBJECTIVES This study sought to investigate the effect of pacing mediated heart rate modulation on catheter-tissue contact and impedance reduction during radiofrequency ablation in human atria during atrial fibrillation (AF) ablation.

BACKGROUND In AF ablation, improved catheter-tissue contact enhances lesion quality and acute pulmonary vein isolation rates. Previous studies demonstrate that catheter-tissue contact varies with ventricular contraction. The authors investigated the impact of modulating heart rate on the consistency of catheter-tissue contact and its effect on lesion quality.

METHODS Twenty patients undergoing paroxysmal AF ablation received ablation lesions at 15 pre-specified locations (12 left atria, 3 right atria). Patients were assigned randomly to undergo rapid atrial pacing for either the first half or the second half of each lesion. Contact force and ablation data with and without pacing were compared for each of the 300 ablation lesions.

RESULTS Compared with lesion delivery without pacing, pacing resulted in reduced contact force variability, as measured by contact force SD, range, maximum, minimum, and time within the pre-specified goal contact force range ($p < 0.05$). There was no difference in the mean contact force or force-time integral. Reduced contact force variability was associated with a 30% greater decrease in tissue impedance during ablation ($p < 0.001$).

CONCLUSIONS Pacing induced heart rate acceleration reduces catheter-tissue contact variability, increases the probability of achieving pre-specified catheter-tissue contact endpoints, and enhances impedance reduction during ablation. Modulating heart rate to improve catheter-tissue contact offers a new approach to optimize lesion quality in AF ablation. (The Physiological Effects of Pacing on Catheter Ablation Procedures to Treat Atrial Fibrillation [PEP AF]; NCT02766712) (J Am Coll Cardiol EP 2018;4:483-90) © 2018 by the American College of Cardiology Foundation.

Radiofrequency catheter ablation generates resistive and conductive heating of cardiac tissue to cause cell death and scar formation, with the goal of halting electrical conduction. Critical to this is consistently achieving sufficiently elevated tissue temperatures (1,2). Initially identified factors affecting tissue temperature and lesion formation included quantity of radiofrequency energy delivered over time (wattage) and temperature conduction properties to surrounding tissue. Recently, catheter-tissue contact has been shown to affect lesion size and consistency by modulating how much of the

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ABBREVIATIONS AND ACRONYMS

AF = atrial fibrillation

AV = atrioventricular

emitted radiofrequency energy from the catheter reaches the tissue (3). With this understanding, catheters that measure and provide real-time data on catheter-tissue contact have been developed with the hopes of improving ablation outcomes.

Subsequently, clinical data have shown that achieving specific catheter-tissue contact goals, as defined by contact force, predicts improved outcomes with ablation of atrial fibrillation (AF) (4). In particular, as the percentage of time spent within a pre-specified contact force range increases, the likelihood of ablation success increases (5). However, there are few data on how to best achieve specific contact force goals. It has also been shown that cardiac motion significantly affects contact force (6,7). We attempted to study how cardiac motion can be modulated to improve catheter-tissue contact. In particular, we sought to determine if cardiac pacing to increase the cardiac ventricular rate would improve catheter-tissue contact by reducing the extremes of contact force during ablation lesions. In addition, we assessed whether this pacing-mediated modification of catheter-tissue contact correlated with improved measures of lesion delivery as reflected by impedance reduction during radiofrequency energy delivery.

METHODS

STUDY DESIGN. This study was approved by the Institutional Review Board of the New York University School of Medicine, in compliance with the Declaration of Helsinki. Twenty patients with paroxysmal AF who presented in sinus rhythm were randomized to 1 of 2 study groups. All patients received general anesthesia with intubation to suppress spontaneous respiration and prevent patient movement. Based on previously published data examining contact force and ventricular contraction, 20 patients were enrolled in this study (8). To standardize the potential effect of respiration on catheter motion, ventilation was performed in all patients with a tidal volume of 500 ml and a respiratory rate of 12 breaths per minute. All antiarrhythmics were held for at least 5 half-lives before the procedure. Group 1 received atrial pacing during the first half of each lesion with sinus rhythm during the second half; in group 2, ablation occurred during sinus rhythm for the first half of the lesion and pacing during the second half. Pacing was performed from a single site at twice diastolic threshold up to 10 milliamps for 2.0 ms, using a 20-pole catheter (Livewire Duo-decapolar, St. Jude Medical, Inc., St. Paul, Minnesota) wrapping around the right atrium and extending to the distal coronary sinus. Before ablation, pacing was performed at 500 ms from a site in which atrial capture was demonstrated to be consistent. Atrial pacing, rather than ventricular pacing, was selected to maintain the hemodynamics of atrioventricular (AV) synchrony and to ensure consistent ventricular timing without the risk of ventricular pacing and superimposed AV conduction. Patients who developed AF during the study were cardioverted to sinus rhythm. If AV block occurred when pacing at 500 ms, the rate was sequentially reduced to 550 ms, and if AV block occurred then 600 ms. If AV block occurred at 600 ms, the pacing catheter was to be placed in the right ventricle and paced at 500 ms.

Using the Carto 3 mapping and ablation system (Biosense Webster, Inc., Irvine, California), all participants received ablation lesions at 15 pre-specified locations (12 left, 3 right atria). Six lesions each were placed around the left and right pulmonary veins at the following locations: superior-posterior, mid posterior, inferior-posterior, superior-anterior, mid anterior, inferior-anterior. As is standard in our electrophysiology laboratory to optimize patient safety and outcome, lesions at the 6 posterior wall locations were limited to 20 s, whereas lesions at the anterior wall locations were limited to 30 s. Three lesions each of 30-s duration were placed at the anterior, mid, and posterior aspects of a typical cavotricuspid isthmus ablation. If the operator noted the ablation catheter to have a dramatic motion indicative of displacement or if catheter motion exceeded 2.5 mm within the magnetic field defined by the mapping system, the lesion was aborted. A repeat lesion was performed after sufficient time was given to allow for tissue cooling, maximally separated from all previous lesions while remaining in the appropriate anatomic location as prescribed by the protocol. All lesions were applied using a THERMOCOOL SMARTTOUCH 3.5-mm ablation catheter (Biosense Webster, Inc.) and a steerable sheath (Agilis NxT, St. Jude Medical). Based on previously published data on typical contact force range goals of other medical centers and the operator contact force goals at our institution, it was pre-specified that the goal contact force for each lesion would be between 10 and 40 g (5). Ablations were applied in a power control mode with immediate application at 30 Watts. Catheter irrigation was applied at 15 ml/h.

Data were extracted from the mapping system and reviewed to assess contact force data. Contact force and impedance was recorded every 50 ms. Data for each lesion were recorded in their entirety, and correlated with both lesion location and the presence or absence of pacing. From each lesion, 4 s of contact