



## The change in the tissue characterization detected by magnetic resonance imaging after radiofrequency ablation of isthmus-dependent atrial flutter

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### ARTICLE INFO

#### Article history:

Received 12 July 2009

Accepted 9 October 2009

Available online 8 November 2009

#### Keywords:

Atrial flutter

Cardiac magnetic resonance

Cavotricuspid isthmus

Radiofrequency ablation

Anatomy

### ABSTRACT

**Background:** Radiofrequency (RF) ablation produces thermal necrosis and electrophysiological conduction block when lesions are transmural. However, the phenomenon of endocardial edema may prevent the ablative energy from reaching the deeper layers of the myocardium.

**Methods:** Sixty-seven patients underwent RF ablation of the cavotricuspid isthmus (CTI) for isthmus-dependent atrial flutter (AFL; 54 males,  $61 \pm 9$  years). Cardiac magnetic resonance (CMR) imaging was performed 1 day before ablation, and the length and morphology of the CTI were determined. In addition, 1 day and 1 month after ablation, the change in the wall thickness, prevalence of a high signal in the delayed enhancement (DE) and T2-weighted images at the CTI were evaluated.

**Results:** Before ablation, DE regions at the CTI were found in 2 patients with a concave type and 1 with a pouch type. No region with a high T2 signal at the CTI was observed in any of the 67 patients. One day after ablation, high T2 signals were detected at the CTI, and the wall thickness was significantly augmented ( $p < 0.0001$ ). CMR also revealed a DE at the CTI in 46 patients (69%). One month after ablation, the thickened wall with a high T2 signal recovered to baseline, and a DE at the CTI was found in 56 patients (84%).

**Conclusions:** CMR is useful for assessing the creation of endocardial edema and scar tissue resulting from CTI ablation. These results may correlate with the anatomical isthmus block after ablation in a large portion of the patients with isthmus-dependent AFL.

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### 1. Introduction

Radiofrequency (RF) ablation of the cavotricuspid isthmus (CTI) is the treatment of choice for patients with isthmus-dependent atrial flutter (AFL) when its high efficacy is considered [1–3]. The previous studies showed that the specific anatomy of the CTI may be one of the causes for the difficulties with non-successful CTI ablation [4–9]. Evidence for such anatomical characteristics has been reported by angiographic studies [6,7], multi-slice computed tomography [10,11], and cardiac magnetic resonance (CMR) imaging study [8,9]. In addition, the endocardial surface complexity, wall thickness along the CTI [4,7,11], and ablation mediated tissue edema might play a crucial role in formation of conduction gaps and non-transmural lesions along the ablation line [5,12–14]. Although a definite agreement on a feasible approach to visualize the endocardial structures and estimate the isthmus thickness has not been well established, several investigators

have used intracardiac echocardiography for this purpose [13,15]. However, only a few studies have examined the acute edematous changes in the atrium after RF ablation using imaging techniques [16,17]. Recently, several studies showed that the capability of direct visualization of atrial scar tissue by delayed enhancement (DE)-CMR that results from RF ablation would allow further insight into the mechanism of this procedure, and a direct comparison of the electrophysiologic results with the anatomical data [18–20]. In this study, CMR was performed on the patients undergoing CTI ablation to assess the variation in the isthmus anatomy, prevalence of ablation mediated tissue edema and scar formation, and its impact on catheter ablation.

### 2. Methods

#### 2.1. Study population

The study population consisted of 67 patients (54 males; mean age,  $61 \pm 9$  years) undergoing CTI ablation for the treatment of typical isthmus-dependent AFL (Table 1). Structural heart disease was present in 7 patients (10%); coronary heart disease in 3, hypertrophic cardiomyopathy in 3, and valvular heart disease in 1. All patients provided their written informed consent and were enrolled between October 2006 and March 2008. Typical AFL was diagnosed when (1) the surface electrocardiogram was predominantly negative in leads II, III and aVF, and positive in lead V1, with a regular atrial rate between 240 and 340 bpm; (2) the intracardiac electrogram displayed the following

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**Table 1**  
Clinical characteristics of the patients.

No. of patients, <i>n</i>	67
Age, years	61 ± 9 (range; 26–79)
Male gender, <i>n</i> (%)	54 (81)
BMI (kg/m <sup>2</sup> )	25 ± 4
Hypertension	28 (42)
Structural heart disease, <i>n</i> (%)	7 (10)
History of heart failure, <i>n</i> (%)	2 (3)
Left atrial size, mm	40 ± 6
Left ventricular ejection fraction, %	67 ± 8

The values are the mean ± SD. BMI = body mass index.

activation sequence: high right atrium then low right atrium, a counterclockwise CTI activation sequence followed by left atrial activation; and (3) an isthmus participation in the arrhythmic circuit as demonstrated by entrainment maneuvers. The patients underwent CMR imaging 1 day before the ablation to determine the length, morphology and characterization of the CTI. CMR imaging was also performed 1 day after and 1 month after the ablation. Patients with contraindications for CMR imaging (e.g., pacemaker or claustrophobia), or who had previously undergone cardiac surgery or catheter ablation of isthmus-dependent AFL were excluded.

## 2.2. Catheter ablation procedure

All patients underwent a standard, previously described technique for the CTI ablation [21]. All antiarrhythmic medications were discontinued for at least 5 half-lives before the procedure with the exception of amiodarone, which was discontinued at least 6 weeks before the procedure. The procedures were performed with the patients fully anticoagulated. Three steerable electrode catheters were used; an 8-pole catheter was positioned within the coronary sinus, and a 20-polar Halo catheter (Biosense Webster, Diamond Bar, California, USA) was positioned around the tricuspid annulus to simultaneously record the right atrial activation in the lateral wall and lower right atrial isthmus, respectively. An 8-mm tip catheter (Blazer II 5770T; Boston Scientific, San Jose, California, USA) was used for the ablation. All ablation catheters were available in different curves as supplied by the manufacturer, or were equipped with an adjustable curve size. Electrograms were recorded digitally (EPMed Systems, Inc., Mount Arlington, New Jersey, USA), and were filtered at a bandpass setting of 30 to 500 Hz for bipolar recordings. The anatomic information, which was analyzed offline, was not used during the ablation. The RF ablation was performed during AFL in the patients who presented in AFL, or during pacing from the proximal coronary sinus at a cycle length of 600 ms in the patients who were in sinus rhythm. A continuous application of the RF energy during a pullback of the ablation catheter from the tricuspid annulus toward the inferior vena cava was used to create linear lesions on the CTI. RF energy was delivered at a maximum temperature setting of 60 °C and a maximum power output of 40 W for 60 s and up to 120 s for each lesion (EPT-1000TC generator, EP Technologies, Boston Scientific), guided by the changes in the local electrograms. The procedural endpoint was defined as a

complete bidirectional isthmus block described elsewhere [1], and recording widely separated local double potentials along the ablation line during atrial pacing [22]. After a bidirectional block was achieved, a 30 min observation period was allowed during which the persistence of the isthmus block was verified.

## 2.3. Data acquisition with the magnetic resonance imaging

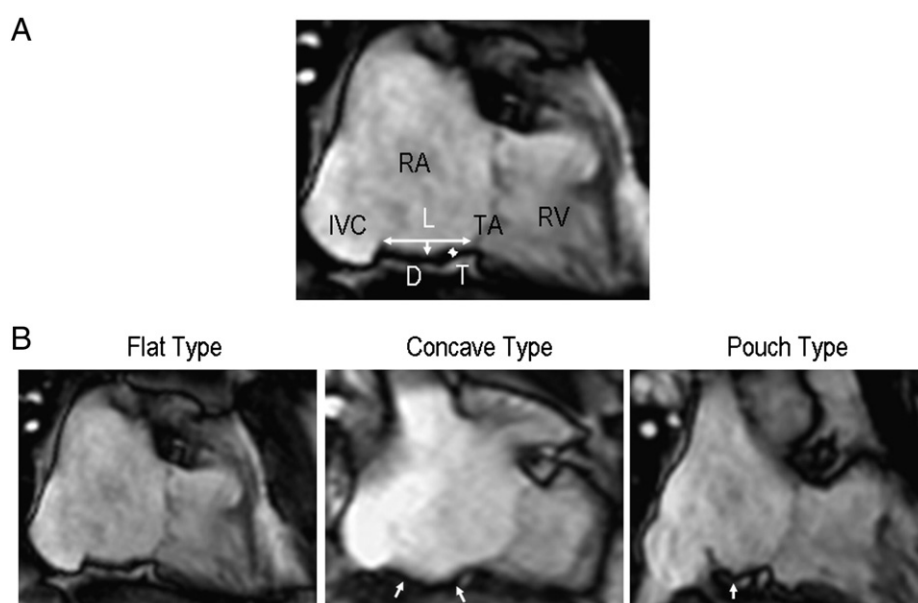
CMR was performed on a 1.5-T magnetic resonance imaging scanner (Achieva, Philips Medical System, Best, The Netherlands) equipped with a Nova gradient and a 5-element cardiac synergy coil was used. The images were acquired using a breath-hold balanced steady-state free-precession cine CMR imaging performed in the long-axis (4-chamber), sagittal (2-chamber), axial and coronal orientations with retrospective electrocardiogram gating to achieve 30 phases/R–R interval [23]. Complete coverage of the right atrium was achieved with 16 to 22 transverse slices acquired during a retrospective electrocardiogram-gated, cine pulse sequence. All of the images were acquired during a breath-hold in expiration (1 to 2 slices per breath-hold depending on the subject's heart rate and tolerance to breath-holding) and were used to evaluate the CTI morphology during the cardiac cycle. Typical scan parameters were: 6-mm slice thickness, no gap between slices, pixel size = 2.0 × 2.0 mm, repetition time/echo time = 2.56/1.03 ms, and 15 views/segment.

The DE-CMR images were acquired 15 min after a bolus injection of gadolinium diethylene triamine pentaacetic acid contrast (Magnevist, Bayer Schering Pharma, Berlin, Germany; 0.15 mmol/kg) using a respiration-navigated, electrocardiogram-gated, gradient echo pulse sequence. Typical acquisition parameters were: repetition time/echo time = 4.0/2.0 ms, and inversion time = 230 to 270 ms. Electrocardiogram gating was used to acquire a small subset of phase-encoding views during the diastolic phase of the right atrial cardiac cycle. The time interval between the R-peak of the electrocardiogram and the start of the data acquisition was defined using the cine images of the right atrium. Fat saturation was used to suppress the fat signal. The echo time of the scan (2.0 ms) was chosen so that fat and water were out of phase, and the signal intensity of the partial volume fat-tissue voxels was reduced, which allowed for an improved delineation of the right atrial wall boundary. The inversion time value for the DE-CMR image scan was obtained for each patient by visual inspection of a preliminary acquisition of a single-slice multi-inversion time series. A typical scan time for the DE-CMR image study was 5 to 10 min depending on subject's respiration and heart rate. If the first acquisition did not have an optimal inversion time or had substantial motion artifacts, the scan was repeated.

Furthermore, we applied a T2-weighted triple inversion recovery sequence (repetition time = 2 × R-to-R interval; echo time = 65 ms; inversion time = 140 ms) to investigate the myocardial condition. Details of this sequence are described elsewhere [24]. In short, a pair of slice-selective and non-selective 180 inversion pulses was applied to null the blood signal, and a third inversion pulse was applied to null the fat signal.

## 2.4. Measurement parameters

The length, wall thickness and morphology of the CTI were evaluated in the sagittal view, and assessed on the latest diastolic frame, confirmed by the opening of the tricuspid valve on the next frame by using cine images (Fig. 1A). The length of the CTI



**Fig. 1.** A. Sagittal plane passing by the center of the inferior (IVC) and superior vena cava and by the right ventricle (RV). Visualization of the right atrium (RA) and tricuspid annulus (TA). Measurements of the length (L), depth (D), and wall thickness (T) of the cavotricuspid isthmus (CTI) on the latest diastolic frame, confirmed by the opening of the tricuspid valve on the next frame by using cine images. B. The morphology of the CTI was divided into three types: flat, concave, and pouch types.

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