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Original article

Comparison of the origin and coupling interval between ectopy with and without atrial fibrillation initiation

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ABSTRACT

Background: Differentiation of atrial fibrillation (AF) trigger ectopy from other ectopy is often difficult. The purpose of this study was to compare the origin and coupling intervals (CI) between AF-trigger and non-AF-trigger ectopy.

Methods: This study consisted of 120 patients with AF who underwent an initial ablation. Isoproterenol was infused up to 20 μg/min to provoke ectopy and AF. We measured the CI of all ectopy provoked by an isoproterenol infusion. The %CI was calculated as the CI of the ectopy/P-P interval of the preceding 2 beats.

Results: A total of 117 patients had at least one ectopy, and AF was induced in 56 (47%) patients. Of the 276 ectopies observed in this study, 211 (76%) originated from pulmonary veins and 77 (28%) were AF-trigger ectopy. AF-trigger ectopy more frequently originated from pulmonary veins (PVs) (74 vs. 3, $p < 0.001$) and had a significantly shorter CI (201 ± 70 ms vs. 365 ± 147 ms, $p < 0.001$) and lower %CI ($29 \pm 11\%$ vs. $55 \pm 14\%$, $p < 0.001$) than that of non-AF-trigger ectopy. A receiver operating characteristics analysis revealed that a %CI of 40% was the best cut-off value for differentiating whether it was an AF-trigger or not. The identified trigger group, including patients with provoked AF-trigger ectopy or ectopy with a low %CI (<40%), had a significantly better AF recurrence-free survival rate than the other group (88% vs. 65%, $p = 0.004$).

Conclusions: AF-trigger ectopy predominantly originated from PVs and had a short CI. These findings may be useful for estimating whether ectopies are an AF-trigger or not.

Introduction

The elimination of atrial fibrillation (AF)-trigger ectopy has been an essential procedure in the catheter ablation of AF since Haïssaguerre et al. revealed that AF is triggered by ectopy that mostly originates from pulmonary veins (PVs) [1–5]. An isoproterenol (ISP) infusion is reported to be an effective method for the identification of AF-trigger ectopy [6,7]. However, in the clinical setting, an ISP infusion often provokes not only AF-trigger, but also non-AF-trigger ectopy; and thus, the differentiation of these ectopies is sometimes difficult.

An ectopy with a short coupling interval (CI) could be more arrhythmogenic due to the induction of unidirectional block and shortening of the action potential duration resulting in reentrant tachyarrhythmias. Therefore, we hypothesized that AF-trigger ectopy has shorter CIs than the others. The purpose of this study was to compare the CIs between AF-trigger and non-AF-trigger ectopy.

Methods

Study population

The study population consisted of 120 consecutive patients who underwent a first-time catheter ablation of AF from September 2014 to April 2015 at our institution. Patients with

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hypertrophic obstructive cardiomyopathy, significant coronary artery disease, severe valvular disease, or a previous history of catheter ablation and cardiac surgery were excluded. Informed consent was obtained from all individual participants included in the study. The protocol was approved by our institutional review board.

Electrophysiological study

All patients underwent transesophageal echocardiography within 24 h before the procedure, and those who had any left atrial thrombi were excluded. Antiarrhythmic drugs were discontinued 3 to 5 half-lives before the catheter ablation except for amiodarone, which was discontinued at least 2 months before the ablation. β-Blockers were continued during the perioperative period.

An electrophysiological study and ablation were performed under intravenous sedation using dexmedetomidine with an initial injection of 6 μg/kg/h for 10 min followed by 0.2–0.7 μg/kg/h during the procedure. A 20-pole diagnostic catheter was positioned in the coronary sinus (CS) and right atrium for pacing and recording. A 20-pole catheter was placed in the right atrium and superior vena cava (SVC). Three long sheaths were introduced into the left atrium through a single transseptal puncture site. PV potentials were simultaneously recorded with two circumferential 20-pole catheters (Lasso; Biosense Webster, Diamond Bar, CA, USA and Optima; St. Jude Medical, St. Paul, MN, USA) and a 3.5-mm externally irrigated-tip ablation catheter (Thermocool; Biosense Webster) (Fig. 1a). After transseptal access, a single bolus of heparin (5000 IU) was administered. The heparin infusion was adjusted to maintain an activated coagulation time (ACT) of 300 s or more by checking the ACT every 30 min [8]. If patients were in AF, they were converted to sinus rhythm by electrical cardioversion. During sinus rhythm, ISP was infused at 5, 10, and 20 μg/min at 2-minute intervals to provoke ectopy and AF. The observation of provoked ectopy was continued until the heart rate recovered to the baseline level after the completion of the ISP infusion protocol. The origin of all the provoked ectopy was identified and its CI was measured. To determine the exact location of the ectopy, we moved the electrode catheters, if necessary and ensured the ectopy was the same as the one recorded before moving the catheter by checking the propagation of the other catheters and the same coupling interval. An ectopy was defined as an AF-trigger ectopy when it preceded AF or atrial firing. Atrial firing was defined as the occurrence of two or more successive atrial complexes with a return cycle of <250 ms and a subsequent cycle length of <300 ms. Ectopy with a similar (±10%) CI and activation sequence was classified as an identical one. The %CI was calculated as the CI of the ectopy/P-P interval of the preceding 2 beats (Fig. 1b). If induced AF continued for >1 min, electric cardioversion was performed to check the reproducibility and the AF induction protocol was continued. Electric cardioversion was performed five times at most. The ISP infusion was discontinued if the systolic blood pressure decreased to <80 mmHg, the patient complained of severe chest tightness, or electrocardiographic changes suggestive of myocardial ischemia were observed.

Catheter ablation procedure

The PV isolation procedure was performed using an ipsilateral double-circular mapping catheter technique under the guidance of a three-dimensional mapping system (CARTO3, Biosense Webster). After the completion of the PV isolation, non-PV-trigger ectopy was attempted to be eliminated by ablating the earliest activation sites other than triggers originating from the superior vena cava, which was eliminated by the isolation of the vein. Then, an induction of atrial tachyarrhythmias was attempted by burst pacing from the CS at a cycle length of 300 ms, with a sequential decrement down to the refractory effective period or to 200 ms. If a regular atrial tachycardia was induced, a block line across the reentrant circuit was created for macro-reentrant tachycardia and the earliest activation site was ablated for focal tachycardia. Finally, 40 mg of adenosine triphosphate was infused to reveal and eliminate any dormant PV conduction. Radiofrequency energy was delivered for 20–40 s at each site using a 3.5-mm externally irrigated-tip ablation catheter. The radiofrequency energy was delivered with a power limit of 35 W, and temperature limit of 43 °C.

Follow up

All patients were seen in our hospital at 1- to 3-month intervals. All antiarrhythmic drugs were stopped after the catheter ablation. AF episodes after discharge were assessed by the patients' complaints, 12-lead electrocardiogram (ECG), and 24-hour Holter ECG recordings. The patients with AF recurrences were recommended to receive a second ablation procedure. Anticoagulation therapy was prescribed for a minimum of 3 months and potentially discontinued in the case of a low thromboembolic risk ($CHADS_2$ score 0 or 1). AF recurrence was defined when patients developed

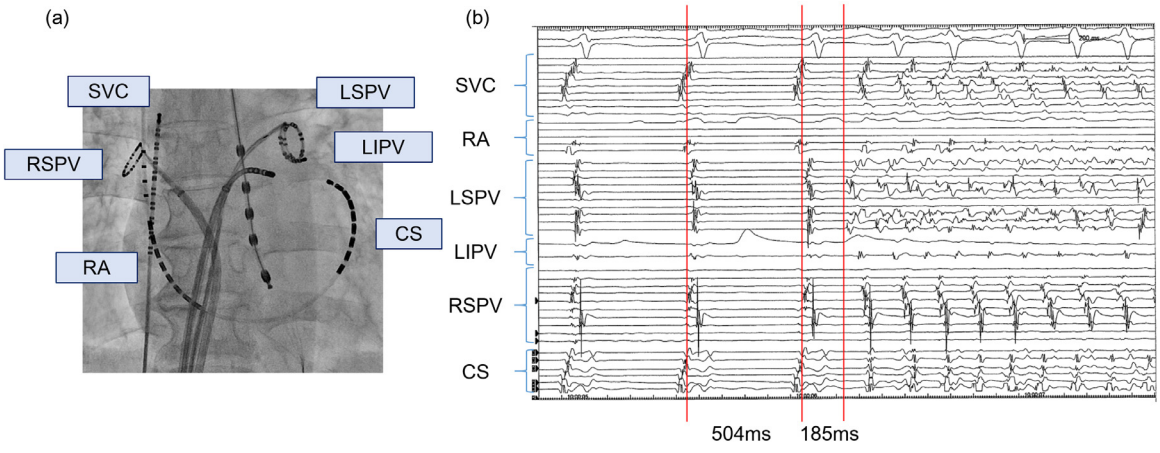

Fig. 1. (a) Positions of the mapping catheters. (b) An example of the %CI calculation: We measured basal atrial cycle length and CI at the earliest site of first ectopy initiating atrial fibrillation. At a basal atrial cycle length of 504 ms, an ectopy was provoked from the LSPV with a CI of 185 ms. %CI = (185/504) × 100 = 37%. CI, coupling interval; LSPV, left superior pulmonary vein; LIPV, left inferior pulmonary vein; RSPV, right superior pulmonary vein; CS, coronary sinus; RA, right atrium; SVC, superior vena cava.

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