Importance of nonpulmonary vein foci in catheter ablation for paroxysmal atrial fibrillation (



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BACKGROUND Pulmonary vein (PV) isolation is an established treatment strategy for paroxysmal atrial fibrillation (PAF). However, the recurrence rate of PAF is 8% to 37%, despite repeated procedures, and the catheter ablation strategy for PAF with non-PV foci is unclear.

OBJECTIVE The purpose of this study was to assess the PAF ablation strategy for non-PV foci.

METHODS The study included 304 consecutive patients undergoing PAF ablation (209 males, age 63.0 ± 10.4 years) divided into 3 groups: group 1 (245 patients) with no inducible non-PV foci; group 2 (34 patients) with atrial fibrillation (AF) originating from non-PV foci and all the foci successfully ablated; and group 3 (25 patients) with AF originating from non-PV triggers, but without all foci being ablated or with persistently inducible AF.

RESULTS Mean follow-up period was 26.9 \pm 11.8 months, and AF recurrence rates since the last procedure were 9.8%, 8.8%, and 68.0% in groups 1, 2, and 3, respectively. There was no statistically significant difference in recurrence rate between

Introduction

Most ectopic beats that initiate paroxysmal atrial fibrillation (PAF) originate from the pulmonary veins (PVs); thus, pulmonary vein isolation (PVI) has become the established treatment strategy.^{1,2} However, the recurrence rate after PVI for PAF still is 8% to 37%, despite multiple procedures.^{3–6} Several studies have addressed the importance of non-PV foci in PAF,^{3,4,7–9} which tend to be located at sites such as the superior vena cava (SVC), left atrial free wall (LAFW), crista terminalis (CT), coronary sinus ostium, ligament of Marshall, left atrial appendage, and interatrial septum.^{4,8,10} Non-PV foci are sometimes difficult to identify and eliminate, with several recent studies reporting that atrial fibrillation (AF) originating from these sources has a worse outcome than AF from PV sources.³ However, several of

groups 1 and 2 (P = .89); however, there were statistically significant differences between groups 3 and 1 (P < .0001) and groups 3 and 2 (P < .0001). The patients in group 2 had an AF-free outcome to equivalent to those who had PV foci in group 1 (P = .83).

CONCLUSION Success rates can be improved for PAF ablation if non-PV foci are detected and eliminated.

KEYWORDS Catheter ablation; Atrial fibrillation; Pulmonary vein isolation; Mapping; Nonpulmonary vein foci

ABBREVIATIONS AAD = antiarrhythmic drug; AF = atrial fibrillation; CA = catheter ablation; CFAE = complex fractionated atrial electrogram; CT = crista terminalis; LA = left atrium; LAFW = left atrial free wall; LAPW = left atrial posterior wall; PAF = paroxysmal atrial fibrillation; PV = pulmonary vein; SVC = superior vena cava

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these reports failed to consider whether the non-PV foci had been completely eliminated during the procedures. Therefore, this study aimed to evaluate the impact of catheter ablation (CA) targeting the elimination of PAF foci, specifically those at non-PV sites.

Methods

Study population

In this study, we investigated 304 consecutive patients with drug-refractory, symptomatic PAF who underwent their first CA procedure between September 2009 and June 2011. Patients who had previously experienced AF lasting more than 24 hours or had severe structural abnormalities (2 patients with severe mitral regurgitation and 1 with a huge atrial septal defect) were excluded because we aimed to assess the importance of AF triggers while minimizing the influence of the AF substrates. All patients included in the study were refractory or intolerant to more than 1 antiar-rhythmic drug (AAD) before the CA procedure. During the

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CA procedure, we analyzed ectopic beats initiating AF. The patients were divided into 3 groups on a per protocol basis: group 1 comprised those without any inducible non-PV triggers in each session, including repeat procedures; group 2 comprised those with AF originating from non-PV triggers, with or without PV triggers, and in whom all non-PV triggers had been successfully ablated not later than the final procedures; and group 3 comprised patients with AF originating from non-PV triggers, with or without PV triggers, but in whom it was impossible to identify the location of AF foci definitively and to ablate completely despite repeat procedures. In group 3, we included those cases in which complete elimination of the non-PV triggers had not been possible and those in which we could not identify or ablate the non-PV foci because the AF was only induced a few times. The study was approved by the ethical committee of Kokura Memorial Hospital.

Electrophysiologic study and mapping procedure

AADs were discontinued at least 5 half-lives before CA, except for amiodarone, which was discontinued at least five days before ablation. The presence of LA thrombi was excluded by transesophageal echocardiography. Each patient provided written informed consent to undergo electrophysiologic study in the fasting state under conscious sedation.

The procedure involved inserting a 20-pole catheter through the right jugular vein. The proximal portion was positioned along the SVC and CT, and the distal portion was placed in the coronary sinus. A 10-pole catheter was positioned at the His-bundle area to record the His-bundle potential and to pace the right ventricle.

Following the standard Brockenbrough technique, we introduced two 10-pole circular mapping catheters and an ablation catheter into the LA. We estimated the location of the AF initiation foci using the endocardial atrial activation sequences from the SVC, CT, His bundle, PV, LA posterior wall (LAPW), and coronary sinus catheters (Figure 1). An electroanatomic mapping system (CARTO, Biosense Webster, Diamond Bar, CA; or EnSite, NavX, St. Jude Medical, St. Paul, MN) was typically used to provide additional guidance and to minimize fluoroscopy time.

Induction of ectopic beats initiating AF

We analyzed the initiating foci using electrode catheters when spontaneous ectopic beats initiated AF. When no spontaneous ectopic beats were observed before the PVI procedure, we intravenously injected adenosine triphosphate (ATP, 20–40 mg) to induce AF. After PVI, we used a bolus injection of ATP during continuous infusion of isoproterenol (ISP, 1–5 μ g/kg/min) to search for non-PV foci. If AF was not initiated by the ATP and ISP infusion, we induced sustained AF by rapid atrial pacing during ISP infusion, which was terminated by intracardiac defibrillation. After restoration of sinus rhythm, we investigated whether there had been any spontaneous reinitiation of AF. Non-PV foci were defined as the earliest ectopic sites where the ectopic



Figure 1 Positions of catheters for induction of nonpulmonary vein foci. CS = coronary sinus; CT = crista terminalis; LAPW = left atrial posterior wall; LSPV = left superior pulmonary vein; RSPV = right superior pulmonary vein; SVC = superior vena cava.

beats initiated AF. Any solitary ectopic beat that did not initiate AF was excluded from the analysis.

Catheter ablation

PVI was performed using 2 circular lines encircling the ipsilateral PVs in all 3 groups. We aimed to ablate non-PV foci consecutively in groups 2 and 3. Linear ablation or complex fractionated atrial electrogram (CFAE) ablation was added, as appropriate. We created LA roof and floor linear lesions to prevent roof-dependent atrial tachycardia when 2 PVI circles were too close (within 1 cm). A 3.5-mm or 4-mm open-irrigated-tip ablation catheter (ThermoCool, Biosense Webster, Diamond Bar, CA; or Cool Path, St. Jude Medical, St. Paul, MN) was used. PVI was considered successful in the acute setting if all ostial PV potentials recorded on the circular mapping catheter during sinus rhythm or coronary sinus pacing had been abolished (ie, entrance block). Exit block was confirmed by pacing from a circular mapping catheter with antral pacing from the ablation catheter. When a non-PV focus was identified, we performed limited area ablation of the earliest ectopic sites, LAPW and SVC. For ectopy from the LAPW, we performed a box-shaped linear ablation around the ectopy by creating roof and floor lines. For ectopy from the SVC, we performed SVC isolation from a site proximal to the SVC ectopic focus. We performed linear ablation at the cavotricuspid isthmus in patients with documented or inducible cavotricuspid isthmus-dependent atrial flutter. The end-point of the linear lesions was complete bidirectional conduction block, confirmed by pacing from the appropriate sites.

Power delivery during radiofrequency ablation was adjusted for the ablation site, and the temperature at the ablation catheter as well as the impedance drop was Download English Version:

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