Electrophysiology

Prevalence and clinical outcome of phrenic nerve injury during superior vena cava isolation and circumferential pulmonary vein antrum isolation using radiofrequency energy

Shinsuke Miyazaki, MD,[a] Eisuke Usui, MD,[a] Shigeki Kusa, MD,[a] Hiroshi Taniguchi, MD,[a] Noboru Ichihara, MD,[a] Takamitsu Takagi, MD,[a] Jin Iwasawa, MD,[a] Akio Kuroi, MD,[a] Hiroaki Nakamura, MD,[a] Hitoshi Hachiya, MD,[a] Kenzo Hirao, MD,[b] and Yoshito Iesaka, MD[a] *Ibaraki, Tokyo, Japan*

Background Phrenic nerve injury (PNI) is recognized as an important complication during atrial fibrillation ablation. This study aimed to investigate the incidence and outcome of PNI during superior vena cava isolation (SVCI) and circumferential pulmonary vein isolation (CPVI) using radiofrequency (RF) energy and the factors associated with its occurrence.

Methods and results Five hundred sixty-seven consecutive patients who underwent SVCI after CPVI without substrate modification who completed a 12-month follow-up were retrospectively analyzed. Point-by-point RF applications were applied with maximum energy settings of 35 W and 30 seconds for the SVCI. In the former 210 patients, sites where pacing captured the PN were avoided whenever possible; however, the maximum power was 35 W. In the latter 357 patients, RF energy was delivered regardless of PN capture; however, the power at PN capture sites was limited to 10 W during continuous diaphragmatic movement monitoring on fluoroscopy. Circumferential pulmonary vein isolation and SVCI were successfully achieved in all. Twelve patients (2.1%) had PNI during SVCI but not during CPVI. Phrenic nerve injury completely recovered in all patients a median of 8.0 months after the procedure. The prevalence was higher in the former period (3.8% vs 1.1%; $P = .03$). A multivariate logistic regression analysis revealed that the study period (odds ratio 3.546; 95% CI 1.051-11.965; $P = .041$) was the sole independent predictor for identifying patients with PNI during SVCI.

Conclusions Phrenic nerve injury occurred in 2.1% of the patients. All occurred during SVCI but not during contemporary CPVI. Energy titration and continuous diaphragmatic movement monitoring significantly decreased the incidence during SVCI. (Am Heart J 2014;168:846-53.)

Since a seminal article reported that paroxysmal atrial fibrillation (AF) is most often triggered by sources inside pulmonary veins (PVs),[1] radiofrequency (RF) catheter ablation of AF is increasingly being performed.[2,3] Electrical PV isolation is established as an effective therapeutic strategy; and with the rapid development of technology nowadays, circumferential PV antrum isolation (CPVI), under guidance using 3-dimensional mapping, has become the standard and cornerstone of AF ablation.[4,5] Other thoracic veins, such as the superior vena cava (SVC), have also been recognized as important sources of AF,[6,7] and electrical isolation is an established therapy for arrhythmogenic SVCs.[8]

Because the right phrenic nerve (PN) runs near the anterior part of the right superior PV (RSPV) before entering the diaphragm,[9] PN injury (PNI) has been recognized as an important complication during AF ablation[10] since seminal reports were published in 2006 by Sacher et al[11] and Bai et al[12] However, both those multicenter retrospective studies consisted of heterogenous designs, methods, and populations. Furthermore, they included procedures undertaken before 2005, and few patients undergoing SVC isolation (SVCI) were included. Anatomically, the course of the right PN is much closer to the SVC than the RSPV, thus PNI is a specific concern during SVCI. However, the prevalence of PNI during SVCI has not been evaluated. This study

From the [a]Cardiovascular Center, Tsuchiura Kyodo Hospital, Ibaraki, Japan, and [b]Heart Rhythm Center, Tokyo Medical and Dental University, Tokyo, Japan.
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Reprint requests: Shinsuke Miyazaki, MD, Cardiology Division, Cardiovascular Center, Tsuchiura Kyodo Hospital, 11-7 Manabeshin-machi, Tsuchiura, Ibaraki 300-0053, Japan.
E-mail: mshinsuke@k3.dion.ne.jp
0002-8703

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aimed to investigate the incidence and clinical outcome of PNI during SVCI and contemporary CPVI using RF energy and evaluate the factors associated with the occurrence of PNI.

Methods

Study population

The study design was retrospective. The inclusion criteria of this study were patients who underwent electrical SVCI after CPVI for AF between October 2010 and June 2013 at our institute. The exclusion criteria were (1) patients who underwent any substrate modification after CPVI for persistent/long-standing persistent AF and (2) those who did not complete 12 months of follow-up after the procedure. Five hundred sixty-seven patients met the above criteria and were included in this study. Superior vena cava isolation was not performed in 111 patients because no SVC potentials were identified despite mapping during the procedure. Atrial fibrillation was classified according to the HRS/EHRA/ECAS 2012 Consensus Statement.[10] All patients gave written informed consent.

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Mapping and ablation protocol

All antiarrhythmic drugs were discontinued for at least 5 half-lives before the procedure. All patients were effectively anticoagulated for >1 month before the procedure. Transesophageal echocardiography was performed to exclude any atrial thrombi. An enhanced cardiac computer tomography was performed to evaluate any relevant cardiac anatomy before the procedure. The surface electrocardiogram (ECG) and bipolar intracardiac electrograms were continuously monitored and stored on a computer-based digital recording system (LabSystem PRO; Bard Electrophysiology, Lowell, MA). The bipolar electrograms were filtered from 30 to 500 Hz. A 7F 14-pole 2-site mapping catheter (Irvine Biomedical Inc, Irvin, CA) was inserted through the right jugular vein and positioned in the coronary sinus for pacing and internal AF cardioversion. The electrophysiologic study was performed under minimal sedation obtained with pentazocine and hydroxyzine pamoate.

Ablation procedure

The ablation was performed according to the strategy described previously.[13] In brief, after a transseptal puncture, 2 long sheaths (SL0; St Jude Medical, Minneapolis, MN) were introduced into both superior PVs. Pulmonary venography during ventricular pacing and contrast esophagography were performed to obtain the relative locations of the PV ostia vis-a-vis esophagus. A 100 IU/kg body weight of heparin was administered after the transseptal puncture, and heparinized saline was additionally infused to maintain the activated clotting time at 250 to 350 seconds. Two circular mapping catheters (Lasso; Biosense Webster, Diamond Bar, CA) were placed in the superior and inferior PVs, and the left- and right-sided ipsilateral PVs were circumferentially and extensively ablated guided by a 3-dimensional mapping system (CARTO3; Biosense Webster). Posteriorly, ablation was performed anatomically in the left atrium (LA), approximately 1 to 3 cm from the PV ostia. Anteriorly, ablation was performed on the edge of the left PVs guided by the earliest PV potentials. The electrophysiologic end point was the achievement of bidirectional conduction block between the LA and PVs.[14] Radiofrequency current was delivered point by point with a 3.5-mm externally irrigated-tip quadripolar ablation catheter (Thermocool; Biosense-Webster) with a power of up to 35 W, target temperature ≤38°C, and irrigation rate of 30 mL/min. The power was limited to 20 W on the posterior wall close to the esophagus. After completing the CPVI, a 30-mg bolus of adenosine triphosphate was injected to unmask any dormant PV conduction, and any gaps responsible for dormant conduction were eliminated by additional RF applications.[15,16]

Superior vena cava isolation and evaluation of PNI

Electrical SVCI was empirically performed after the CPVI if any SVC potentials were identified during pacing from the high right atrium (Figure 1). Guided by SVC angiography, a circular mapping catheter was placed anatomically just above the SVC-right atrium (RA) junction at the level of LA roof, and the position was optimized electrophysiologically to observe separated SVC potentials during high right atrium pacing. Radiofrequency energy was delivered point by point for 30 seconds each using a 4-mm tip nonirrigated catheter in a temperature-controlled mode with the maximum temperature set at 50°C and maximum power at 35 W. When the power did not reach the target power, the maximum temperature was increased up to 57°C, and the catheter contact was slightly changed to reach the maximum power. Before the RF delivery, high output pacing (10 V and 2 milliseconds) was performed at every site. The end point of ablation was to eliminate all SVC potentials on the mapping catheter. Two different ablation strategies were applied to eliminate breakthroughs at areas where the PN was captured by pacing.

Between October 2010 and December 2011 (the former period), whenever possible, we tried to find sites without PN capture close to the sites with PN capture and delivered RF energy at those sites. If conduction gaps still remained in the area with PN capture, RF energy was delivered at sites where pacing induced the weakest diaphragmatic contractions, while monitoring the diaphragmatic movement on fluoroscopy.