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RESEARCH

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Effect on sinus cycle length and atrioventricular node function after high-power short-duration versus conventional radiofrequency catheter ablation in paroxysmal atrial fibrillation

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Abstract

Background: The efficacy and safety of high-power, short-duration (HPSD) radiofrequency catheter ablation for atrial fibrillation (AF) have been demonstrated in several studies. We aimed to evaluate and compare the effects of the conventional method and the HPSD method for AF ablation on the sinus and AV node function in patients with paroxysmal AF.

Methods: The medical records of patients with paroxysmal AF who underwent pulmonary vein isolation (PVI) were retrieved from a prospectively collected AF ablation registry at a large-sized tertiary center. The HPSD group ($n=41$) was distinguished from the conventional ablation group ($n=198$) in terms of the power (50 W vs. 20–40 W) and duration (6–10 s vs. 20–30 s) of radiofrequency energy delivery during PVI. Peri-procedural changes in cardiac autonomy were assessed in terms of the changes in sinus cycle length (SCL), block cycle length (BCL), and effective refractory period (ERP) of the atrioventricular node (AVN).

Results: The SCL, BCL, and ERP of the AVN at baseline and post-ablation were not significantly different between the conventional ablation group and the HPSD group. Shortening of the SCL, BCL, and ERP of the AVN was observed immediately after AF ablation in both groups. One-year recurrence of AF/atrial flutter (35.1% vs. 20.3%; $P=0.011$) and atrial flutter (13.8% vs. 4.7%; $P=0.015$) were higher in the HPSD group than in the conventional ablation group.

Conclusion: Both the HPSD and the conventional ablation method resulted in post-ablation vagal modification as evidenced by the shortening of SCL, BCL, and ERP of the AVN. One-year recurrence of atrial flutter and AF/atrial flutter was higher in patients who underwent the HPSD method.

Keywords: Atrial fibrillation, Autonomic nervous system, Pulmonary vein isolation, Radiofrequency catheter ablation

Introduction

Circumferential pulmonary vein isolation (PVI) has been a cornerstone for ablative treatment in patients with paroxysmal atrial fibrillation (AF) [1]. PVI causes significant alterations of cardiac autonomic function and can also block or eliminate the triggers of AF that originate from pulmonary veins (PVs) [2–5]. The

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resulting modulation of cardiac autonomic tones is caused by the delivery of radiofrequency (RF) energy to the pulmonary vein-left atrium junction where mixed ganglionated plexi (GP) are located [6]. Four major GPs are located around the PV antrum [7], and the additive effects of the modified cardiac autonomic function after PVI have been reported [2, 3].

Recently, several studies have compared the effectiveness and safety of high-power, short-duration (HPSD) radiofrequency catheter ablation (RFCA) with conventional lower-power ablation in AF [8–10]. However, there has not been any study regarding the modification of the intrinsic cardiac autonomic nervous system after PVI using the HPSD method. Therefore, we aimed to evaluate the changes of sinus node and AV node function as an indirect measure of cardiac autonomic function in patients with paroxysmal AF who underwent PVI using the HPSD or the conventional ablation method.

Methods

Patient population

This single-center, retrospective observational study involved consecutive patients with drug-refractory or intolerant, symptomatic paroxysmal atrial fibrillation who received their first RFCA using the CARTO three-dimensional mapping system (Biosense Webster, Irvine, CA, USA). Of the 919 patients who underwent AF ablation between January 2016 and July 2020 at Asan Medical Center (Seoul, Republic of Korea), we excluded patients who met any of the following criteria: persistent AF, age ≥ 75 years, advanced chronic kidney disease, ejection fraction ≤ 35%, left atrium (LA) diameter ≥ 50 mm, valvular heart disease, hypertrophic cardiomyopathy, additional LA linear ablation or superior vena cava isolation, prior AF catheter ablation or Maze operation and 5-s time-controlled RF ablation with 50 watts (Fig. 1). The patients were divided into the conventional RF ablation group and the HPSD RF ablation group according

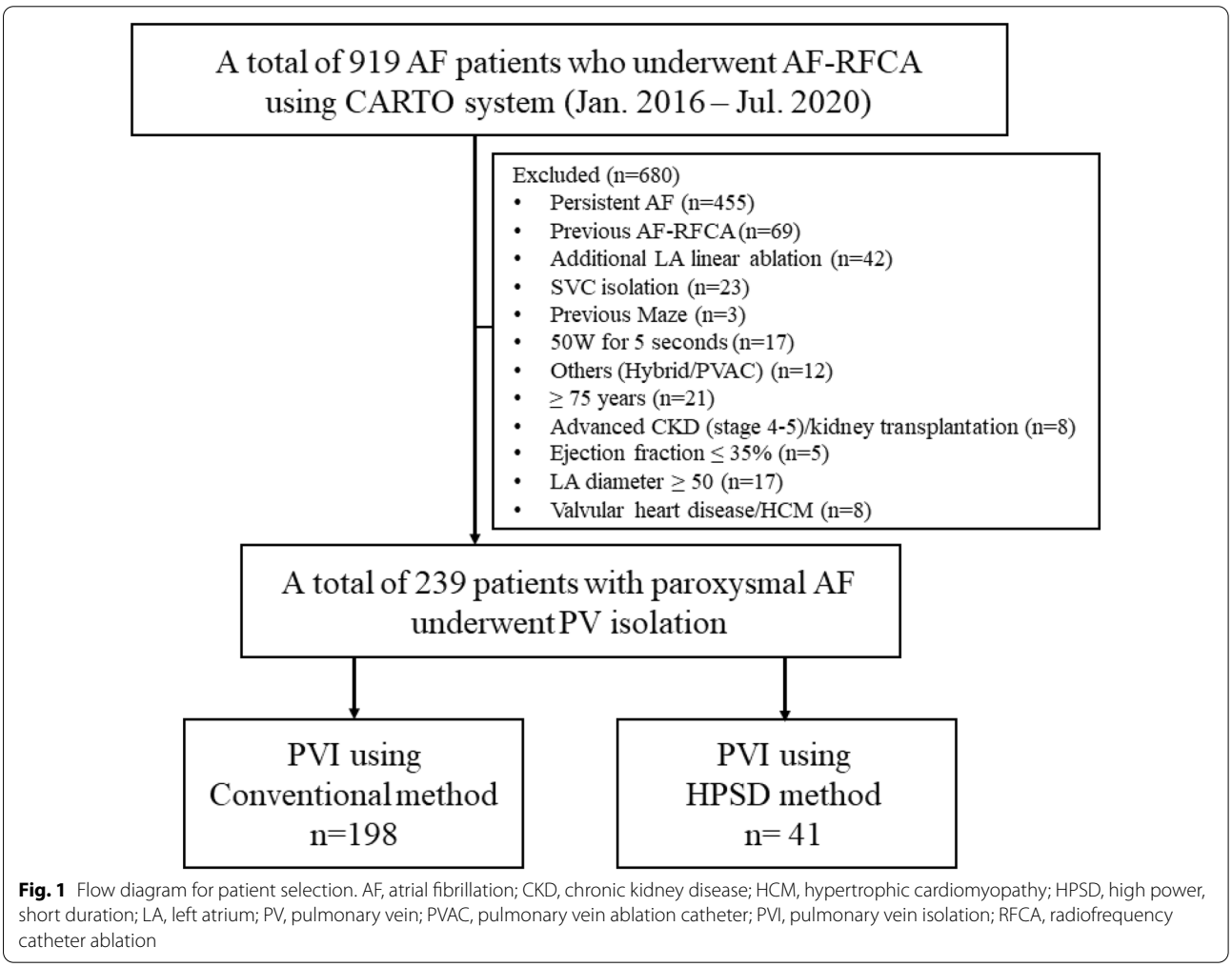

Fig. 1 Flow diagram for patient selection. AF, atrial fibrillation; CKD, chronic kidney disease; HCM, hypertrophic cardiomyopathy; HPSD, high power, short duration; LA, left atrium; PV, pulmonary vein; PVAC, pulmonary vein ablation catheter; PVI, pulmonary vein isolation; RFCA, radiofrequency catheter ablation

to the method applied in RF ablation for circumferential PVI. Written informed consent for data collection was obtained from all patients. This study was approved by the institutional review board of Asan Medical Center (approval number: 2021-0274).

AF radiofrequency catheter ablation

Antiarrhythmic drugs were discontinued for at least five half-lives before the AF ablation. Uninterrupted oral anticoagulation was administered in all patients and supplementary unfractionated heparin was administered to adjust the activated clotting time between 300 and 350 s during the procedure. Remifentanil with/without midazolam was used for sedation/analgesia. A duodecapolar catheter was placed in the coronary sinus and right atrium. Transseptal catheterization was performed under the guidance of hemodynamic monitoring, fluoroscopy, and intracardiac echocardiography (SOUNDSTAR catheter, Biosense Webster). Three-dimensional electroanatomic geometry of the LA was formed with a multipolar mapping catheter (Lasso or PentaRay, Biosense Webster). PVI was performed using a 3.5-mm irrigated tip contact-force sensing RF ablation catheter (THERMOCOOL SMARTTOUCH® SF bidirectional navigation catheter, Biosense Webster). The RF applications were delivered using the power-controlled mode, a temperature limited to 42 °C, and normal saline irrigation (15 mL/min). Circumferential PVI was performed in a sequential point-by-point fashion to avoid gap formation.

In the conventional ablation group, patients underwent PVI using delivery energy of 30–40 watts for the anterior wall and 20–30 watts for the posterior wall of LA using a target contact force of 10–30 g. In the HPSD ablation group, RF energy was delivered with a power output of 50 W for 10 s in the anterior wall and 6 s in the posterior wall for each point [11, 12] The procedural endpoint was the entrance block of all PVs. Isoproterenol infusion (5–10 μg/min) with burst atrial pacing was carried out after ablation to induce non-pulmonary vein triggers or atrial tachyarrhythmia.

Electrophysiologic study

Multiple electrocardiographic leads (lead I, aVF, and V1, filtered between 0.05 and 100 Hz) and intracardiac bipolar electrograms (filtered between 30 and 500 Hz) were simultaneously displayed and recorded on a digital electrophysiologic recording system (CardioLab, Prucka, GE Healthcare, WI, USA). The pacing stimuli were delivered by a programmable digital stimulator (Bloom DTU 215B, Fischer Medical Technologies, CO, USA) at twice the diastolic threshold with a pulse width of 2 ms. Atrial electrical stimulation was performed by pacing maneuvers at one of the electrodes pair of the multielectrode catheter located at the right atrium or coronary sinus. Sinus cycle length (SCL), AV nodal (AVN) block cycle length (BCL), and AVN effective refractory period (ERP) were measured at baseline and immediately after AF ablation. The BCL of the AVN was evaluated by measuring the pacing cycle length at which Wenckebach AV block or 2:1 AV block occurred during incremental atrial pacing with decremental steps of 10–20 ms. The ERP of the AVN was assessed by atrial pacing at an 8-beat drive train with a fixed cycle length followed by premature extrastimuli with 10–20 ms decrements. In patients with baseline AF rhythm, an electrophysiologic study was performed after direct-current cardioversion. When AF rhythm persisted after pulmonary vein isolation, direct-current cardioversion was performed for sinus conversion and the SCL, BCL, and ERP of the AVN were measured.

Post-ablation follow-up and Holter monitoring

Serial follow-up of 12-lead electrocardiogram (ECG) and telemetry monitoring was performed during the index hospitalization after ablation, and the recurrence of atrial arrhythmia was also monitored. The follow-up visits were scheduled at 1, 3, 6, and 12 months after the RFCA and every 6 months thereafter. At each visit, 12-lead ECG was performed. Twenty-four-hour Holter monitoring was carried out at discharge and 3, 6, and 12 months after catheter ablation of AF. Arrhythmic recurrence was defined as the episode of documented AF or atrial tachycardia/atrial flutter (AFL) lasting at least 30 s after the 3-month blanking period [1]. The use of antiarrhythmic drugs after AF ablation was determined at the physician's discretion.

Statistical analysis

Continuous variables were compared by *t* test and are expressed as mean ± standard deviation. Categorical variables were compared with the chi-squared test or Fisher's exact test as appropriate and are reported as percentages. Paired *t* test was used for the comparison of electrophysiological parameters (i.e., SCL, BCL, and ERP of the AVN) between pre-ablation and immediately after ablation. Mean heart rate measured at 24-h Holter monitoring 3, 6, and 12 months after AF ablation were compared with the mean heart rate measured at the pre-ablation Holter monitoring. The cumulative probability and survival curves were constructed from Kaplan–Meier estimates and compared using the log-rank test. P values < 0.05 were considered statistically

significant. All statistical analyses were performed using SPSS software version 20.0 (IBM Corp., Armonk, NY, USA).

Results

Patient characteristics

Among the 239 patients who underwent their first RFCA, PVI using the conventional ablation method and HPSD ablation method were performed in 198 patients and 41 patients, respectively (Fig. 1). The baseline clinical characteristics of the entire population are shown in Table 1. The mean age of the population was 58.6 ± 9.1 years, 67.4% were male, and the mean LA anterior–posterior diameter was 39.3 ± 4.9 mm. There was no significant difference in the baseline characteristics between the conventional group and the HPSD group.

Changes in electrophysiological parameters after AF ablation

The SCL was not significantly different between the two groups at both baseline and post-ablation ($P = 0.194$

Table 1 Baseline characteristics of the patients

Variables	Total ($N = 239$)	Conventional ($N = 198$)	HPSD ($N = 41$)	P value
Age, year	58.6 ± 9.1	58.3 ± 9.3	60.3 ± 8.2	0.198
Male sex	161 (67.4)	133 (67.2)	28 (68.3)	0.889
Height, cm	166.8 ± 9.0	167.0 ± 9.1	165.6 ± 8.2	0.367
Weight, kg	69.5 ± 10.7	69.5 ± 11.0	69.4 ± 9.2	0.964
Body mass index, kg/m^2	24.9 ± 2.8	24.9 ± 2.8	25.3 ± 2.9	0.337
Hypertension	86 (36.0)	72 (36.4)	14 (34.1)	0.788
Diabetes mellitus	26 (10.9)	20 (10.1)	6 (14.6)	0.410
Stroke	19 (7.9)	15 (7.6)	4 (9.8)	0.750
Heart failure	5 (2.1)	3 (1.5)	2 (4.9)	0.204
CHA_2DS_2-VASc	1.3 ± 1.1	1.3 ± 1.1	1.5 ± 1.2	0.177
Coronary artery disease	8 (3.3)	5 (2.5)	3 (7.3)	0.141
Chronic renal insufficiency	13 (5.4)	9 (4.5)	4 (9.8)	0.246
Anticoagulation with NOAC	226 (95.0)	186 (94.4)	40 (97.6)	0.697
B-type natriuretic peptide, pg/mL	73.5 ± 71.8	71.5 ± 72.1	83.3 ± 70.1	0.349
LA diameter, mm	39.3 ± 4.9	39.2 ± 4.9	39.5 ± 4.9	0.676
LV EF, %	61.3 ± 5.3	61.3 ± 4.8	61.0 ± 7.3	0.338
Cavotricuspid isthmus ablation	104 (43.5)	87 (43.9)	17 (41.5)	0.771

Data are presented as mean ± standard deviation or number (%)

HPSD, high power, short duration; LA, left atrium; LV EF, left ventricular ejection fraction; NOAC, non-vitamin K antagonist oral anticoagulant

Table 2 Changes in the SCL, BCL, and ERP of the AVN after ablation

	Conventional		HPSD		P value*
	Mean	SD	Mean	SD	
Baseline SCL, msec	978.0	184.4	932.9	148.1	0.194
Post-ablation SCL, msec	757.7	163.6	738.1	108.2	0.514
Difference, msec (baseline–post-ablation)	220.3	169.1	194.8	161.6	0.434
Baseline AVN BCL, msec	402.6	65.1	386.3	78.1	0.308
Post-ablation AVN BCL, msec	335.4	52.2	320.6	47.8	0.200
Difference, msec (baseline–post-ablation)	67.2	57.7	65.6	66.2	0.910
Baseline AVN ERP, msec	330.2	82.4	305.0	77.7	0.206
Post-ablation AVN ERP, msec	261.3	45.1	247.3	35.8	0.179
Difference, msec (baseline–post-ablation)	68.9	71.6	57.7	71.9	0.523

* Group comparison at each time point using paired *t* test

AVN, atrioventricular node; BCL, block cycle length; ERP, effective refractory period; HPSD, high power, short duration; SCL, sinus cycle length

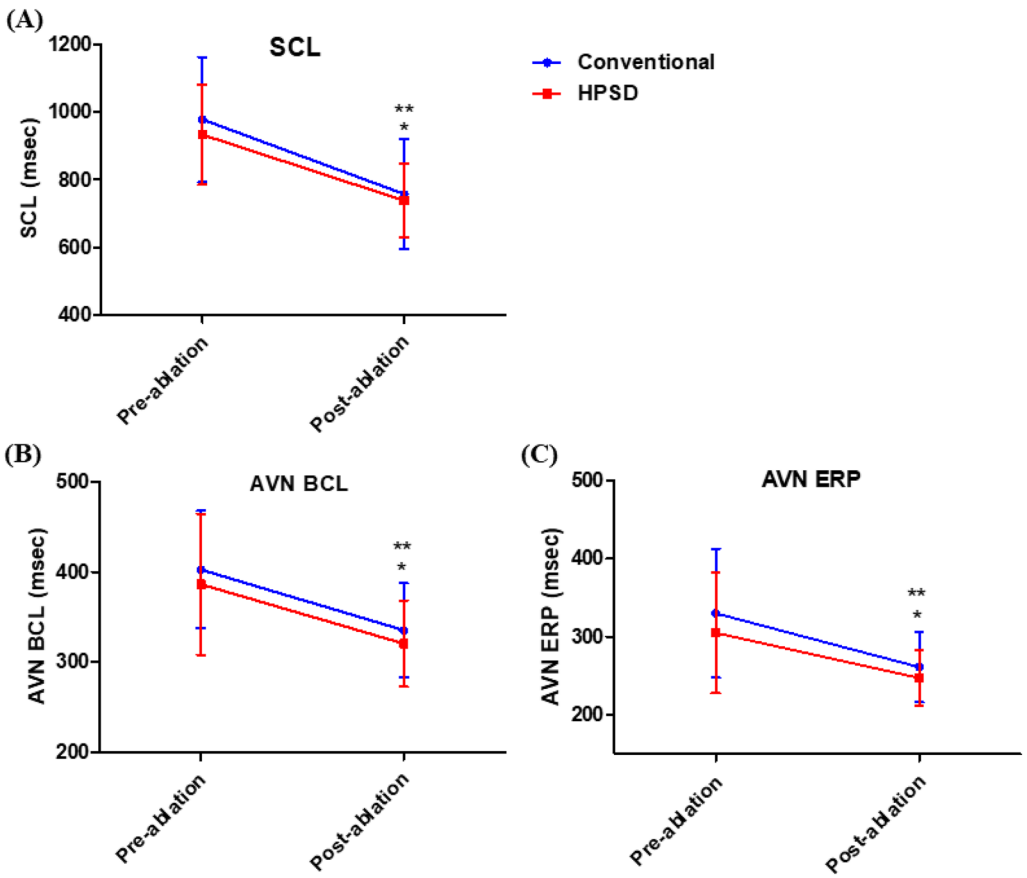

Fig. 2 Changes in the SCL, BCL, and ERP of the AVN after ablation. Sinus cycle length (**A**), block cycle length (**B**), and effective refractory period of the AV node (**C**) before and after AF ablation in patients who underwent AF ablation using the conventional ablation method (blue lines) or the HPSD method (red lines). *$P < 0.001$ between pre-ablation and post-ablation in the conventional group. **$P < 0.001$ between pre-ablation and post-ablation in the HPSD group. AVN, atrioventricular node; BCL, block cycle length; ERP, effective refractory period; HPSD, high power, short duration; SCL, sinus cycle length

and 0.514, respectively; Table 2). Immediate post-ablative SCL was shorter than the baseline value in both groups (both $P < 0.001$; Fig. 2A) and the differences in the SCL between the two time points were 220 ± 169 ms and 195 ± 162 ms in the conventional ablation group and the HPSD ablation group, respectively (Table 2). The post-ablation decrements of SCL were not significantly different between the two groups ($P = 0.434$) and between patients with recurrence during follow-up and those without ($P = 0.808$; Additional file 1: Table S1).

The BCL of the AVN was not significantly different between the two groups at both baseline and post-ablation ($P = 0.308$ and 0.200, respectively; Table 2). Immediate post-procedural BCL of the AVN was shorter than the baseline value in both groups (both $P < 0.001$; Fig. 2B). The differences in the BCL of the AVN between the two time points were 67 ± 58 ms and 66 ± 66 ms in the conventional group and the HPSD group, respectively (Table 2). The post-ablation decrements of BCL were not significantly different between the two groups ($P = 0.910$). Moreover, there was no significant difference in the decrements of BCL between patients with AF/AFL recurrence during follow-up and those without ($P = 0.795$; Additional file 1: Table S1).

The ERP of the AVN was not significantly different between the two groups at both baseline and post-ablation ($P = 0.206$ and 0.179, respectively; Table 2). Post-ablation ERP of the AVN was shorter than the baseline value in both groups (both $P < 0.001$; Fig. 2C) and the differences of the ERP between the two time points were 69 ± 72 ms and 58 ± 72 ms in the conventional group and the HPSD group, respectively (Table 2). The decrements of ERP of the AVN after PVI were not significantly different between the two groups ($P = 0.523$). Further, there was no significant difference between patients with AF/AFL recurrence during follow-up and those without ($P = 0.986$; Additional file 1: Table S1).

Table 3 Changes in the mean heart rate during 1-year follow-up

Mean HR	Conventional		HPSD		P value*
	Mean	SD	Mean	SD	
Baseline	72.1	12.9	72.4	11.2	0.921
3-month	71.4	9.4	75.7	12.7	0.081
6-month	71.3	8.3	73.6	8.0	0.257
12-month	71.2	8.7	70.0	8.9	0.585

* Group comparison at each time point using paired *t* test

HPSD, high power, short duration; HR, heart rate

Table 4 Procedural outcomes

	Conventional	HPSD	P value
Procedure time, min	167.2 ± 36.4	140.2 ± 35.6	< 0.001
Ablation time, min	48.3 ± 11.1	27.6 ± 8.1	< 0.001
Fluoroscopy time, min	9.8 ± 5.4	7.6 ± 3.1	0.013
Fluoroscopy DAP, mcGym2	184.6 ± 227.1	160.1 ± 106.2	0.501
Acute PV reconnection (n = 239)	50 (25.3)	10 (24.4)	> 0.999
Acute PV reconnection, after Isoproterenol (n = 167)	26 (19.7)	2 (5.7)	0.072
Pericardiocentesis	3 (1.5)	1 (2.4)	0.531
Cardiac tamponade	2 (1.0)	1 (2.4)	0.433
Atrioesophageal fistula	0 (0)	0 (0)	-
Stroke/thromboembolic event	0 (0)	0 (0)	-
Phrenic nerve paralysis	0 (0)	0 (0)	-

Data are presented as mean ± standard deviation or number (%)

DAP, dose-area product; HPSD, high power, short duration; PV, pulmonary vein; RFCA, radiofrequency catheter ablation

Change in the mean heart rate during 1-year follow-up

There were no significant differences in the mean heart rate between the two groups at each time point (Table 3). We compared the mean heart rate measured in 24-h Holter monitoring performed before the index ablation procedure with those at 3-, 6-, and 12-month after ablation; as a result, we found that there were no remarkable changes in the mean heart rate during follow-up after ablation in both groups (Additional file 1: Figure S1). A statistically significant change in the mean heart rate was not found between baseline and 3-, 6- and 12-month after ablation in both groups.

Procedural outcomes

The total procedure time, ablation time, and fluoroscopy time were significantly shorter in the HPSD group than in the conventional group (Table 4). Acute PV reconnection during the waiting period was observed in 25.3% of the conventional group and 24.4% of the HPSD group (P > 0.999). And it was more frequently observed in the conventional group after isoproterenol infusion, however, there was no statistical significance (19.7% versus 5.7%, P = 0.072). One patient in the HPSD group underwent pericardiocentesis due to delayed cardiac tamponade that occurred 87 days after AF ablation. Two patients in the conventional group underwent pericardiocentesis due to tamponade that occurred during the ablation procedure; the remaining one patient in the conventional group underwent drainage due to pericarditis. No patients developed symptoms suggestive of atrioesophageal fistula after 3 months following the index procedure. There were no cases of a thromboembolic event, stroke, or phrenic nerve paralysis occurring during post-procedural follow-up (Table 4).

Post-ablation recurrence of atrial tachyarrhythmia

During a median follow-up period of 754 days (IQR, 380–1057), recurrence of AF or AFL was documented in 67 patients (AF, $n = 54$ [80.6%]; AFL, $n = 13$ [19.4%]). The median time to recurrence was 186 days (IQR, 115–334 days). The incidence of 1-year recurrence of AF/AFL and AFL were significantly higher in the HPSD group than in the conventional group (Log-rank $P = 0.011$ and 0.015, respectively). However, the incidence of 1-year recurrence of AF was not different between the two groups (Log-rank $P = 0.125$; Fig. 3A–C). Cox multivariate regression analyses showed that LA diameter was the only independent predictor of the recurrence of AF and the HPSD ablation was significant predictor of the AFL recurrence (Additional file 1: Tables S2–S4).

Repeated ablation

A total of 15 patients underwent repeated RF ablation due to recurrent AF (80.0%) or AFL rhythms (20.0%) at a median follow-up duration of 483 days (IQR, 266–660) until the repeated procedure. The cumulative rates of repeated ablation were not significantly different between the conventional group and the HPSD group (10.3% vs. 5.6%; $P = 0.384$). Among the 15 patients who underwent repeated ablation, PV reconnection was observed in 11 (73.3%) patients. The ablation method was not significantly associated with PV reconnection at the time of the redo procedure (PV reconnection in the HPSD group: 100% [2/2] vs. PV reconnection in the conventional group: 69.2% [9/13]; $P > 0.999$).

Discussion

The main findings in the current study are as follows: (1) in patients with paroxysmal AF, mean SCL, BCL, and ERP of the AVN were all shortened immediately after circumferential PVI regardless of the level of RF power used for ablation, and (2) the recurrences of AFL and AF/AFL during 1-year follow-up were higher in those who

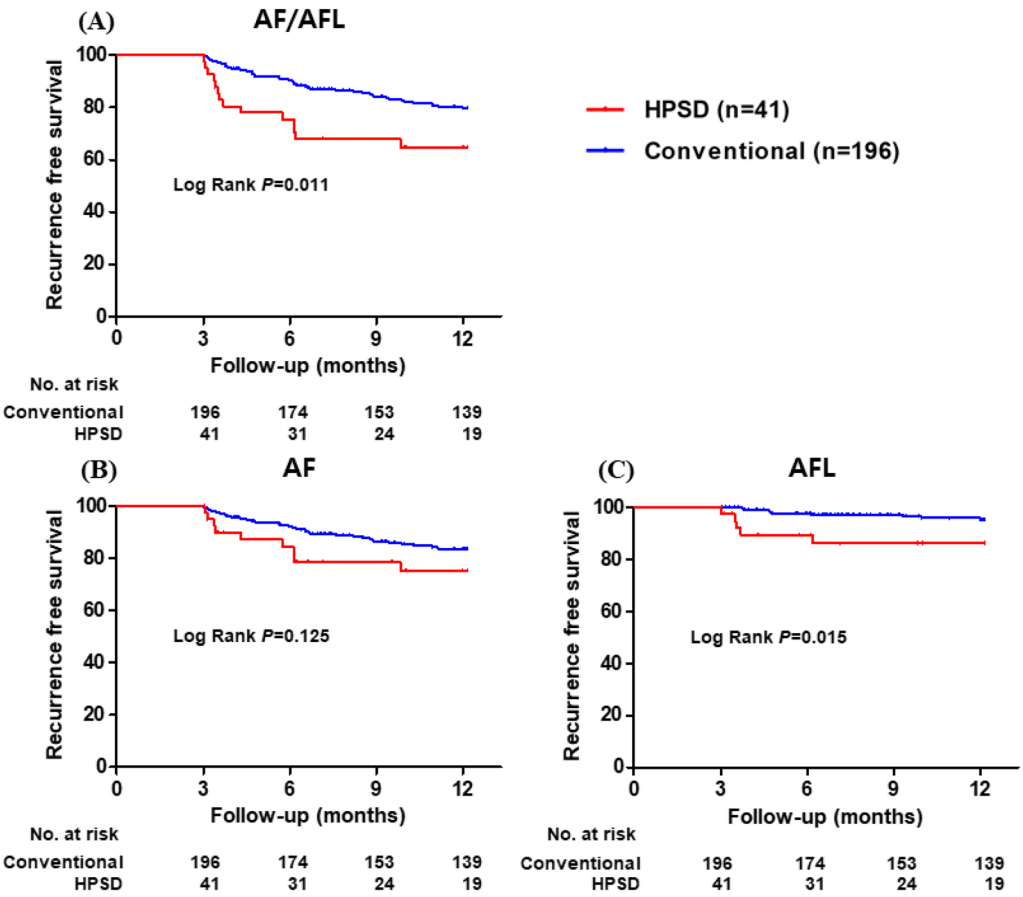

Fig. 3 Kaplan–Meier curves for freedom from arrhythmic recurrence according to the ablation methods. AF, atrial fibrillation; AFL, atrial flutter

underwent HPSD ablation compared with those who underwent conventional ablation.

Several studies reported that PVI in patients with AF increases the resting heart rate and attenuates the heart rate variability [2, 13, 14]. These phenomena were largely explained by the modulative effect on the sympathovagal balance of the intrinsic cardiac autonomic nervous system. It is well-known that intracardiac GPs are located at the roots of PVs and that the GPs are neurally connected to the sinoatrial node [15, 16]. Due to the anatomical proximity of the GPs and PV antrum, changes in the sympathovagal balance after PVI also affect the neurally connected sinoatrial node, which can lead to a faster resting heart rate. Consistent with these findings, the shortening of SCL was commonly observed in both groups in our study. In our HPSD strategy delivering (500 J in the anterior walls) led to similar changes in SCL, BCL, and ERP of the AVN, which could indicate that such amount of energy is sufficient to impact the superior left and right atrial GPs. Because the inferior left and right GPs are not anatomically covered by the current PVI scheme, neither of the ablation strategies can cause any effect on those GPs [17]. Considering that acetylcholinesterase-positive cells are located in the subendocardial atrial myocardium, shallow lesion formation by HPSD might induce a vagal denervation effect [18].

Changes in the electrophysiologic property of AVN during AF ablation have not been reported in humans. An animal study by Zhang et al. [19] showed that ablations of the cardiac anterior right GP and inferior right GP had a negative dromotropic effect on AVN, lasting for only a short period. Another canine study on the acute effect of GP ablation showed no significant changes in the AVN function [20]. This discrepancy in the response of AVN by GP ablations could be explained by the variable destructed contents of parasympathetic and sympathetic elements of the GPs. Adrenergic and cholinergic nerves are highly collocated in GPs, indicating that it is difficult to selectively target either parasympathetic or sympathetic neurons during the ablation procedures [21]. The shortening of the BCL and ERP of the AVN, which was observed immediately after AF ablation in our study, could be explained by the vagal modulation effect.

In previous studies, increased heart rate after PVI persisted for 3 to 6 months or up to 12 months [2, 4, 13]. In

contrast, our results did not show significant changes in the mean heart rate between pre- and post-ablation 24-h Holter monitoring. A possible reason for this discrepancy is that the pre-ablation mean heart rate might have been overestimated because almost all patients showed an episode of paroxysmal AF in the pre-ablation Holter test.

Several studies reported that vagal denervation effect by PVI was associated with a lower recurrence of atrial tachyarrhythmia after ablation [2, 13, 22], while others did not report such beneficial effect of PVI on clinical recurrence [4, 14]. In our study population, there was no significant difference in the post-PVI shortening of SCL, BCL, and ERP of the AVN between patients with AF/AFL recurrence after ablation and those without (Additional file 1: Table S1). Albeit the relatively small number of patients and short follow-up duration of our study, the incidence of AFL recurrence was higher in the HPSD group than in the conventional ablation group (Fig. 3). Another large-scale study showed similar results recently [10], and the higher recurrence of AFL in patients treated with the HPSD method is a major concern. Although the very low risk of esophageal injury is one of the main advantages of HPSD ablation over conventional ablation, shallow lesion formation might contribute to the recurrence of AFL. Bortone et al. [9] showed that the delivery of higher power compared with conventional energy guided by unipolar modification leads to higher single pass PVI, shorter procedural time, and equivalent clinical outcome. The energy delivery per point was higher in Bortone et al. (412.3 ± 39.4 J) than in our study (300–500 J), which is the most likely explanation for the higher recurrence in the HPSD group in our study. Furthermore, the minimal time for chronic lesion formation at 50 W was suggested as 11.0 s [23], which is beyond the value used in our study. Mohanty et al. [24] reported that the lesion duration of < 10 s per point was associated with conduction recovery and a high rate of conduction recovery in the LA posterior wall facing the esophagus in patients who underwent HPSD AF ablation. The optimal duration of energy delivery in HPSD ablation to create a durable lesion in AF is still unclear yet. Furthermore, we consider the risk of the conduction recovery due to the lower total energy delivery in the HPSD method based on our result. When we compared the perimeters of circumferential PVI except for carina sites according to the ablation methods, there were no significant differences in the lesion perimeters (Additional file 1: Table S5). Finally, one animal experiment showed that a power greater than 40 W in fixed ablation index did not lead to higher lesion volume [25], which might have contributed to the higher AF/AFL recurrence in patients in the HPSD group.

We aimed to evaluate the change of the electrophysiologic parameters as an indirect measure of the cardiac autonomic function and compare the arrhythmic recurrence after PVI according to the different RF power. However, there are several limitations to the current study. First, this was a single-center observational study from a prospective cohort registry, and the conventional ablation group included patients who underwent PVI with a range of RF power (20 to 40 watts). Second, among the total of 239 patients, only 127 patients who had undergone Holter tests at all time points during the 1-year follow-up (i.e., baseline, 3, 6, and 12 months after ablation) were included in the analysis of mean heart rate. Third, we did not measure the heart rate variability through Holter monitoring, which could be useful for tracking the long-term changes in sympathovagal neurocardiac function. It may be postulated that elevated sympathetic tone resulting from pain or physical discomfort during the invasive ablation procedure could have contributed to the heightened sympathetic tone; however, it is highly unlikely because we titrated the sedatives and analgesics according to the level of pain and consciousness and all patients well-tolerated the procedure. Fourth, we could not compare and present the rates of the first pass isolation, vagal response during PVI, such as sinus pause, and the steam pop according to the different ablation methods due to the lack of data.

Conclusions

Post-PVI shortening of the SCL, BCL, and ERP of the AVN, which could be caused by vagal modification, was commonly observed in patients who underwent PVI using conventional ablation or HPSD ablation. The HPSD ablation method was associated with shorter procedure time, shorter ablation time, and shorter fluoroscopy time. The 1-year recurrence of AFL and AF/AFL was higher in patients undergoing HPSD ablation.

Abbreviations

AF: Atrial fibrillation; AFL: Atrial flutter; AVN: Atrioventricular node; BCL: Block cycle length; ECG: Electrocardiogram; ERP: Effective refractory period; GP: Ganglionated plexi; HPSD: High power, short duration; IQR: Interquartile range; LA: Left atrium; PV: Pulmonary vein; PVI: Pulmonary vein isolation; RF: Radiofrequency; RFCA: Radiofrequency catheter ablation; SCL: Sinus cycle length.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s42444-022-00063-1.

Additional file 1: Figure S1. Comparison of the mean heart rate during 1-year follow-up. **Table S1.** Comparison of the changes in the EP parameters between recurred and non-recurred patients. **Table S2.** Cox regression analysis of clinical parameters predictive of AF/AFL recurrence. **Table S3.** Cox regression analysis of clinical parameters predictive of AF recurrence. **Table S4.** Cox regression analysis of clinical parameters predictive of AFL recurrence. **Table S5.** Perimeters of the PVI lesion according to the ablation methods.

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Author contributions
UD analyzed and interpreted the data and was a major contributor in writing the manuscript. MK analyzed the patient data. MC, GN, and KC supervised the interpretation of the data and manuscript. JK designed the work, revised the manuscript, and made a final decision on the manuscript. All authors read and approved the final manuscript.

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Availability of data and materials
The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
This study was approved by the institutional review board of Asan Medical Center (Institutional Review Board No. 2021-0274).

Consent for publication
Not applicable.

Competing interests
JK received research grants from Boston Scientific, Metronic, and Abbott. He received honoraria from Biosense Webster, Boston Scientific, and Pfizer.

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