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Different characteristics of postoperative atrial tachyarrhythmias between congenital and non-congenital heart disease

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Abstract

Purpose The chance of encountering tachyarrhythmias has been increasing in adult congenital heart disease (CHD) patients with previous open-heart surgery, along with the improvement of their longevity. However, the characteristics of these arrhythmias remain to be elucidated.

Methods We examined the characteristics of atrial tachyarrhythmias (ATs) in 26 consecutive CHD patients (M/F 17/9) referred for catheter ablation and compared them with 16 non-CHD patients with cardiac surgery (M/F 11/5).

Results The CHD group was younger and had a longer period from cardiac surgery until the occurrence of ATs compared with the non-CHD group (44.8 ± 19.5 vs. 67.6 ± 12.5 years old, and 23.3 ± 13.2 vs. 6.3 ± 4.9 years, respectively, both $P < 0.05$). Multiple ATs were equally induced in both groups, 12 in CHD (46.1%) and 5 in non-CHD (31.3%). Although the prevalence of macro-reentrant ATs (cavo-tricuspid isthmus-dependent atrial flutter (AFL) or intra-atrial reentrant tachycardia (IART)) was comparable, the mechanisms were different between the 2 groups (AFL and IART), 34% and 27% in CHD and 71% and 24% in non-CHD, respectively. Furthermore, focal AT (FAT) was noted in 9 patients (34.6%) in CHD but none in non-CHD ($P < 0.05$). Electroanatomical mapping showed that the surface area and low-voltage area (LVA) of the right atrium were significantly larger in CHD than in non-CHD (197.1 ± 56.4 vs. 132.4 ± 41.2 cm^2, and 40.8 ± 33.3 vs. 13.6 ± 9.0 cm^2, respectively, both $P < 0.05$). Ten out of 14 FATs (71.4%) were highly associated with LVA, especially near the crista terminalis.

Conclusions The development of ATs in CHD patients could be associated with large atrial remodeling, resulting in complicated ATs.

Keywords Atrial tachyarrythmias · Congenital heart disease · Catheter ablation

1 Introduction

The lifespan of patients with congenital heart disease (CHD) after cardiac surgery has been improving along with the advances in operative procedures and devices. Indeed, the number of adult patients with surgically corrected CHD has been increasing [1]. As a result, the incidence of complex arrhythmias in such patients late after the surgery has been rising, posing particular challenges in management [2, 3]. Atrial tachyarrhythmia (AT) occurring late after cardiac surgery for CHD or acquired heart disease is associated with increased morbidity and mortality of these groups of patients [4, 5]. However, the detailed characteristics of these arrhythmias remain to be elucidated. In addition, the management of postoperative AT with antiarrhythmic drugs is often difficult and is sometimes accompanied by side effects [6]. Recently, the evolution of catheter ablation is remarkable, and it has been applied to various tachyarrhythmias including complicated ATs in the CHD patients. The use of a 3-dimensional (3D) electroanatomical mapping system has enabled us to better understand the mechanisms involved in these procedures [7].

In the present study, we thus aimed to evaluate clinical and electrophysiological characteristics of postoperative AT in the CHD patients by comparing those with acquired heart disease in the era of the 3D mapping technology.

2 Materials and methods

2.1 Study population

We enrolled 42 consecutive adult patients referred for catheter ablation for drug-refractory ATs after open-heart

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surgery between January 2009 and May 2014, including 26 with CHD and 16 with acquired heart disease. Patients with atrial fibrillation alone were excluded. Informed written consent was obtained from all patients prior to the study.

2.2 Electrophysiological study

All antiarrhythmic drugs were discontinued for at least 5 half-lives before the electrophysiological study. All patients were studied under non-sedated state. For mapping and pacing, standard multi-electrode catheters were placed in the coronary sinus, around the tricuspid isthmus, His bundle region, and right ventricle. An ablation catheter (non-irrigated 4- or 8-mm tip catheter, and/or irrigated-tip catheter) was advanced into the right atrium (RA) through the right femoral vein. Unfractionated heparin was given as a bolus injection to maintain an activated clotting time between 200 and 300 s throughout the electrophysiological study and catheter ablation therapy.

2.3 Mapping and classification of AT

Mapping was performed using a 3D electroanatomical mapping system; the CARTO™ system (Biosense Webster Inc., Diamond Bar, CA, USA) in most patients (5 patients used CARTO UNIX™, 6 patients used the CARTO XP™, and 22 patients used CARTO 3™ system) and the Ensite NavX™ system (St. Jude Medical Inc., Minnetonka, MN, USA) in the 3 patients, in the remaining 6 patients, we did not use 3D mapping systems. The details of the technology used for electroanatomical mapping were previously described [8, 9].

First, we made a substrate map of a whole right atrium during sinus rhythm or ATs. Then, we induced ATs by a programmed atrial pacing if patients had sinus rhythm and created an activation map to identify the underlying mechanism and select target sites for ablation. A coronary sinus or right atrium electrogram was used as a timing reference. A window of interest was adjusted according to the cycle length of ATs.

Based on an activation map and electrophysiological properties, we divided induced-ATs into 3 different types of AT: (1) cavo-tricuspid isthmus-dependent atrial flutter (AFL); (2) intra-atrial reentrant tachycardia (IART), which was a macro-reentrant tachycardia related to scar tissue, suture lines or prosthetic materials; and (3) focal AT (FAT), which was characterized by a concentric wave of electrical activity emanating from a point origin. A macro-reentrant tachycardia was defined by being identified both a continuous sequence of activation and similar activation time range to the tachycardia cycle length. On the other hand, a focal AT was characterized by less activation time range compared with the tachycardia cycle length.

2.4 Radiofrequency catheter ablation

After mapping, a radiofrequency catheter ablation procedure was performed. Ablation was performed at the anatomical isthmus sites of tachycardia circuits for AFL and IART and performed at the earliest excitation sites for focal ATs. The isthmus of a tachycardia circuit in IART was identified by an entrainment pacing and an activation pattern of local potentials. At each site, radiofrequency current was applied for 60 to 120 s. In the case of non-irrigated ablation, we used a 8-mm (Ablaze™, Lifeline Inc., Tokyo, Japan) or a 4-mm (Navistar™, Biosense Webstar, Diamond Bar, CA, USA) tip catheter. The tip temperature was set at 55 °C and the maximum output at 50 W. We also used a 3.5-mm irrigated-tip catheter (Navistar Thermocool™ Biosense Webstar, Diamond Bar, CA, USA) for 66.7% of the procedures. The tip temperature was set at 40 °C and the maximum output at 40 W, with saline flow of 17 to 30 mL/min. Procedural success was defined as follows: bidirectional block along the cavo-tricuspid isthmus for AFL, and the termination of tachycardia during ablation [10] and achievement of non-inducibility for IART or FAT. We confirmed these success criteria by using the programmed electric stimulation as needed under isoproterenol administration.

2.5 Examination of the RA geometry by CARTO system

To examine the properties of arrhythmogenic substrate, we measured a whole RA surface and a low-voltage area for evaluating atrial volume and the degree of the damage. The low-voltage area was defined as less than 0.5 mV by the CARTO XP™ and/or CARTO 3™ system [7].

2.6 Statistical analysis

Continuous data are expressed as the mean ± standard deviation (SD). Continuous variables were compared by Student's t test. The χ^2 test was used to analyze the qualitative data. Non-parametric tests were used to compare continuous variables if there was a non-normal distribution. A P value of < 0.05 was considered to be statistically significant.

3 Results

3.1 Patient characteristics

Table 1 shows the etiology of patients with CHD and those with an acquired heart disease. Tetralogy of Fallot was the most frequent in the CHD group, followed by atrial sepal defect and ventricular septal defect. In the non-CHD group, valvular heart diseases were noted in 68% (11 out of 16), and there were no patients with aortic valve replacement for bicuspid aortic valve. Table 2 shows the patient characteristics in both groups. The CHD group was younger and had a longer period from the cardiac surgery to the occurrence of ATs compared with the non-CHD group (44.8 ± 19.5 vs. 67.6 ± 12.5 years old and 23.3 ± 13.2 vs. 6.3 ± 4.9 years, respectively, both $P < 0.05$). There was no significant difference in sex, left ventricular function measured by echocardiographic parameters, serum BNP level, or medications, whereas the prevalence of hospitalization for heart failure was higher in the non-CHD group (Table 2).

Atriotomy incisions were confirmed in all patients with CHD except 4 patients [3 with tetralogy of Fallot (TOF) and 1 ventricular septal defect (VSD)] whose data were unavailable. There were 4 patients with complex CHD excluding TOF: 2 with pulmonary atresia (PA), 1 tricuspid atresia (TA), and 1 corrected transposition of the great arteries (cTGA). One patient with PA underwent Fontan operation with atriopulmonary connection because of hypoplastic RV and the other VSD closure and RVOT reconstruction, followed by tricuspid valve replacement 7 years after the first operation. One patient with TA underwent Fontan operation with atriopulmonary connection, and another patient with cTGA Blalock–Taussig shunt and Rastelli operation. In contrast, in the non-CHD group, 10 out of 16 patients were confirmed to have atriotomy incision with clinical or operative records: 3 underwent aortic valve replacement (AVR), 1 coronary artery bypass grafting (CABG), 1 double-valve replacement (DVR), 4 mitral valve replacement (MVR), and 1 left atrial (LA) tumor.

3.2 Mechanisms of atrial tachycardia

Figure 1 shows the prevalence of induced ATs in the CHD and the non-CHD groups. Multiple right-sided ATs were equally induced in both groups; 12 patients in the CHD group (46.1%) and 5 patients in the non-CHD group (31.3%) although there were 2 left-sided ATs (one of each group) whose detailed mechanisms were unknown. In the CHD group, the proportion of the 3 types of ATs was equally noted: AFL (34%), IART (27%), and FAT (31%). In contrast, in the non-CHD group, AFL accounted for a large portion of induced ATs (71%), followed by IART (24%). Furthermore, FAT was recognized in 9 patients (34.6%) in CHD but none in non-CHD ($P < 0.05$). The breakdown of ATs according to etiologies is shown in Table 3. In the CHD group, AFL was widely seen in all etiologies except complex HD, and IART was recognized in all etiologies except VSD. FAT was more frequently seen in TOF (5/9, 56%) compared with other etiologies in the CHD group. On the other hand, in the non-CHD group, AFL was recognized in all patients

Table 1 Classification of the study groups

	Diagnosis	Number of patients
Congenital heart disease (CHD) ($n = 26$)	Tetralogy of Fallot	9
	ASD	8
	VSD	5
	Pulmonary atresia	2
	cTGA	1
	Tricuspid atresia	1
Acquired heart disease (non-CHD) ($n = 16$)	AVR	5
	CABG	4
	MVR/MVP	4
	DVR	2
	Atrial myxoma	1

ASD atrial septal defect, *AVR* aortic valve replacement, *CABG* coronary artery bypass grafting, *cTGA* corrected transposition of the great arteries, *DORV* double-outlet right ventricle, *DVR* double-valve replacement, *MVR/MVP* mitral valve replacement/mitral valvuloplasty, *VSD* indicates ventricular septal defect

Table 2 Patient characteristics

	CHD ($n = 26$)	Non-CHD ($n = 16$)	P value
Gender (male/female)	17/9	11/5	n.s.
Age (year-old)	44.8 ± 19.5	67.6 ± 12.5	< 0.05
Surgery to diagnosis of AT (years)	23.3 ± 13.2	6.3 ± 4.9	< 0.01
Surgery to RFCA (years)	25.5 ± 13.5	7.5 ± 5.6	< 0.01
Hospitalization for HF (n, %)	5 (19.2)	5 (56.3)	< 0.05
Medication (n, %)			
ACEI/ARB	10 (38.5)	11 (68.8)	n.s.
Beta-blocker	14 (53.8)	12 (75.0)	n.s.
Antiarrhythmic drugs	10 (38.5)	2 (12.5)	n.s.
Echocardiographic data			
LAD (mm)	38.5 ± 7.9	43.0 ± 6.6	n.s.
FS	0.31 ± 0.09	0.27 ± 0.12	n.s.
BNP (pg/ml)	162 ± 234	171 ± 216	n.s.

Results are expressed as mean ± SD

ACEI angiotensin-converting enzyme inhibitor, *ARB* angiotensin II receptor blocker, *BNP* brain natriuretic peptide, *CHD* indicates congenital heart disease group, *FS* fractional shortening, *LAD* left atrium dimension, *non-CHD* acquired heart disease group, *n.s.* not significant

except a patient with LA tumor. IART was frequently seen in patients with MVP/MVR (3/4, 75%).

3.3 Substrate analysis of atrial tachyarrhythmias

RA surface area measured by an electroanatomical mapping system (Fig. 2) was significantly larger in the CHD group compared with the non-CHD group (197.1 ± 56.4 vs. 132.4 ± 41.2 cm^2, $P < 0.05$). The low-voltage area was also larger in the CHD group compared with the non-CHD group (40.8 ± 33.3 vs. 13.6 ± 9.0 cm^2, $P < 0.05$). Cycle length of macro-reentrant ATs were comparable between the 2 groups (CHD vs. non-CHD: AFL CL; 283.6 ± 46.1 vs. 269.9 ± 38.8 ms, n.s.; IART CL; 274.7 ± 52.0 vs. 251.4 ± 46.9 ms, n.s.). Table 4 shows the patient characteristics of FAT, which was only noted in the CHD group. Low-voltage area in FAT patients was located adjacent to the crista terminalis, which was the most frequent origin of FATs (64.3% of FATs), followed by the coronary sinus ostium, anterior wall, and tricuspid annulus (Table 4).

3.4 Follow-up after catheter ablation

The recurrence of ATs was noted in 6 patients in the CHD group during the follow-up period (median 505 days): 3 with FATs, 2 macro-reentrant atrial tachycardias defined by no iso-electrical line, and 1 unknown mechanism. The recurrence of macro-reentrant atrial tachycardia was noted

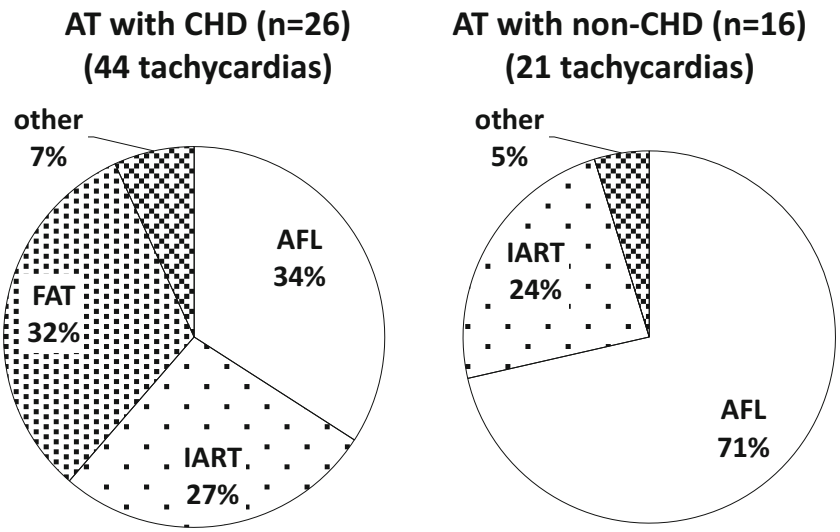

Fig. 1 Mechanisms of atrial tachycardia in the CHD and the non-CHD groups. AFL: cavo-tricuspid isthmus-dependent atrial flutter, FAT: focal atrial tachycardia, IART: intra-atrial reentrant tachycardia, and Others: 2 atrio-ventricular node reentrant tachycardia and 2 AT originated from left atrium

Table 3 Patient numbers of atrial tachyarrhythmias in congenital and non-congenital heart diseases

	Etiologies		Multiple ATs	AFL	IART	FAT	Others ATs
CHD	TOF	($n = 9$)	4	5	3	5	3
	ASD	($n = 8$)	4	5	5	1	
	VSD	($n = 5$)	2	5		2	
	Complex CHD	($n = 4$)	2		3	1	
Non-CHD	AVR	($n = 5$)	1	5	1		1
	CABG	($n = 4$)		4			
	MVR/MVP	($n = 4$)	3	4	3		
	DVR	($n = 2$)	1	2			
	LA tumor	($n = 1$)			1		

AFL cavo-tricuspid isthmus-dependent atrial flutter, *ASD* atrial septal defect, *ATs* atrial tachyarrhythmias, *AVR* aortic valve replacement, *CABG* coronary artery bypass grafting, *CHD* congenital heart disease, *complex CHD* complex congenital heart disease, including 1 patient with corrected transposition of the great arteries, 2 pulmonary atresia, and 1 tricuspid atresia, *DVR* double-valve replacement, *FAT* focal atrial tachycardia, *IART* intra-atrial reentrant tachycardia, *LA tumor* left atrial tumor, *MVR/MVP* mitral valve replacement/plasty, *non-CHD* acquired heart disease, *Other ATs* 2 with atrioventricular nodal reentrant tachycardias in patients with ASD and 2 atrial tachycardias originated from left atrium in each patient with ASD and DVR, *TOF* tetralogy of Fallot, *VSD* ventricular septal defect

in 2 patients in the non-CHD group. In both groups, all patients with recurrent arrhythmias were successfully treated by 2nd session of RFCA or antiarrhythmic agents (mainly sodium channel blockers) except one case in the non-CHD group.

4 Discussion

The major findings of the present study were that (1) AFL and IART were similarly involved in the mechanisms of postoperative development of ATs in the CHD patients, (2) FAT was noted in the CHD group but not in the non-CHD group, and (3) the posterior low-voltage site adjacent to the crista terminalis, but not the incision scar, was a frequent site of FAT.

4.1 Mechanisms of postoperative ATs

ATs are often seen in patients with open-heart surgery, and macro-reentrant ATs are the common mechanism, especially AFL [11]. Pap et al. reported the mechanisms of ATs in 100 patients with open-heart surgery, in which the macro-reentrant ATs were dominant; AFL and IART were seen in 88 and 49 patients, respectively, while FAT was seen only in 11 patients

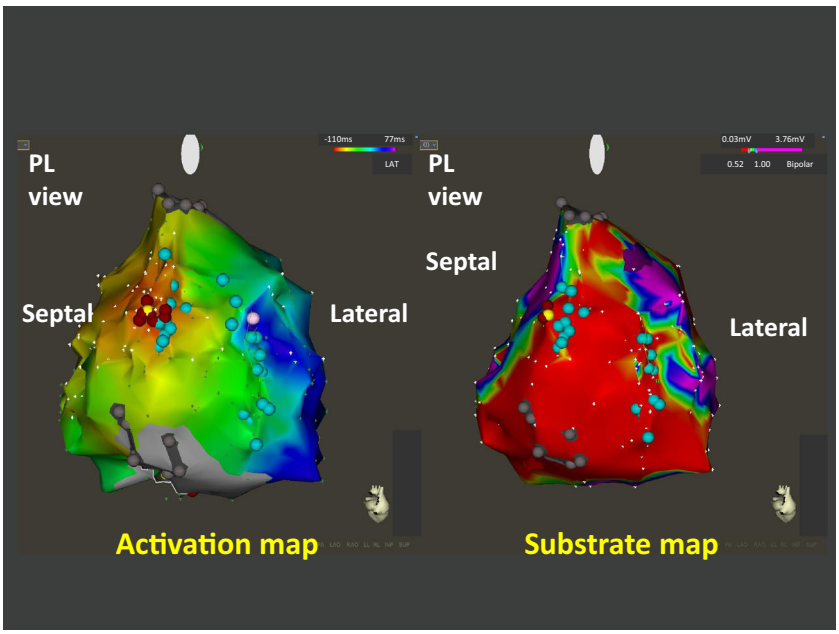

Fig. 2 Representative electroanatomical mapping of focal atrial tachycardia in a patient with postoperative tetralogy of Fallot. The activation map (left panel) and the substrate map (right panel) are shown (postero-lateral view). The double potential lines (blue tag) are recorded in the posterior wall (as crista terminalis) and the lateral wall (as the incision line). The activation map shows that a focal atrial tachycardia arises from the crista terminalis. An extensive low-voltage area (< 0.5 mV) was noted around the crista terminalis in the posterior wall. The atrial tachycardia was terminated by the ablation therapy (yellow tag) and additional application was performed (red tag)

Table 4 Patient characteristics of FAT

Pt no.	Age/ sex	CHD	Origin of FAT	Other ATs	LVA of RA (%)	Position of LVA
1	36/F	TOF	CT	AFL	30.7	PW
2	41/F	TOF	CT	None	5.5	PW
3	54/F	TOF	CT	None	22.9	PW
4	19/M	TOF	CS	AFL	9.0	CS
5	48/M	TOF	Tricuspid annulus	None	NA	Posterior-lateral wall
6	20/F	VSD	CT (2sites), CS	AFL	8.5	Posterior-lateral wall
7	60/M	VSD	Antero-septal wall	AFL	NA	Lateral wall
8	63/M	ASD	Antero-lateral wall	AFL, IART	37.1	Anterior-lateral and PW
9	19/M	PA	CT (3sites), CS	None	46.4	Posterior-lateral wall

AFL cavo-tricuspid isthmus-dependent atrial flutter, *ASD* atrial septal defect, *CS* coronary sinus ostium, *CT* crista terminalis, *cTGA* corrected transposition of the great arteries, *FAT* indicates focal atrial tachycardia, *IART* intra-atrial reentrant tachycardia, *LVA of RA*, percentage of the low-voltage area of right atrium, *PA* pulmonary atresia, *PW* posterior wall, *TOF* tetralogy of Fallot, *VSD* ventricular septal defect

[11]. Mah et al. demonstrated that macro-reentrant tachycardia was noted in 95 out of 127 induced ATs (75%) in 58 patients with TOF and double-outlet right ventricle, while there were only 13 FATs (10%) [12]. In the present study, the frequency of macro-reentrant tachycardia was as high in the both groups (CHD and non-CHD; 61% and 95%, respectively) as in previous studies [11, 12]. On the other hand, the breakdown of macro-reentrant ATs differed between the CHD and the non-CHD groups. AFL was noted in the non-CHD group 3 times higher than IART, whereas AFL and IART were similarly noted in the CHD group. This could be attributed to the difference of mechano-electrical remodeling, which is deeply involved in the development of IART mechanism [13, 14]. In addition, FAT was noted only in the CHD group. The frequency of FAT in the CHD group (32%) was more than that in previous studies (10–16%) [12, 15]. This could come from the difference of patient criteria [12] or etiologies of CHD [15]. In TOF, residual right ventricular volume and pressure overload are often persisted, resulting in the progression of right atrial and/or ventricular remodeling which could be the mechanisms of FAT [13, 14]. The other possibility could be the difference in the induction manner of ATs, as in the present study, unlike the previous studies [10, 11], we attempted to induce ATs repetitively even by using isoproterenol.

4.2 Properties of arrhythmogenic substrate

The progression of atrial mechano-electrical remodeling due to residual or developed hemodynamic abnormalities causes ATs in CHD patients [14, 16]. A surgical incision line could be one of the main substrate of the macro-reentrant AT. However, the substrate is not enough for development of the reentrant AT. IART was frequently noted in those who underwent invasive atriotomy [17]. Nakagawa et al. reported that large low-voltage area in patients after surgical repair of CHD had multiple narrow channels associated with non-CTI-dependent macro-reentrant AT [7]. In the present study, most of the IARTs (10 out of 12 circuits) were involved in ATs associated with atriotomy incisions (incision-AT) in the CHD group. An incision was identified as a linear lesion of double potentials which was apparently distinguished from CT. The circuit was evaluated by a post-pacing interval method. The circuit of AT was considered to be involved in the incision when the post-pacing intervals around the incision matched the AT cycle length. In addition, incision-ATs were more frequently noted in patients with atrial septal defect (ASD) (5/8, 63%) or complex CHD (3/4, 75%) compared with those with VSD (0/5, 0%) or TOF (3/9, 33%). It is possible that atriotomy incisions in patients with ASD and complex CHD were lager compared with those with VSD or TOF. A similar tendency was noted in non-CHD patients. Four out of 5 IARTs in the non-CHD group had incision-ATs and the remaining one lower-loop reentrant AT. In addition, the 4 patients underwent the atriotomy for an atrioseptostomy: 3 with mitral valve replacement/mitral valve plasty, and 1 LA tumor. These atriotomy incisions might be larger compared with other non-CHD patients who had atriotomy mainly for retrograde cardioplegia cannulation through the coronary sinus ostium. Thus, large atriotomy lesion could be an important substrate for incision-ATs in both patients with CHD and those with non-CHD.

The precise etiology and prevalence of FAT in the CHD patients remain to be elucidated. Pap et al. showed that FAT originated from the vicinity of surgical scar, in addition to predilection sites, after open-heart surgery in the CHD patients [11]. In contrast, de Groot et al. reported that the origin of FAT arises not only from the sites near surgically created barriers or from a known predilection sites but also from other damaged atrial sites. They showed that the atrium of the CHD patients contains fibrotic tissue areas, giving rise to local dissociation

in conduction and hence favoring the development of focal activity [18]. In general, the crista terminalis is the site of predilection for FAT [19].

In the present study, the low-voltage area was frequently noted in the right atrial posterior wall when excluding atriotomy incisions, and FAT often originated from the vicinity of crista terminalis adjacent to the low-voltage area (Table 4). FATs were frequently seen in patients with TOF (5/9; 56%), who were susceptible to volume overload of the right heart system. RV volume was enlarged in TOF patients [the average RV end diastolic volume index ($n = 7$), 189 ± 81 ml]. Furthermore, all the 5 patients with FATs (4 with transannular patch and 1 pulmonary valvotomy) finally underwent a pulmonary valve replacement during the follow-up period. In contrast, the remaining 4 without FAT (2 with transannular patch and 2 RV outflow resection) did not require a pulmonary valve replacement during the follow-up period. This suggests that long-lasting atrial hemodynamic load might be involved in the development of a FAT substrate in the RA, especially in the RA posterior near the CT.

4.3 Effectiveness of ablation therapy

Although the ablation therapy for ATs has high acute success rate, the number of AT recurrence is also high [10, 12, 20]. Mah et al. reported that 20 out of 58 patients with postoperative TOF and double-outlet right ventricle needed additional ablation therapy for ATs within 3 years [12]. They pointed out the possibility that recurrence rate might be reduced by treating both the CTI and lateral RA wall simultaneously [12]. De Groot et al. also reported that recurrent AT was noted in 59% of the CHD patients within 1 year after the first ablation therapy and that half of recurrent ATs originated from different locations [10]. Recently, Roca-Luque et al. also reported that 44.7% presented with atrial tachyarrhythmias recurrence after the first ablation for IART in patients with CHD and that the predictors of recurrences were non-CTI-related IART, long PR interval, and previous or induced atrial fibrillation [20]. In the present study, acute success rate was 90.7%, and 19% of AT recurrence was seen within 3 years (6 out of 26 in CHD and 2 out of 16 in non-CHD). The mechanisms for AT recurrence included FAT in 4 and macro-reentrant atrial tachycardia in 4. The AT recurrence rate in the present study was relatively low compared with previous reports [10, 12]. This could be explained by the difference in RFCA procedure methods; in the present study, we attempted to make complete block lines in both the RA free-wall incision line to IVC and CTI and to induce non-clinical FAT repeatedly, even when targeting AT was macro-reentrant AT. In the present study, all the patients in both groups with recurrent arrhythmia maintained sinus rhythm with a 2nd session of ablation therapy or antiarrhythmic agents, except one case in the non-CHD group.

4.4 Study limitations

Several limitations should be mentioned for the present study. First, the present study had a relatively small size of the cohort. In addition, the etiologies of CHD may also be limited when extrapolating the present findings to other types of CHD. Second, the arrhythmias in the present study might have depended on the nature of surgical interventions/techniques that changed over time depending on the etiologies. Third, the detection and classification of ATs could be influenced by the use of the 3D mapping systems and their types. In addition, the advancements in technology during the present study period are likely to influence the acute success and recurrence rate of ATs, especially after the introduction of CARTO SOUND™ system [21] and contact force sensing catheter [22]. Fourth, the mechanisms of ATs were mainly examined by analyzing electro-anatomic activation maps. In the present study, the entrainment techniques were not always performed because of unstable tachycardia conditions and/or non-captured low-voltage area. Finally, since the present study excluded patients with atrial fibrillation alone, only ATs of right atrial origins may have included in the present study. There are some reports of ATs of left atrial origins, especially in non-CHD patients [23]. The present study could underestimate the frequency of ATs in the non-CHD patients.

5 Conclusions

In the present study, we were able to demonstrate that the mechanisms of ATs are different between postoperative patients with CHD and those with acquired heart diseases, which could be explained, at least in part, by the difference in atrial anatomical and/or electrical remodeling.

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Compliance with ethical standards The present study was approved by the Tohoku University Institutional Review Board (No. 2015-1-106).

Conflict of interest The authors declare that they have no conflict of interest.

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