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Acute myocardial infarction treatment : from prehospital care to secondary prevention

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Chapter 11

Long-term outcome after ablative therapy of post-operative atrial tachyarrhythmias in patients with congenital heart disease and characteristics of atrial tachyarrhythmia recurrences.

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ABBREVIATIONS

AT= atrial tachyarrhythmias
CHD= congenital heart disease
AFL= atrial flutter
IART= intra-atrial re-entrant tachycardia
FAT= focal atrial tachycardia
AF= atrial fibrillation

ABSTRACT

Background

Catheter ablation has evolved as a possible curative treatment modality for atrial tachyarrhythmias (AT) in patients with congenital heart defects (CHD. However, data on long-term outcome is scarce. We examined characteristics of recurrent AT after ablation of post-operative AT during long-term follow-up in CHD patients.

Methods and Results

CHD patients (N=53, 27 male, 38±15 yrs) referred for catheter ablation of AT were studied during a follow-up period of 5±3 years.

After ablative therapy of the first AT (N=53, 27 atrial flutters (AFL), CL= 288±81 ms; 22 intra-atrial re-entrant tachycardias, (IART), CL= 309±81 ms; 5 focal atrial tachycardias (FAT), CL= 380±147 ms, success rate: 65%), AT recurred (59% within the first year) in 29 patients, 15 underwent repetitive ablative therapy. Mechanisms underlying recurrent AT was similar in 7 patients (IART: 2, AFL: 5). The location of arrhythmogenic substrates of recurrent AT (IART, FAT) was different for all but one patient. After 5±3 yrs, 5 patients died due to heart failure, 3 were lost to follow-up and the remaining patients had sinus rhythm (31), AT (5) or AF (14). Anti-arrhythmic drugs were used by 18 (57%) sinus rhythm patients.

Conclusion

Successive post-operative AT in CHD patients developing over time may be caused by different mechanisms, including focal and reentrant mechanisms. Recurrent AT originated from different locations suggesting that these new AT were not caused by arrhythmogenicity of previous ablative lesions. Long-term outcome is often complicated by development of AF. Despite frequent need for repeat ablative therapy, most patients are in sinus rhythm.

INTRODUCTION

Atrial tachyarrhythmias (AT) occurring late after cardiac surgery for congenital heart disease (CHD) or acquired heart disease are associated with hemodynamic deterioration, increased risk of thromboembolism and even cardiac death.[1-5] Management of post-operative AT with anti-arrhythmic drugs is often not successful and accompanied by side effects.[1,5-8] In recent years, catheter ablation has evolved as a feasible curative treatment modality for these AT.[9-16] As the arrhythmogenic substrate in patients with prior cardiac surgery is often complex detailed mapping prior to ablation is essential for successful ablative therapy.[17,18]

The first studies of ablative therapy of post-operative AT described ablation procedures using only fluoroscopy. During these procedures, multiple catheters were often required to comprehend the mechanism of the AT. Technological advancement over the years resulted in introduction of 3-dimensional electro-anatomical mapping techniques such as the CARTO™ system.[19,20] By visualizing the electrical activation of the heart chamber mapped in a 3-dimensional reconstruction, these systems are able to facilitate ablative therapy. Ever since their implementation, numerous articles reported on the outcome of ablative therapy of post-operative AT.[10,12,21-26] However, data of long-term outcome is scarce[25,26] and there is a lack of information about characteristics of successive post-operative AT for individual patients.

The aim of this study was to evaluate long-term outcome after ablation of late post-operative AT and to examine characteristics of recurrent AT in a large cohort of patients with predominantly complex congenital heart defects.

METHODS

Study Population

The study population consisted of 53 consecutive patients with congenital heart disease and post-operative, drug refractory AT referred for ablation to our center between 2000 and 2004. Data regarding congenital defects and surgical history were obtained from hospital records. The first visit to the out-patient clinic was 4 weeks after ablation. After this visit, patients were seen every 6 months. Evaluation prior to ablation and during the follow-up period included history, physical examination, ECG, Holter monitoring and echocardiographic examination.

Mapping Procedure

Mapping was performed using a 3-D electro-anatomical mapping system (CARTO™, Biosense-Webster, Diamond Bar, CA, USA). A detailed description of the underlying technology of electro-anatomical mapping has been given previously.[19,20] A 7F Navistar (4mm tip,

2 bipolar electrode pairs, inter-electrode distance 2 mm, Biosense-Webster, USA) was used for mapping and ablation. Bipolar electrograms were filtered at 10-400Hz. A bipolar atrial electrogram recorded by a 6F diagnostic catheter (Biosense-Webster) positioned in the RA served as a temporal reference. A sensor taped on the back served as a location reference.

If AT was not present at the onset of the procedure, it was induced using programmed electrical stimulation. 3-D bipolar activation and voltage maps were constructed during AT to 1) identify the underlying mechanism, and 2) select target sites for ablation. Stability parameters (variability in cycle length, local activation time and beat to beat difference of the catheter's location) were used to exclude signals with low amplitudes due to poor contact of the catheter's tip with the endocardial wall. The local activation time was determined by automatically marking the maximum amplitude of each bipolar potential.

If necessary, markings were adjusted manually. The peak-to-peak amplitude of bipolar electrograms was used to construct colour coded voltage maps. In case of fractionated potentials, the peak-to-peak amplitude of the largest deflection was measured. Areas of scar were delineated using a cut-off value of 0.1 mV.[18]

Classification of Atrial Tachycardia

Based on activation maps, three different types of AT were distinguished:

1) typical atrial flutter (AFL): a single (counter)-clockwise, cavo-tricuspid isthmus dependent macro-reentrant circuit, 2) intra-atrial reentrant atrial tachycardia (IART): a macro-reentrant tachycardia involving scar tissue, suture lines or prosthetic materials,

3) focal atrial tachycardia (FAT): electrical activation originating from a small, circumscribed region from where it expands to the remainder of the atria.

Ablation Procedure

After mapping, a radiofrequency catheter ablation procedure was performed. At each site, radiofrequency current was applied for 60 seconds. In case of non-cooled ablation, tip temperature was set at 70°C and the maximum output at 50W. During ablation using an irrigated-tip catheter (19% of the procedures), temperature was limited to 45-50°C and power to 40-45 W with saline flow of 20 ml/min. Each lesion was tagged on the electroanatomical map. Success was defined as (1) in AFL patients: establishment of a line of conduction block over the cavo-tricuspid isthmus, (2) in IART/FAT patients: termination during ablation.

Statistical Analysis

Data were expressed as mean value ±SD or median (range). Statistical significance was defined as $P<0.05$. One-way ANOVA test was used to compare fluoroscopy time and procedure time required for ablation of different types of tachycardias. Survival free from arrhythmia recurrence was analyzed by method of Kaplan-Meier with corresponding log-rank test for differences in distribution between the curves. The 2 groups were defined as

patients who underwent a successful ablation procedure and patients in whom ablation was not successful.

RESULTS

Characteristics of the study population

The study population consisted of 53 patients (27 male, median age 35 (6-80) yrs). Major common congenital heart defects included transposition of the great arteries (TGA: N=4), univentricular hearts (UVH: N=15), ventricular septal defect (VSD: N=2), coarctation of the aorta (CoA: N=2), atrial septal defect (ASD: N=11), tetralogy of Fallot (ToF: N=10) or valvular heart disease (VHD: N=9). Characteristics of the study population are given in Table 1.

Table 1. Characteristics of the study population.

CHD (number, gender)	Surgical Procedures
TGA (N=4, 3M)	Mustard procedure
UVH (N=14, 7M)	Fontan procedure (atrio-pulmonary conduit, N=11) Mustard operation followed by Jatene procedure (N=1)
Ebstein's anomaly (N=1,M)	Conduit left ventricle –pulmonary artery (N=1) Blalock shunt (N=1) Glenn shunt and ASD closure (N=1)
VSD (N=2, 1M)	surgical closure defect
CoA (N=2, 2F)	resection stenotic part and interposition of a graft
ASD (N=11, 5M)	surgical closure defect
ToF (N=10, 5M)	total correction (N=9) closure VSD and creation Blalock-Taussig shunt (N=1)
VHD (N=9, 5M)	valve replacement (N=8) surgical valvotomy (N=1)

CHD= congenital heart defect, N=number of patients, M=male, F=female UVH= univentricular hearts, TGA= transposition of the great arteries, VHD= valvular heart disease, CoA= coarctation of the aorta, ASD= atrial septal defect, VSD= ventricular septal defect, ToF= tetralogy of Fallot

Time to post-operative atrial tachyarrhythmias and first intervention

Figure 1 shows age at 1) the time of the first surgical procedure, 2) the onset of the AT and 3) the first ablation procedure. Patients are grouped according to major common congenital defect; the groups are ranked according to the earliest averaged age at time of cardiac surgery. Age at time of the first surgical procedure ranged from 0 to 55 (median: 7) years. Median age at onset of AT was 31 (4-73) years; AT developed 18 (6 months to 44) years after the first surgical intervention. The first ablation procedure was performed at the median age of 38 (6-80) years. On average, median time between the onset of AT and the first ablation procedure was 4 years.

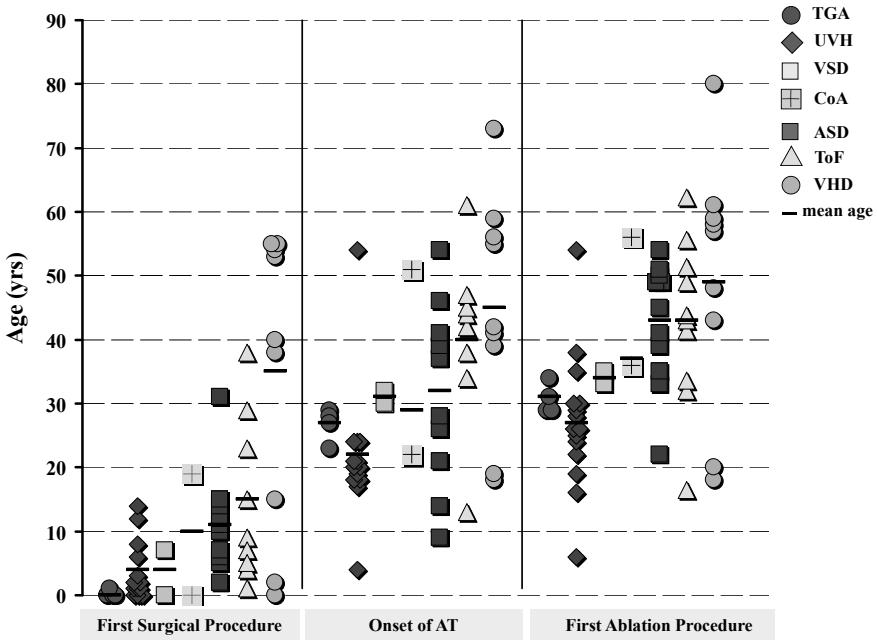

Figure 1. Age at the time of the first surgical procedure, onset of the AT and first ablation procedure for each patient separately. Patients are grouped according to major congenital/acquired heart disease and groups are ranked according to the earliest averaged age at time of cardiac surgery.
TGA = transposition great arteries, UVH = univentricular hearts, VSD = ventricular septal defects, CoA = coarctatioa aortae, ASD = atrial septal defect, ToF = tetralogy of Fallot, VHD=valvular heart disease.

Outcome first ablation procedure

In the entire study population, mapping revealed 27 AFL (cycle length= 288±81 ms), 22 IART (cycle length= 309±81 ms) and 5 FAT (cycle length= 380±147 ms) at the first ablation procedure.

In one patient, 2 AT were eliminated during the same procedure. Successful ablative therapy was achieved in 65% (N=35) of all AT; 20% (N=11) of AT did not terminate and the other AT converted to either another AT (N=4, 7%) or AF (N=4, 7%) during ablation.

In case of AFL, termination during ablation and assessment of a bi-directional conduction block over the cavo-tricuspid isthmus was achieved in 67% (N=18). Despite entrainment demonstrating cavo-tricuspid isthmus dependent conduction, 18% (N=5) of the AFL did not terminate during ablation. Conversion from AFL to AF during ablation occurred in the other 15% (N=4). In those patients, a bi-directional conduction block was assessed after electrical cardioversion to sinus rhythm.

Fifty-five percent of the IART terminated during ablation; conversion from IART to another regular AT or AF occurred in respectively 14% (N=3) and 5% (N=1). Target areas for ablation of IART were located between 1) areas of scar tissue (N=20), 2) scar tissue areas

and the inferior caval vein (N=2). The critical path of the re-entrant circuit was located in the left atrium in only 3 patients. In 27% (N=6), AT did not terminate during ablation despite extensive mapping.

All FAT (N=5) were successfully eliminated by ablation at the site of earliest activation. The majority of the FAT also originated from the right atrium; 1 FAT emerged from the left side of the inter-atrial septum.

Recurrent atrial tachyarrhythmias

Mean follow-up after the first ablation procedure was 5±3 (2.5-9) years. AT recurred in 29 patients and 15 of them underwent therefore more than one ablation procedure. Time between ablative therapy and recurrences of AT are shown in Figure 2. Recurrences after the second ablation procedure occurred in seven patients. In one patient, 9 different AT were ablated during a follow-up period of 6-years (not shown). As demonstrated in Figure 2, most AT often re-appeared within the first year after ablative therapy.

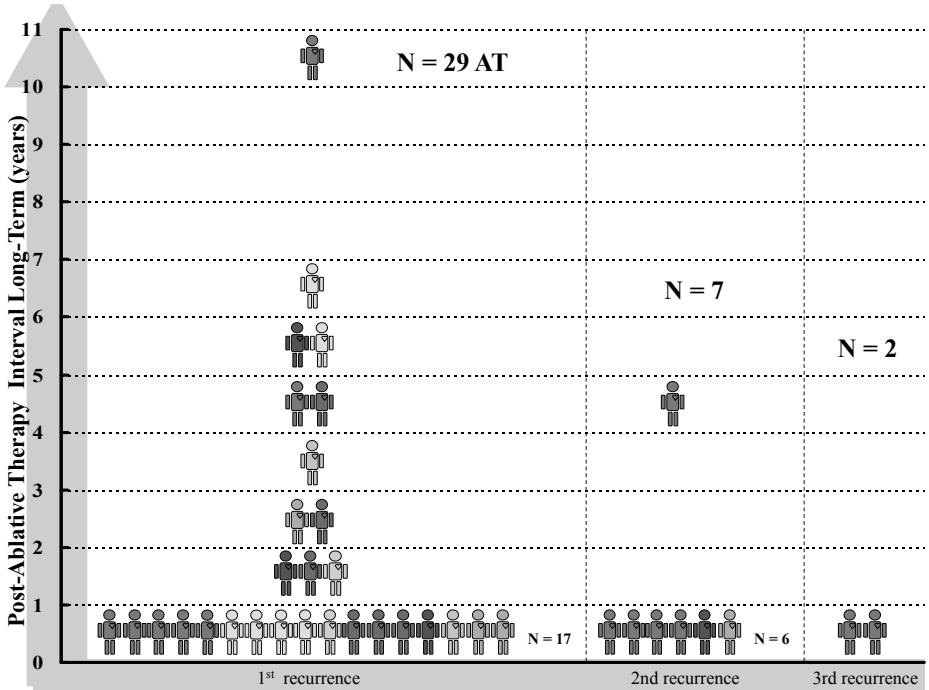

Figure 2. Long-term time interval between ablative therapy and recurrences of AT. Twenty-nine patients experienced one or more recurrences.

Mechanism and location of the arrhythmogenic substrate of recurrent atrial tachyarrhythmias

Figure 3 shows schematic representations of the atria demonstrating the location of the arrhythmogenic substrate of recurrent IART and/or FAT for patients undergoing repetitive ablative therapy (N=15); patients with recurrent AFL (N=5) are not shown. The mechanism underlying the AT is represented by a symbol and the number indicates the order of recurrences. The outcome of the ablation procedure is represented by the colour of the symbol (green: elimination of the AT, red: unsuccessful ablation procedure). In 7 patients with recurrent AT, the underlying mechanism of successive AT was similar, either IART (N=2) or AFL (N=5). Eight patients presented with successive AT caused by different mechanisms, including IART+FAT (N=3), AFL+FAT (N=1), IART+AFL (N=2), AFL+FAT+AF (N=1) or IART+FAT+ AF (N=1). Interestingly, the re-entrant circuit of IART, or the origin of an FAT of consecutive AT was different for the majority of the patients. In one patient, the crucial pathway of the re-entrant circuit of 2 successive IART was located between the inferior caval vein and the atriotomy scar (first patient in the upper panel).

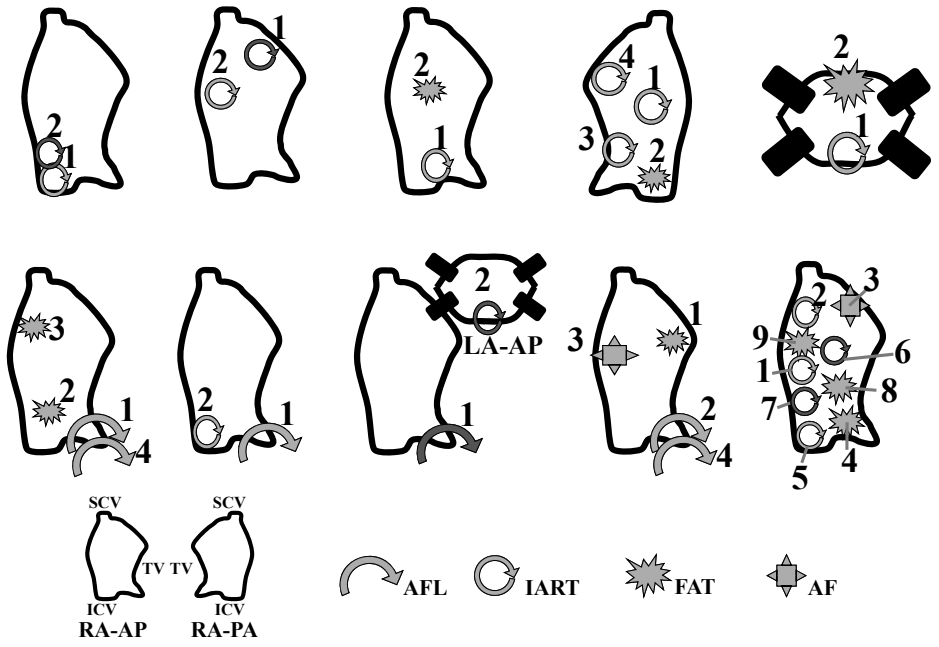

Figure 3. Schematic representations of the atria demonstrating the location of the arrhythmogenic substrate of recurrent AT for patients undergoing repetitive ablative therapy. The mechanism underlying the AT is represented by a symbol and the number indicates the order of recurrences. The outcome of the ablation procedure is represented by the colour of the symbol (green: elimination of the AT, red: unsuccessful ablation).

Long-term outcome

During the follow-up period, a total of 77 catheter ablation procedures were performed. In 4 patients, 2 AT were eliminated during the same procedure. Eighty-one distinct AT (29 incessant) were mapped and treated with ablative therapy. In the entire study population, mapping revealed 34 AFL (CL= 372±99 ms), 32 IART (CL= 275±75 ms), 13 FAT (CL= 307±76 ms) and 2 "focal" AF. Ablative therapy was succesful in 69% of all AT; 19% of AT did not terminate and the other AT converted to either another AT (5%) or AF (7%) during ablation. Fluoroscopy time during mapping and ablation of IART (55±26* minutes) was significantly longer than during AFL (42±27 minutes) or FAT (40±25 minutes) procedures, P =0.03. The

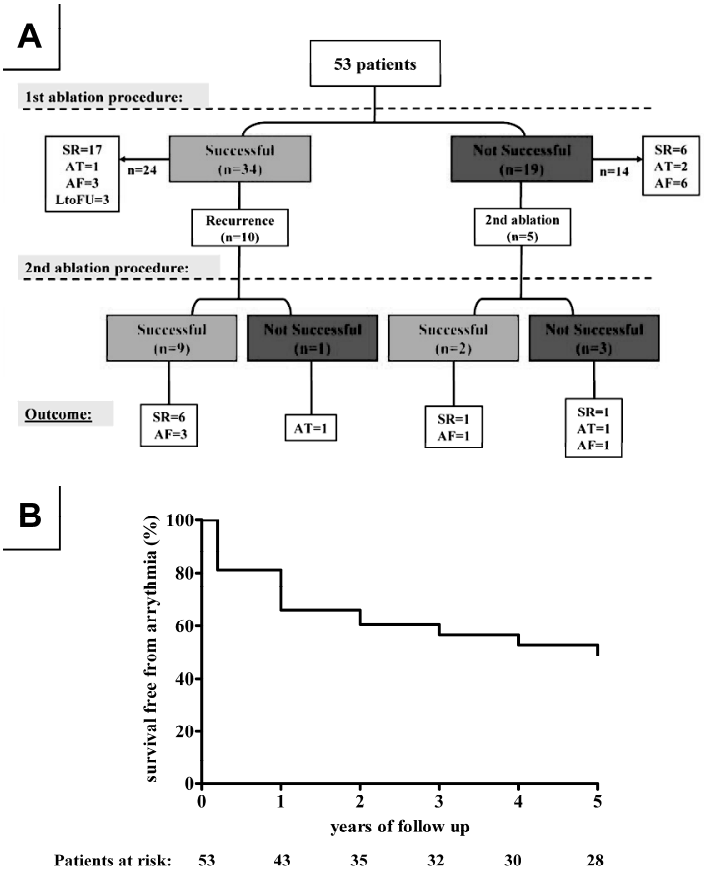

Figure 4.

Panel A: Flowchart describing the acute success, recurrences, additional ablation procedures and final outcome in the total patient population. Panel B: Kaplan Meier curve with survival free from arrhythmia for all patients (with and without acutely successful ablation). AF = atrial fibrillation, AFL = atrial flutter, AT = atrial tachycardia, LtoFU = lost to follow up, SR = sinus rhythm.

procedure time required for ablative therapy of IART was also longer (300±100* minutes, compared to AFL: 229±76 minutes, FAT: 211±66 minutes, P=0.001).

The relation between the results of the ablation procedure and long-term outcome is demonstrated in Figure 4. During last follow-up visit of the 50 patients excluding 3 subjects lost for follow-up, they had either sinus rhythm (N=31, 59%), a regular AT (N=5, 9%) or AF (N=14, 26%). Five patients died due to progressive heart failure 34±28 months after the ablation procedure; rhythm prior to death was sinus rhythm (N=2) and AF (N=3).

Paroxysms of AT were recorded in 12 sinus rhythm patients who underwent a successful ablation procedure. Anti-arrhythmic drugs were used by 18 patients with sinus rhythm. Persistent AF developed during the follow-up period in 14 patients. Seven patients had AF despite a successful ablation; in the other 7 patients, AF resulted from progression of AT to AF. Eleven of the 19 patients with an unsuccessful ablative therapy had persistent AT at the onset of the ablation procedure. Surprisingly, 7 patients who had one or more unsuccessful ablation procedures (no termination during ablation, conversion to AF or another AT) remained in sinus rhythm during the follow-up period.

DISCUSSION

This study reports on characteristics of recurrent AT after ablative of late post-operative AT during long-term follow-up in a large cohort of patients with predominantly complex congenital heart defects.

The majority of the ablation procedures were guided by 3-dimensional electro-anatomical mapping techniques enabling accurate localization of the arrhythmogenic substrate. The key findings of our study are that though ablative therapy of post-operative AT is most often successful, a large number of patients presented with recurrent AT. However, repeated ablative therapy of recurrent AT was effective in maintaining sinus rhythm in most of the patients. As the arrhythmogenic substrate of patients who had multiple ablation procedures was located at different atrial sites it is most likely that recurrent AT are the result of diffuse electro-pathological alterations of atrial tissue and/or progressive atrial myopathy instead of arrhythmogenicity of prior ablative lesions. Despite recurrent AT in many patients, the majority of the study population was in sinus rhythm at the end of the follow-up period.

Atrial tachyarrhythmia mechanism

The mechanism underlying late post-operative AT in our study population was variable; often AFL and IART, less frequently FAT and rarely focal AF. In a large number of patients, different mechanisms gave rise to successive AT.

Consistent with other reports on the mechanism underlying post-operative AT in patients with congenital heart disease, IART and AFL were most often observed.[8,27]

FAT were less frequently observed. We previously demonstrated that FAT arise mainly from areas where conduction is abnormal.[28,29] The atria of patients with CHD contain areas of fibrotic tissue giving rise to local dissociation in conduction and hence favor development of focal activity.[30,31] Reports on focal AF in CHD patients are rare and the mechanism underlying this AT in our patient population has recently been described in detail.[28,29]

Ablative therapy

Most ablation procedures performed in this study population were guided by a 3-dimensional electro-anatomical mapping system. Triedman et al. demonstrated the beneficial effect of an electro-anatomical mapping system over a conventional, fluoroscopy based mapping technique on the outcome of ablative therapy of post-operative AT. [32] However, compared to their ablation results, in our study 28% of the IART did not terminate during ablation despite the use of a 3-dimensional electro-anatomical mapping technique. This outcome emphasizes that ablation of IART remains very difficult despite facilitating mapping techniques.

Crucial pathways of the reentry circuit of most IART were located between areas of scar tissue, indicating necessity of accurate delineation of low voltage areas. [18] In patients who had multiple ablation procedures, target sites for ablation of successive AT were located at different atrial sites suggesting that new AT were not caused by arrhythmogenicity of previous ablative lesions. Most recurrences occurred in the first year after ablative therapy.

As the reentry circuit of post-operative AT in patients with CHD often consist of multiple re-entrant pathways a new reentry circuit may develop after ablation giving rise to early recurrences. Also, these new AT may simply be the result of diffuse electro-pathological alterations of the atrial tissue. Late recurrences also indicate progression of atrial myopathy.

After successful elimination of the AT, we did not induce other AT. It can be hypothesized that the incidence of redo-procedures can be reduced by additionally ablating other inducible AT. However, low voltage areas and prosthetic materials are present throughout the atria and multiple reentry circuits may therefore be possible. Extensive ablation at different sites in the atria would be required to eliminate additional IART (with unknown clinical relevance). This might increase the chance of constructing incomplete lesions which may in turn be pro-arrhythmic.

Another interesting finding is that in some patients who had several ablation procedures mapping revealed different mechanism underlying the AT; e.g. an IART during the first ablation procedure and a FAT in the next procedure. To our knowledge, the presence of different mechanisms underlying consecutive AT in patients with CHD has so far not been reported.

Surprisingly, despite some unsuccessful ablation procedures (no termination or conversion to another AT or AF) patients converted to sinus rhythm after the ablation procedure and remained in sinus rhythm during the follow-up period.

Atrial Fibrillation

At the end of the follow-up period, 26% of the patients had AF. Kirsh et al. demonstrated that AF is not an uncommon AT in CHD patients.[33] In some of our patients, AF resulted from progression of recurrent AT. Experimental mapping studies have demonstrated that a single macro-reentrant circuit may degenerate to AF if atrial tissue can not be activated at a high activation rate and fibrillatory conduction occurs consequently. [34] In line with these experiments, we have previously reported on focal activity giving rise to fibrillatory conduction in two patients with CHD. However, AF developed in 7 patients despite successful elimination of the AT by ablative therapy suggesting that different mechanisms causing AF in this patient group may be involved. Further studies in larger populations are required in order to gain insight into the mechanism of AF in this patient group.

Limitations

Holter monitoring was not consistently performed in every patient in order to determine the incidence of AT after ablative therapy. However, the majority of the CHD patients with an AT recurrence immediately visited the hospital because of symptoms. Data in this study are based on only 15/29 patients with recurrent AT who underwent more than one ablation procedure.

During the mapping procedure, crucial pathways of reentrant circuits were mainly selected by analyzing electro-anatomical activation maps. Entrainment techniques could not always be used as pacing in low voltage areas was often difficult and frequently resulted in conversion to another AT. When one AT was successfully ablated, we did not try to induce other AT.

When one AT converted to another AT, we did not target this AT as well. Consequently, we do not know whether ablation of multiple AT during one ablation procedure could have prevented future recurrences. In addition, irrigated tip ablation was performed in only a minority of the patients and 8 mm tip catheters were not used. Hence, the applied mapping and ablation techniques may account for a number of recurrences observed in this study.

CONCLUSION

Focal and reentrant mechanisms underlie late post-operative AT in patients with CHD.

Successive AT developing over time may be caused by different mechanisms. The complexity of the reentrant circuit is associated with the complexity of the CHD and corresponding extensiveness of surgical procedures. In patients who had multiple ablation procedures, the AT originated from different atrial sites suggesting that these new AT were not caused by arrhythmogenicity of previous ablative lesions. Recurrent AT occurred frequently after successful ablation and occurred mainly in the first year after treatment. The long-term outcome is often complicated by development of AF. However, the majority of the patients were in sinus rhythm.

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