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Predictors and Characteristics of Atrial Fibrillation Induced During Supraventricular Tachycardia Ablation Procedures

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Abstract

Introduction: Atrial fibrillation (Afib) inducibility during electrophysiological study (EPS) has been examined as an electrophysiological parameter, atrial vulnerability, related to clinical occurrence and recurrence of AF in many studies. However, the majority of these studies examined Afib induced in patients with paroxysmal supraventricular tachycardia (PSVT) who had clinically documented Afib prior to SVT ablation procedure.

Aim of Study: The purpose of this study was to determine the prevalence, predictors and charactaristics of Afib induced during PSVT ablation procedures.

Patients and Methods: Clinical and procedural data of all consecutive patients who underwent catheter ablation for PSVT from 2009 to 2015 were reviewed retrospectively.

Results: The study group included 347 studies (median age 39 years, 40.1% male), the PSVT type was AVNRT in 50.1% of patients, AVRT in 43.8% and AT in 5.2%. Afib was documented in 76 studies (21.9%). Patients with induced Afib were younger, more males, taller and had shorter tachycardia cycle length (TCL). Afib was induced more frequently in AVRT patients (61.3%) then AVNRT (34.7%) and AT (4%). In multivariate analysis, TCL and tall stature were the sole independent predictors for induction of Afib among other predictors that included age, gender and type of the tachycardia (Nagelkerke R square = 0.124 and p<.0001). The induction of Afib was associated with significantly longer study duration especially in AVRT utilizing a left lateral AP. Ablation of the primary PSVT was found to significantly minimizes Afib episodes (p<0.0001). The total number of Afib episodes were 153 episodes. Pacing induced Afib was the most prevalent mode of initiation (41.2%) followed by degeneration of PSVT (30.1%), catheter induced (15.7%) and spontaneously induced (13.1%). Spontaneous termination (55.6%) was the most prevalent mode of termination followed by DC shock (25.5.2%), organization to PSVT (19%) with a higher percentage of males among patients who had DC shocks.

Conclusions: The prevalence of Afib induced during PSVT ablation is 21.9%. Induced Afib was found to significantly prolong PSVT ablation procedure times. Short TCL and tall stature were independent predictor for Afib inducibility. Understanding the characteristics of initiation and termination may help to reduce the frequency of induced Afib.

Key Words: *Atrial fibrillation – Paroxysmal supraventricular tachycardia – Catheter ablation.*

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Introduction

ATRIAL fibrillation (Afib) is the most common sustained cardiac arrhythmia. Afib is associated with increased risk of all-cause mortality and morbidity including stroke, heart failure, dementia, embolic events, hospitalization and impaired quality of life. Patients with paroxysmal supraventricular tachycardia (SVT) have a higher risk than the remaining population to develop atrial fibrillation **[1,2]**.

The inducibility of Afib during electrophysiological studies (EPS) have been examined as an electrophysiological parameter (atrial vulnerability) related to clinical occurrence and recurrence of Afib in many studies **[3]**. In fact Afib inducibility during EPS was independent predictor for recurrence of Afib after catheter ablation in multivariate analysis in many studies **[4-6]**. However, the majority of these studies examined induced Afib in patients of SVTs who had clinically documented Afib prior to SVT ablation procedure. Character-

Abbreviations:

Afib	: Atrial fibrillation.
PSVT	: Paroxysmal supraventricular tachyacrdia.
AVNRT	: Atrioventricular nodal reentrant tachycardia.
AVRT	: Atrioventricular nodal reentrant tachycardia.
AT	: Atrial tachycardia.
AP	: Accessory pathway.
EPS	: Electrophysiologic study.
TCL	: Tachycardia cycle length.
HTN	: Hypertension.
DM	: Diabetes mellitus.
COPD	: Chronic obstructive pulmonary disease.
DC	: Direct Current Cardioversion.

istics of SVT patients without prior AFib who had inducible Afib episodes during their EPS and description of such episodes hadn't been fully delineated.

Patients and Methods

Study population:

We retrospectively reviewed data of all consecutive patients who underwent EPS/ablation procedures for PSVT over the period from September 2009 to the end of August 2015. The study included all Patients who presented for ablation of PSVT. We excluded patients who were younger than 18 years, those who had documented Afib episodes prior to SVT ablation, those were the presenting arrhythmia was atrial flutter, negative EP studies and studies in which data could not be retrieved from the EP recording system (Prucka, GE, USA).

Data collection:

Clinical data were collected including: Age, gender, height, weight, body mass index and presence of structural heart disease. Procedural data included type of the tachycardia [Atrioventricular nodal reentrant tachycardia (AVNRT), Atrioventricular reentrant tachycardia, (AVRT), Atrial tachycardia (AT) or dual Tachycardia)], Tachycardia cycle length (TCL, defined as first stable sustained tachycardia cycle length, cases with just echo beats were considered non-inducible), the duration of the study (from catheter placement to catheter removal), the use of drug provocation (atropine and/or adrenaline). In AVRT patients, we collected data regarding presence of manifest pre-excitation and site of accessory pathway (AP) which was classified as right, left and septal.

In patients who developed Afib, we collected the number of Afib episodes, mode of initiation of each episode which was classified as spontaneously induced Afib, pacing induced Afib, degeneration of SVT to Afib and catheter induced [This mode was thought to be the mechanism of initiation when one of the catheters showed the earliest intracardiac signal on the beat that initiated the episode in the absence of other modes (pacing or degeneration) or when annotated by the operator]. We also collected the mode of termination of each episode which was classified as spontaneous cardioversion to sinus rhythm, organization of Afib to either flutter or SVT and DC shock. Lastly, we collected the duration of each episode which was classified as less than 30 seconds, from 30 seconds to less than 1 minute, from 1 minute to less than 30 minutes and more than 30 minutes.

In pacing induced episodes, we collected the type of pacing (burst, decremental burst or extrastimulus), pacing channel (Atrial or ventricular), pacing coupling interval, rhythm before pacing and cycle length of rhythm before pacing.

Statistical analysis:

Data were coded and entered using the statistical package SPSS (Statistical Package for the Social Science; SPSS Inc., Chicago, IL, USA) version 20. Categorical data was summarized using median, minimum and maximum in quantitative data due to abnormal distribution and using frequency (count) and relative frequency (percentage) for categorical data. Comparisons between quantitative variables were done using the non-parametric Mann-Whitney tests for variables with two categories and Kruskal-Wallis test for more than two categories[7]. For comparing categorical data, Chi square (X^2) test was performed. Exact test was used instead when the expected frequency is less than 5 (Chan, 2003b) [8]. p-values less than 0.05 were considered as statistically significant. Non-parametric spearman correlation was used for correlating two abnormally distributed continuous variables. Paired samples t-test was used for comparison between number of episodes before and after ablation. Multivariate linear regression model included age, height, type of tachycardia and TCL. Analysis was done per patient for predictors of atrial fibrillation and per episode for Characteristics of episodes.

Results

We reviewed data of 445 studies for patients who had electrophysiological studies for PSVT over the study duration. Ninety-eight studies were excluded (31 studies for patients younger than 18 years old, 13 studies due to pre-existence of Afib, 30 studies for atrial flutter ablation, 20 negative EPS and 4 studies due to failure to retrieve data from the EP recording system). Off the 347 studies included, Afib was induced in 76 studies (21.9%).

Baseline characteristics:

In a total of 347 patients were studied, the median age of the study population was 39 years (range from 18 to 81 years). Males represented 40.1% (139 patients) of the study population. The median height and weight were 165cm and 77kg respectively while the median BMI was 27.7. Data regarding the presence of structural heart disease were available for 200 patients with the majority of them (90.5%) having no structural heart disease as detailed in Table (1).

Clinical tachycardia was inducible in 323 studies (93.1%) with a median cycle length (TCL) of 340msec (220msec-500msec). Drug provocation (Atropine 17.3% and/or adrenaline 4.3%) was used in 21.6% of studies. The median duration of the procedure was 75min (ranging from 21min-267 min). AVNRT occurred in 174 patients (50.1%) including 3 patients (1.7%) with atypical AVNRT (two fast-slow and one slow-slow). AVRT occurred in 152 patients (43.8%) including 14 patients (9.2%) with antidromic AVRT. AT occurred in 18 patients (5.2%). Dual tachycardia occurred in 3 patients (two patients with AVNRT & AVRT and the third one with AVNRT & AT).

In AVRT patients (154 patients), manifest pre-excitation was evident in 95 patients (61.7%). Site of AP was left in 65 patients (42.2%), right in 42 patients (27.3%) and septal in 47 patients (30.5%). Left lateral AP location was the most frequently encountered, in 39 patients (25.3%) followed by right posteroseptal location in 22 patients (14.3%). Atriofasicular APs occurred in 10 patients (6.5%) and multiple AP location occurred in 9 patients (5.8%).

Atrial fibrillation:

The incidence of induced Afib in our study was 21.9% (76 studies). The difference in baseline characteristics between the Afib group and the non Afib group was shown in Table (2).

The median TCL was significantly shorter in the Afib group [310msec (230-500) vs. 345msec (220-500), p=0.0001]. This difference was statistically significant for AVNRT and AVRT (p=0.003 and p=0.0001 respectively) but not for AT where the TCL was significantly longer in the Afib group (p=0.033, Table 3). The median study duration was significantly longer in the Afib group [85min (33-215) vs 71min (21-267), p=0.0001, studies with dual tachycardia were excluded]. There was no significant difference in induction of Afib was found with respect to the use of drug provocation [16 (21.1%) vs 59 (21.8%), p=0.893]. The distribution of different arrhythmias showed a statistical difference between both groups (p=0.009) with a higher percentage of AVRT in patients who developed Afib, (Fig. 1). A multivariate analysis for predictors of Afib inducibility was performed, including age, gender, height, type of the tachycardia and TCL. Only TCL and tall stature were independent predictors for induction of AF (Table 4). In model summary, r=0.357, r^2=0.127 and p<.0001.

Characteristics of Atrial fibrillation episodes:

The total number of Afib episodes was 153 episodes. Thirty-three (43.4%) of patients had more than one episode (13 patients had 2 episodes, 12 patients had 3, 2 patients had 4, 3 patients had 5 and one patient had 6, 7 or 12 episodes). Ablation of the primary arrhythmia was found to significantly minimizes the number of Afib episodes, as only 7 episodes (6 patients) were induced after successful elimination of the primary arrhythmia representing 4.6% of all episodes (CI 1.403-2.255, t=8.551, p<0.0001).

There was no significant difference in the number of episodes within the same study when tested against type of arrhythmia (p=0.267), presence of structural heart disease (p=0.636) or the use of drug provocation (p=0.983). The number of episodes showed no correlation with the TCL (r=–0.182 and p=0.125), age (r=–0.084 and p=0.472) or procedural time (r=0.215 and p=0.063).

As regard the mode of initiation for Afib episodes, pacing induced Afib was the most prevalent mode of initiation (63 episodes, 41.2%) followed by degeneration from SVT (46 episodes, 30.1%), catheter induced (24 episodes, 15.7%) and spontaneously induced (20 episodes, 13.1%).

There was no statistically significant difference between the different modes of initiation, considering the age (p=0.172, p=0.286), duration of the study (p=0.138, p=0.476), length of episode (p=0.460, p=0.235), presence of structural heart disease (p=.222) and the use of drug provocation (p=.563). However, number of episodes within each study was significantly lower when the mode of initiation of Afib was catheter induced (p=0.017, p=0.047) as shown in Fig. (2). Moreover, there was significant difference in the mode of initiation when the TCL was considered (p=0.026, p=0.006) as shown in Fig. (2), episodes initiated with degeneration from SVT had the shortest median TCL (297.5min, 240-455min) followed by pacing induced (302.5min, 240-455min) then catheter induced and spontaneously induced by the same median TCL (320min, 255-450min).

As regard to type of arrhythmia, the distribution of different modes of initiation was significantly different (p=0.001; Fig. 3) with pacing being the most frequent mode in AVNRT, degeneration from SVT the most frequent in AVRT and catheter induced in AT.

In pacing induced episodes, 52 episodes (82.5%) were induced while pacing in the atrium. Burst

pacing was the most frequent form of pacing inducing Afib in 53 episodes (84.1%) then decremental burst pacing inducing 7 episodes (11.1%) and extra-stimulus pacing inducing 3 episodes (4.7%). The median coupling interval of the pacing stimulus that induced AF was 260msec with a range from 210 to 600msec. Induction of Afib occurred in 27 episodes (43.5%) when the rhythm before pacing was the clinical tachycardia (median TCL was 285msec) represented as 23 AVRT episodes (18 episodes with atrial pacing) and 4 AVNRT episodes (3 episodes with atrial pacing). As regard to the mode of termination. Spontaneous termination was the most prevalent (85 episodes,55.6%) followed by DC shock (39 episodes, 25.5%) then organization (29 episodes, 18.9%).

As regard to the duration of the episode, 52 episodes (34.4%) were less than 30 seconds (median 8.5sec), 24 episodes (15.9%) were from 30 to 59 seconds (median 41 sec), 70 episodes (46.4%) were from 1 to 30 minutes (median 6.2min) and 5 episodes (3.3%) were longer than 30 minutes.

Table (1): Details of patients having structural heart disease.

Variables	Number of patients	Details
Structurally normal heart	181	–
Mitral valve disease	7	Mild mitral regurge (5 patients) Moderate mitral regurge (2 patients)
Dilated cardiomyopathy	5	–
S/P surgery	4	Mitral valve replacement Atrial septal defect closure Tetralogy of Fallot repair Right atrial myxoma excision
Congenital conditions	3	Ventricular septal defect Patent ductus arteriosus Persistent left superior venacava

Table (2): Differences between both groups in baseline characteristics.

	Afib group		Non-Afib group		*p*-value
	Value	n	Value	n	
Age, y	33.5 (18-71)	76	41 (18-81)	271	0.001
Gender, (Male)	38 (50%)	76	101 (37.3%)	271	0.045
Height, cm	170 (145-192)	59	165 (147-196)	192	0.0001
Weight, kg	78 (54-119)	59	76 (43-140)	192	0.917
BMI	27.5 (19.1-40.1)	59	27.7 (15.6-56.1)	192	0.172
Structural disease	1 (2.3%)	44	18 (11.5%)	156	0.064

- Data are presented as median, minimum and maximum or number and (%), n=number of patients.

Table (3): Difference between both groups in tachycardia according to arrhythmia type.

	Afib group		Non-Afib group		*p*-value
	Value	n	Value	n	
AVNRT, msec	317.5 (230-450)	26	350 (230-500)	145	0.003
AVRT, msec	305 (240-455)	43	335 (220-490)	90	0.0001
AT, msec	450 (410-500)	3	375 (250-450)	16	0.033

- Data are presented as median, minimum and maximum, n=number of patients.

Table (4): Multivariate linear regression to detect independent predictors of Atrial fibrillation.

	Unstandardized coefficients		Standardized coefficients	95.0% C.I. of B		*p*-value
	B	Std. Error	Beta	Lower	Upper	
Age	–0.002	0.002	–0.047	–0.006	0.003	0.526
Gender	0.034	0.067	0.039	–0.098	0.167	0.610
Height	0.010	0.004	0.183	0.002	0.018	0.017
Type of arrhythmia	0.080	0.064	0.093	–0.045	0.206	0.207
TCL	–0.002	0.001	–0.228	–0.003	–0.001	0.001

- Data are presented as *p*-value and confidence interval.

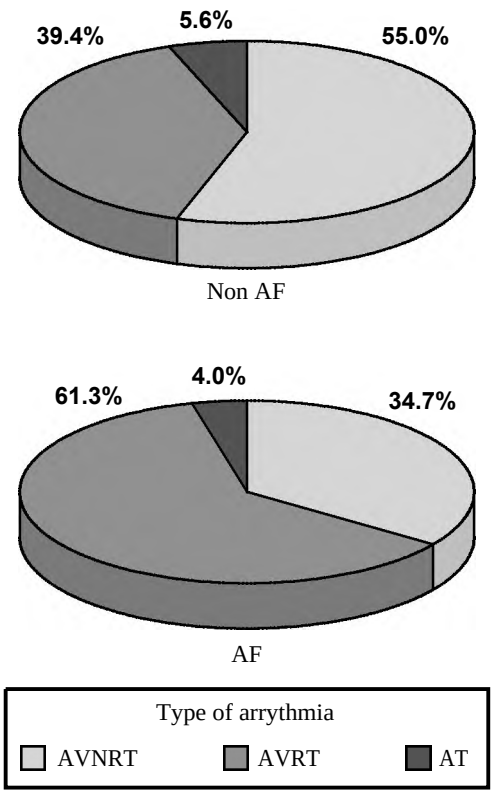

Fig. (1): Distribution of clinical arrhythmia between both groups.

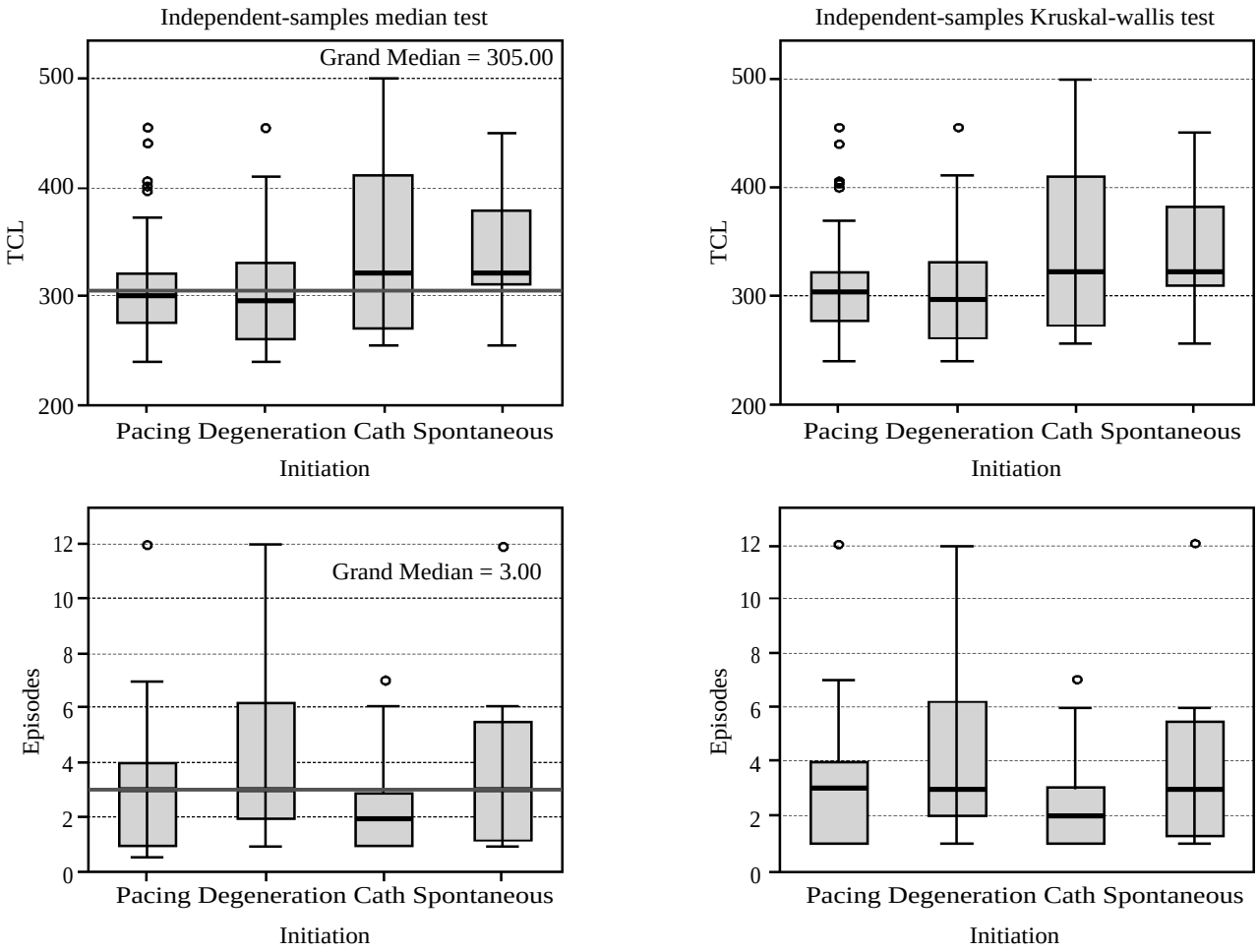

Fig. (2): Boxplot chart for TCL and number of episodes among different modes of initiation, number of episodes was significantly lower when the mode of initiation of Afib was catheter induced (p=0.017, p=0.047) and there was significant difference in the mode of initiation when the TCL was considered (p=0.026, p=0.006).

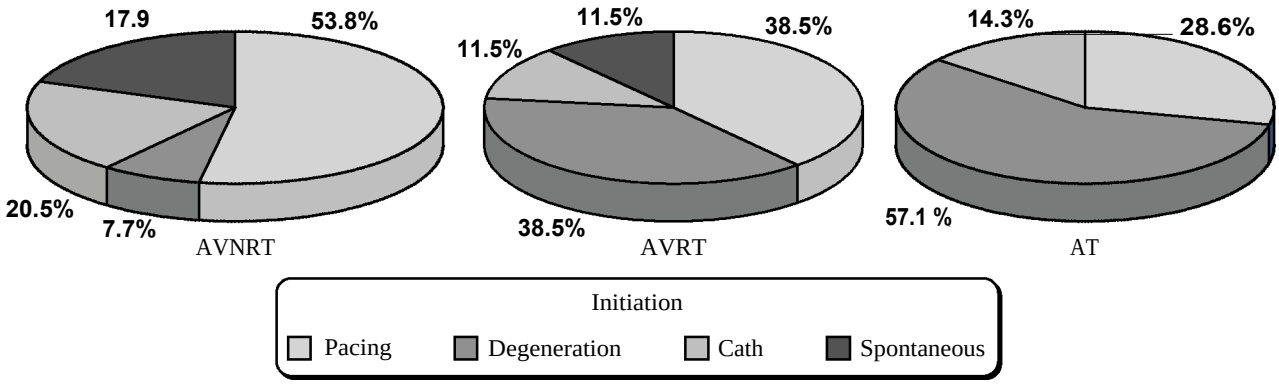

Fig. (3): Distribution of mode of initiation as regard to type of arrhythmia.

Discussion

Little evidence exists about the characteristics of induced Afib in SVT patients who had no prior documented Afib. Also, the description of Afib episodes as regard to mode of initiation and termination has not been fully delineated.

Cagli et al., [9] studied 24 AVRT and 27 AVNRT patients without prior history of Afib (or predisposing factors as organic heart disease, HTN, DM, COPD and thyroid disease) in two groups and assessed several markers of atrial vulnerability (Afib was defined when its duration exceeded 60sec). They concluded that ablation of the AP

results in an 'immediate' decrease in atrial inducibility, thus, denoting the contribution of AP to atrial vulnerability. This concept was first reported in AVRT patients by Hamada et al., [3], as they concluded that there are two mechanisms of Afib in patients with AVRT: One is reversible and dependent on AP related atrial vulnerability, and the other is intrinsic and independent of AP related atrial vulnerability, which is supported by the results of several studies [10-14]. The same concept regarding atrial vulnerability can be extended to Afib induced in AVNRT patients [15-18].

Arimoto et al., [6] studied 109 AVNRT patients without prior Afib in two groups based on age. They assessed new onset clinical Afib as one of their outcomes and found that atrial vulnerability during EPS (induction of Afib, lasting at least 30secs, without isoproterenol infusion) was the only predictor for occurrence of Afib in multivariate analysis among age, gender, LA diameter and EF.

Amasyali et al., [5] studied 533 AVNRT patients in two groups based on prior history of Afib and showed significant reduction in atrial vulnerability (at least 30secs; they excluded Afib induced by catheter manipulation and decremental pacing) after ablation in both groups. They found that both atrial vulnerability after successful ablation and LA diameter >40mm, were independent predictors for Afib recurrence in multivariate analysis that included age, gender, EF, HTN, DM and many other predictors, but in the group with prior Afib (36 patients). In the group without prior Afib (497 patient) atrial vulnerability was observed in 57 patients before ablation and 19 patients after, while clinical Afib occurred in only two patients on follow-up.

To the best of our knowledge, only these studies [5,6,9] reported evidence related to our study (Afib induced during EPS), while the rest of evidence mentioned throughout this discussion is related to clinically documented Afib, which should be interpreted with respect to differences in examined populations and study designs.

Incidence of Atrial fibrillation:

The incidence of induced Afib in our study was 21.9% (76 patients from 347). Afib was induced in 30.3%, 14.9% and 16.7% in patients with AVRT, AVNRT and AT respectively. Previous studies reported incidence of induced Afib was 11.4% in AVNRT [5,6] and 29% in AVRT [9]. Aytemir et al., [19] reported an incidence of 27% in patients with manifest AP, but with prior Afib, while Oddsson et al., [14] reported 44.4% vs. 13.9% in AVRT patients with and without prior Afib respectively.

Ozcan et al., [20] reported an incidence of 27% of Afib in 1573 patients with SVT (they included AFL in the study population (29.6%), which represented 57.5% of patients with clinical Afib). After excluding AFL, the calculated incidence was 9.09%. Of note that, the main difference in contrast to our study was that they considered clinical Afib not that induced during EPS.

Clinical predictors of Atrial fibrillation:

The current study revealed that patients who had Afib were significantly younger, in contrast to the fact that many studies showed that older age seems to be the most clinically relevant predictor of continued clinical Afib after SVT ablation. However, in these studies the mean age of Afib patients was 58,8y±15 [20], 50y±19 [4], 49y±17 [5] and 49y±17 [21]. On the other hand, several studies showed that age wasn't a predictor of clinical Afib in SVT patients [6] with the mean age being 32y±8 [19] and 39y±9.5 [9].

In our study the median age was 39 years which can relate our population more to studies in which age wasn't a predictor of Afib. A finding that can explain why older age wasn't a predictor for Afib in our study, but how could we explain younger age being a predictor for Afib in our study? It has been proven in previous studies that patients with SVT without associated cardiovascular disease when compared to those with cardiovascular disease are younger (37 versus 69 years; p=0.0002) and have faster heart rates (186bpm versus 155bpm; p=0.0006) [22,23]. Since 90% of our patients had no structural heart disease and since short TCL was the independent predictor of induction of Afib in multivariate analysis in our study, these two particular patients characteristics may explain why younger age was a predictor of Afib in the present study. This may suggest that, in a population with absence of structural heart disease, the substrate for Afib may be more related to SVT properties as rapid atrial rates, which is more evident in younger patients, rather than atrial structural abnormalities known to be related to aging.

The relation between Afib and structural heart disease is an established fact in literature. Several studied [4,20] reported correlation between Afib in patients with SVT and structural heart disease (esp. left atrial diameter >40mm that was in some reports an independent predictor) while others didn't [6,14,19,21]. Again, this can be attributed to the difference in studied populations as demonstrated previously as related to age.

Looking at the other side of the coin, Sciarra et al., [24] studied patients referred for Afib ablation and have an underlying SVT that was previously unknown. They found that patients without inducible SVT and treated with Afib ablation were older and had a higher prevalence of echocardiographic signs of structural heart disease (e.g. left atrial diameter) when compared with patients who had only SVT ablation. For example, the mean left atrial diameter was 44.0±2.2 vs. 37.0±3.0 (p<0.01). So, we may suggest that, although the link between Afib and structural heart disease is well established in literature, patient with SVT associated Afib may represent an intermediate zone between patients with Afib who are older and known to be linked to structural heart disease, and patient with SVT who are younger and not known to have this link.

Females represented 59.9% of our population which goes the predominance of female gender in SVT patients [25]. Several studies reported male gender as a predictor for the occurrence of Afib [4,20,21]. Our study revealed that patients who had induced Afib were significantly taller than those who hadn't. Height hadn't been evaluated previously in any of the studies evaluating Afib in SVT patients. However, Tall stature was reported to be associated with clinical Afib in several studies [26,27].

Electrophysiological characteristics:

The median tachycardia cycle length (TCL) in our study was significantly shorter in the Afib group, which was also confirmed in multivariate analysis. Chen et al., [28] observed that TCL at the time of EPS was significantly shorter in WPW patients with Afib than those without Afib. This finding suggests that it is easier to develop Afib in a rapid episode of sustained AVRT. SVT can increase atrial vulnerability as a result of a shortened atrial cycle length, increased sympathetic tone and atrial stretch because of hemodynamic changes that occur during the tachycardia.

Induced atrial fibrillation:

Little evidence exists about the characteristics of Afib episodes induced during EPS in SVT patients. So, we are probably the first to describe in details such characteristics in patients without prior history of Afib.

The current study demonstrated that the most prevalent mode of initiation was pacing induced accounting for 41.2% of the episodes then degeneration from SVT, catheter induced and spontaneous. When the mode of initiation was degeneration from SVT, there was a shorter TCL (median 297.5 min) and a higher percentage of males (76.1%) and AVRT (88.9%). This can be explained in the light of the characteristics of our patients with induced Afib being younger with less structural heart disease, more males, more AVRT and shorter TCL more prone to degeneration. Thus, degeneration may be added to these characteristics. Aytemir et al., [19] reported induced Afib episodes (lasting at least 60sec) in 21 patients having manifest APs and prior history of Afib and didn't report any degeneration induced episodes (wasn't considered). Instead, they reported pacing as mode of induction in 86% and catheter as 3.14%.

In AVNRT patients pacing was significantly the most frequent mode (53.8%) while degeneration from SVT was significantly the least frequent mode (7.7%). This correlates with the results of Amasyali et al., [5] that showed that, in 89 patients with induced Afib (32 with prior clinical Afib and 57 without prior Afib), Afib (lasting at least 30sec) was induced by degeneration of AVNRT in 23 patients (26%) and with programmed atrial stimulation in the resting 66 patients (74%).

In patients with AT the most prevalent mode of initiation was catheter induced. This may be explained by the common association between AT and structural heart disease with associated increased vulnerability within the atria; however, there is no available data regarding characteristics of Afib induced in AT patients. Moreover, the number of episode in AT patients is our study is small to allow us to suggest such relation, so studies with larger numbers are needed. As regard to the mode of termination Amasyali et al., [7] reported that 80% of his patients restored sinus rhythm spontaneously, while 20% required DC shocks which is comparable to our results.

Study limitations:

Firstly, the results of this study should be interpreted in light of the limitations imposed by a retrospective study design. Secondly, data regarding the presence of structural heart was limited and available for only 60% of our population. It would have been interesting to include echocardiographic parameters such as the left atrial diameter among the studied risk factors of Afib in order to provide a more complete risk profile. Finally, there is no standard stimulation protocol for evaluation of atrial vulnerability.

Conclusion:

The prevalence of induced Afib in patients with PSVT without prior documented Afib was 21.9%. Patients with induced Afib were more males,

younger, taller, had shorter TCL and had AVRT more frequently. TCL and tall stature were independent predictors for induction of Afib in multivariate analysis. The induction of Afib was associated with significantly longer study duration. Ablation of the primary arrhythmia was found to significantly reduce atrial vulnerability. Better understanding of the characteristics of episode initiation and termination may help to reduce Afib induction and potentially decreases the throughput time to the EP lab.

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خصائص وعوامل توقع حث التذبذب الأينى أثناء إجراء الدراسة الكهروفيسيولوجيه والكى لحالات تسارع ضربات القلب الفوق بطينى

يهدف البحث إلى تحديد نسبة الحدوث وعوامل التوقع لحدوث استحثاث التذبذب الأذينى أثناء إجراء الدراسة الكهروفيسيولوجيه والكى لحالات تسارع ضربات القلب الفوق بطينى وكذلك الخصائص الكهروفيسيولوجيه للتذبذب الأذينى المستحث. شملت العينة البحثية كل المرضى الذين خضعوا لإجراء الدراسة الكهروفيسيولوجية والكى للبؤر المسببة للتسارع الفوق بطينى فى الفترة من العام ٢٠٠٩ وحتى العام ٢٠١٥ وذلك بأثر رجعى. إجمالى عدد الدراسات الكهروفيسيولوجيه التى تم دراستها ٣٤٧ دراسة. متوسط أعمار المرضى كان ٣٩ عام منهم ٤٠.١٪ ذكور. وقد كان التسارع الفوق بطينى لـ ٥٠.١٪ منهم ناتج عن التسارع الموجى فى العقدة الأذينية البطينية، ٤٣.٨٪ ناتج عن التسارع الموجى بين الأذين والبطين و ٥.٢٪ ناتج عن تسارع أذينى. تم تسجيل حدوث التذبذب الأذينى فى ٧٦ دراسة بنسبة ١٢.٩٪ من إجمالى الدراسات أدى حدوثه إلى زيادة مدة الاجراء بصورة ملحوظة. طول القامة وقصر طول موجه التسارع الفوق بطينى بحد ذاتهم عوامل توقع مستقلة لحدوث استحثاث التذبذب الاذينى. فهم الخصائص والعوامل المسببة لحدوث حث التذبذب الاذينى وكذا المؤدية لتوقفه قد يساعد فى منع حدوثه أثناء هذا الإجراء لاحقاً.