Characterization, Mapping and Catheter Ablation of Recurrent Atrial Tachycardias Following Stepwise Ablation of Long-Lasting Persistent Atrial Fibrillation

Short Title: Rostock et al. ATs following chronic AF ablation

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Abstract:

Background: Atrial tachycardias (AT) often occur after ablation of long-lasting persistent AF (CAF) and are difficult to treat conservatively. This study evaluated mechanisms and success rates of conventional mapping and catheter ablation of recurrent ATs occurring late after stepwise ablation of CAF.

Methods and Results: A total of 320 patients underwent de-novo ablation of CAF using a stepwise ablation approach in 2006/2007 at our institution. This study comprised patients who presented with recurrent ATs at their first redo procedure after initial de-novo CAF ablation (IP). All procedures were guided by conventional mapping techniques, exclusively. Sixty-one patients (63 ± 10 y, female = 14) presented with their clinical AT at their redo procedure 7.7 ± 4.4 months after IP. A total of 133 ATs (2.2 ± 0.9 per patient) were mapped. Forty-four (72 %) were due to reentry, 17 (28 %) were focal ATs. Reentry-ATs were mainly characterized as roof- and perimitral flutter (43 % and 34 %, respectively). Focal ATs mainly originated from the great thoracic veins (pulmonary veins [PV]: 41 %, coronary sinus: 23 %). Forty-five (74 %) patients had conduction recovery of at least 1 PV (mean 1.2 ± 0.8). Overall, 124 (93 %) ATs could be ablated successfully. The mean procedure duration was 181 ± 59 min with a mean fluoroscopy time of 45 ± 21 min. After a mean follow-up of 21 ± 4 months, 50 (82 %) patients were free any arrhythmia recurrences after a single redo procedure.

Conclusions: Although late recurrent ATs may have complex mechanisms, catheter ablation exclusively guided by conventional techniques is highly effective with excellent acute and long-term success rates.

Keywords: atrial tachycardia, atrial fibrillation, mapping, catheter ablation, mechanisms
Catheter ablation of long-lasting persistent atrial fibrillation (CAF) using the stepwise ablation approach requires sequential identification and elimination of arrhythmogenic sources and substrates in both atria and the great thoracic veins to achieve AF termination\(^1\)\textsuperscript{,}\textsuperscript{4}. Since the vast majority of patients convert to an atrial tachycardia (AT) upon AF termination, ablation of these subsequent organized arrhythmias is an integral part of the procedure in an effort to achieve sinus rhythm. It is still a matter of debate whether subsequent ATs occur as a proarrhythmic consequence of the ablation itself or if they reflect unmasked drivers contributing to the global arrhythmogenic processes of CAF\(^5\)\textsuperscript{,}\textsuperscript{6}. Irrespective of its electrophysiological causes, there often is a therapeutic imperative of catheter ablation for subsequent ATs because patients rather have more debilitating symptoms due to a fast ventricular response difficult to manage pharmacologically, and moreover, ATs often recur after cardioversion\(^7\). Thus, the aim of this study was first, to characterize electrophysiological mechanisms of ATs that occur after stepwise ablation of CAF and second, to evaluate the success rates of catheter ablation for late recurrent ATs guided exclusively by conventional mapping techniques.

**Methods**

**Study Population**

A total number of 320 patients underwent de-novo ablation of CAF using the stepwise ablation approach in 2006/2007 at our institution. Of these, 93 (29 %) patients had recurrence of AF, 128 (40 %) recurrence of AT and 99 (31 %) were in sinus rhythm. There were no baseline differences in demographics, co-morbidities, cardiac function and dimensions between patients with AT and AF recurrences. However, patients with AF recurrences had significantly longer AF history than those with AT recurrences (61 ± 29 vs. 85 ± 76 months, \(p = 0.021\)). All but 4 (4 %) patients with AF recurrences underwent a second
ablation procedure for CAF. The remaining 4 patients were left in AF and treated with ventricular rate control. Only 4 (3 %) out of the 128 patients with AT recurrences did not undergo a redo procedure because re-initiation of antiarrhythmic drug treatment sufficiently prevented further arrhythmia recurrences (3 amiodarone, 1 flecainide). Approximately half of the patients with AT recurrence (n = 128) presented with the clinical AT at their first redo procedure (n = 61, 48 %). These 61 patients (mean age 63 ± 10 years, 14 women) are the focus population of this study. In all but 2, AF was terminated through ablation during the index procedure. The patients’ baseline characteristics are presented in table 1.

Antiarrhythmic drug treatment, including amiodarone, was discontinued after the index procedure (IP) for CAF and re-initiated at the discretion of the referring cardiologist or by our institution after at least one recurrence of AT. To characterize only clinical ATs occurring spontaneously after stepwise ablation of CAF, patients with arrhythmia recurrences who presented in sinus rhythm or AF at the redo procedure were excluded from this study. All antiarrhythmic drugs, including amiodarone, were discontinued prior to re-do procedures.

Classification of ATs

Atrial tachycardia was defined, in accordance with the current consensus statement of a joint expert group 8, as an organized atrial activity with a consistent endocardial activation sequence and monomorphic P-waves. An AT with a stable cycle length was deemed macroreentrant, when a consistent post-pacing interval was observed at distant sites of the circuit and the tachycardia cycle length could be demonstrated around the presumed circuit. Focal AT was defined as an atrial activity originating from a discrete site activating the surrounding tissue centrifugally and demonstrating features consistent with a focal mechanism (see below). Localized reentry was defined as atrial activity confined to a small area where continuous signals on the bipoles of the mapping catheter spanned ≥ 85 % of
tachycardia cycle length and respond by consistent post-pacing intervals to entrainment pacing. The “clinical AT” was defined as the tachycardia which was manifest at the beginning of the first redo procedure after the IP for CAF. “Subsequent ATs” were defined as tachycardias that occur after termination of the “clinical AT”.

**Ablation Approach of the Index Procedure**

The IP for CAF was uniformly performed by using the stepwise ablation approach aiming at AF termination in all 320 consecutive patients. Details of this particular ablation approach have been described previously. In brief, as the first step, the pulmonary veins (PV) were electrically isolated during AF using a contiguous circumferential lesion around the ipsilateral PVs. Secondly, complex fractionated atrial electrograms as well as areas displaying specific local activation characteristics (activation gradients, centrifugal activation spreading and localized rapid activity) were targeted for ablation at all sites presumably involved in the arrhythmogenic process perpetuating CAF. These were targeted in both atria and the coronary sinus (CS), according to the respective arrhythmia response to ablation as characterized by local cycle length behavior. In the event of termination of CAF to an AT, these were also targeted for further ablation in an effort to achieve sinus rhythm in all patients through ablation only.

**Electrophysiological Study**

All patients underwent transesophageal echocardiography within 24 hours prior to the procedure to exclude atrial thrombi. The procedure was performed under sedation with propofol and under continuous monitoring of blood pressure and saturation.
Surface electrocardiograms and bipolar endocardial electrograms were continuously monitored and stored on a computer-based digital amplifier/recorder system (Bard Electrophysiology, Lowell, MA, USA).

The following catheters were introduced via a femoral vein access: (1) A steerable decapolar catheter (Inquiry™, IBI, Irvine Biomedical, Inc., Irvine, CA, USA) was positioned within the coronary sinus (CS); (2) a circumferential decapolar diagnostic catheter (Lasso, Biosense-Webster, Diamond Bar, CA, USA) for mapping of the PVs; (3) a non-steerable quadripolar diagnostic catheter (Inquiry™, IBI, Irvine Biomedical, Inc., Irvine, CA, USA) was placed in the right atrial appendage; and (4) a 3.5 mm externally irrigated-tip ablation catheter (Biosense-Webster, Diamond Bar, CA, USA) was used for mapping and ablation. The Lasso and ablation catheter were stabilized with long sheaths (SL0, Daig Inc., St. Jude Medical, St. Paul, MN, USA) continuously flushed with heparinized saline solution. Access to LA was achieved by a single transseptal puncture with the two catheters placed into the left atrium via the same puncture. A single bolus of 50 IU/kg body weight heparin was administered after transseptal puncture. The activated clotting time (ACT) was assessed every 30 minutes and maintained within a range of 250-300 seconds.

**Conventional Mapping and Ablation of Atrial Tachycardia**

**1) PV Re-Isolation**

As a first step, all PVs were mapped with a circumferential mapping catheter and re-isolated in case of electrical recovery. PV re-isolation was performed during AT in all patients with reconnection. For appropriate mapping of single gaps in the circumferential lesion around the ipsilateral PVs, the circumferential mapping catheter was placed sequentially in the superior and inferior ipsilateral veins to assess the earliest activation.
After PV re-isolation, the circumferential mapping catheter was placed into the left atrial appendage (LAA) with the catheter shaft positioned at the superior aspect of the LAA. This catheter then was also used for entrainment and activation mapping.

2) Differentiation between Reentry and Focal Atrial Tachycardia

The following AT characteristics were used to differentiate between focal and reentry mechanisms: i) tachycardia cycle length variation > 15% is in favor of a focal mechanism\textsuperscript{7,10}, ii) stable AT cycle lengths and consistent return cycle lengths in response to overdrive pacing at different pacing sites is indicative for a reentrant mechanism (see below). iii) The presence of a significant isoelectric line in between the P-waves in all 12 ECG leads was suggested to indicate a focal origin\textsuperscript{11,12}. However, it does not indicate a focal mechanism, i.e., automaticity or triggered activity.

3) Entrainment Mapping

Entrainment mapping was performed with a voltage twice the diastolic threshold and a pulse width of 1.0 ms. In the LA, at least six different sites were paced by using the Lasso catheter and the roving mapping catheter (roof, septal, inferior, lateral, posterior and anterior). Entrainment mapping was then continued in the distal and proximal CS, the right atrial appendage (RAA) and the cavotricuspid isthmus, when appropriate. Each site was paced at least twice to ensure consistency. In case of consistent post-pacing intervals, the site with the shortest return cycle (usually < 20 ms longer than the tachycardia cycle length) was considered to be an integral part of the AT circuit (Figure 1). ATs with significant cycle length variation, tachycardia overdrive pacing was performed in order to identify the site with the shortest post-pacing intervals which was considered to be in a close proximity to the AT focus\textsuperscript{13}.
4) Activation Mapping

According to the information gained from the previous steps, confirmation of the assumed mechanism was attempted by conventional activation mapping. For macroreentrant ATs, the entire tachycardia cycle length should be demonstrated within the chamber to which it is confined and around the central obstacle of the circuit, e.g., the mitral annulus in case of perimtrial flutter. In ATs due to localized reentry, most of the tachycardia cycle length can usually be observed traversing the two bipoles of the mapping catheter (Figure 2) and centrifugal activation spreading from this site can be demonstrated. However, in contrast to most focal ATs, localized reentrant ATs can be entrained with consistent post-pacing intervals (Figure 2). Focal ATs were mapped by assessing the earliest endocardial activation in relation to P-wave onset. In cases where the P-wave was superimposed by ventricular repolarization, AV block was induced by adenosine and the intracardiac electrogram with the closest activation to P-wave onset was determined and used for temporal mapping.

5) Procedure Endpoint

The endpoint of the procedure was termination of AT(s) by ablation to sinus rhythm and non-inducibility of any atrial tachyarrhythmia. Inducibility was tested exclusively by burst pacing from two different pacing sites. If the clinical AT converted to a different AT or in case of an induction of another AT, the ablation was continued aiming at termination to sinus rhythm. The mapping protocol is schematically presented in Figure 3.

Follow-Up

All patients were seen regularly every three months in our outpatient clinic. Prior to visits, the patients received at least two Holter ECGs and additional 12-lead ECGs performed by their referring physicians. Seven-day Holter monitors were not routinely obtained. Detailed history of the patients’ symptoms suggestive for potential AT recurrences was taken.
Antiarrhythmic drug treatment, including amiodarone, was discontinued after each procedure in all patients. Patients with more than 1 recurrence of AT were scheduled for a redo procedure and antiarrhythmic drug treatment was re-initiated, if not contraindicated, to bridge the time to the procedure.

**Statistical Analysis**

Continuous data are given as mean ± SD and range in brackets. Comparison between groups was carried out using either the Student’s t-test (i.e., reentry vs. focal ATs and epidemiological parameters of patients with 1 vs. >1 AT). The Wilcoxon rank-sum test was used for the comparison of the index procedural data of patients with 1 vs. >1 AT. A two-tailed p-value < 0.05 was considered significant.

**Results**

**Procedural Data**

The procedure was performed at a mean of 7.7 ± 4.4 [3 – 20] months after the index procedure for CAF. All patients had at least one attempt to treat the AT conservatively by cardioversion. In 10 patients, amiodarone therapy was reinitiated and continued until the redo procedure. In 18 patients, a treatment with class I-C antiarrhythmics was re-initiated but discontinued in all after at least 2 AT recurrences. Eleven patients received beta-blockers for ventricular rate control. The remaining 22 patients were left without antiarrhythmic treatment until the redo procedure. Although it was not predetermined by the study protocol, no patient underwent ablation for AT within the first three months after the index procedure. A mean of 2.2 ± 0.9 [1 – 5] ATs per patient were mapped with an overall number of 133 ATs. The mean procedure duration was 181 ± 59 [60 – 310] min with a mean fluoroscopy time of 45 ± 21 [6 – 102] min.
**PV Conduction Recovery and PV-related ATs**

Electrical conduction recovery of at least 1 PV was observed in 45 (74 %) patients with a mean of 1.6 ± 0.5 PVs per patient. There was no significant difference in the incidence of electrical reconduction of the given PVs (left superior PV – 22 %, left inferior PV – 20 %, right superior PV – 22 %, right inferior PV – 32 %). However, “en-bloc” recovery of ipsilateral PVs was more often observed for the right as compared to the left veins (left PVs – 2% vs. right PVs – 20 %).

In 7 (11 %) patients, the clinical AT was related to a recovered PV and terminated during re-isolation. These ATs had a mean cycle length of 270 ± 40 ms. Only 2 of them terminated directly to sinus rhythm, whereas the remaining five patients converted to a different AT. One patient with AT termination during PV re-isolation did not have a PV tachycardia but the electrically reconnected left inferior PV served as the critical gap for perimital flutter (Figure 4).

**Mechanisms and Ablation of Clinical ATs**

During the first ablation procedure for AF, left atrial linear ablation had been performed in 23 (38 %) patients with 13 mitral isthmus lines and 17 roof lines (2 lines in 7 patients). Of these, 9 (69 %) mitral isthmus lines and 14 (82 %) roof lines were successfully blocked. In the remaining attempted linear ablation, completeness of the lines could not be achieved or evaluation of the line failed (e.g., in case of CS isolation).

Following this first ablation procedure, the clinical AT recurred after cardioversion, persisted over at least several days prior to the EP study and was present at the beginning of the procedure in all 61 study patients. The mean cycle length of the clinical AT was 274 ± 41 ms. The majority of them were due to reentry and less than one-third had a focal mechanism. Notably, the “common types” of right and left atrial macro-reentrant ATs constituted more
than 90% of all clinical ATs with a reentry mechanism while focal ATs predominantly originated from the PVs and the coronary sinus. In 4 patients, adenosine was administrated to unmask P wave morphology without changing or terminating the AT. There was no significant difference in the mean cycle length of reentrant as compared with focal ATs (275 ± 40 ms vs. 271 ± 46 ms, p = ns). Details regarding AT mechanisms, incidence and cycle lengths are presented in table 2.

Termination of the clinical AT by ablation was achieved in 60 (98%) patients. In 1 patient, the AT could not be identified accurately during a procedure of 260 min with a fluoroscopy time of 59 minutes and consequently electrical cardioversion was performed. In only 16 (26%) patients, successful ablation of the clinical AT resulted in conversion to sinus rhythm while 45 (74%) converted to a different AT (Figure 5).

**Mechanisms and Ablation of Subsequent ATs**

Among the 45 patients who converted to a different AT after termination of the clinical AT, a total number of 72 ATs were mapped (Figure 5). The mean cycle length of these subsequent ATs was 343 ± 77 [240 – 700] ms. Similar to the clinical ATs, the majority of these subsequent ATs also had a reentrant mechanism (47 [65 %] vs. 25 [35 %]). In contrast to the clinical ATs, subsequent ATs due to reentry had a significantly shorter mean tachycardia cycle length as compared to focal ATs (319 ± 77 ms vs. 392 ± 72 ms; p < 0.001). Furthermore, the mean cycle lengths of ATs increased gradually with the number of eliminated intermediate subsequent ATs before sinus rhythm was achieved (Figure 5). Detailed data of subsequent ATs are given in table 3. Overall, 64 (89%) of these 72 subsequent ATs could be ablated successfully. Entrainment pacing resulted in termination to sinus rhythm in 3 (4%) cases and conversion to another AT in 2 (3%) patients. Entrainment pacing resulting in induction of AF was not observed.
It is noteworthy that the subsequent ATs to which the clinical AT converted had markedly different mechanisms and spatial localizations as compared to the clinical ATs. The reentrant circuits were mainly localized to relatively uncommon atrial regions, e.g. the anterior LA and the coronary sinus ostium, and did not rotate around predefined anatomical obstacles in over 40% of patients (see table 3). Subsequent focal ATs were largely distributed throughout both atria and were less frequently confined to the great thoracic veins.

There were no statistical differences in demographic, epidemiological, or index procedural data between patients with only one AT compared with those who had multiple ATs. However, the interval between the index procedure for CAF and the first redo procedure for AT was significantly shorter in patients with multiple ATs (table 4).

**Role of Recovered Substrates in the Mechanisms of Recurrent ATs**

Among the 61 study patients, only 18 (30%) presented with ATs that had occurred and had been ablated during the index procedure after termination of CAF. These included 8 perimitral flutters, 4 roof-flutters, 4 cavotricuspid isthmus dependent flutter, 4 PV tachycardias, and 2 focal ATs. Four patients had recurrences of 2 different ATs. In 3 patients with recurrences of perimitral flutter, bidirectional block of the left atrial isthmus could not be achieved during the index procedure.

**Follow-up**

After a mean follow-up of 21 ± 4 months post redo procedure, 50 (82%) patients were free of any arrhythmia recurrences after a single redo procedure. Five (8%) patients experienced recurrence of AT after the first redo procedure and were re-ablated. However, three of these patients still had AT recurrences after their second redo procedure and required a third re-ablation for AT. One of them only became free of AT recurrences after 4 redo
procedures. Two (3 %) patients had AT recurrences after the first redo procedure and refused re-ablation. Notably, none of the patients had recurrences of AF after the first redo procedure.

Discussion

The present study demonstrates five key observations about the mechanisms of recurrent ATs that present late after successful stepwise ablation of CAF. First, only a small proportion of recurrent ATs is dependant on or related to reconnected PVs. Second, the vast majority of clinical ATs has a reentrant mechanism with “common” types of left or right atrial macroreentrant circuits, but rarely resulted from recovered substrates that had been previously ablated. Third, the clinical ATs could be ablated successfully in almost all patients. Fourth, nearly three-fourths terminated by conversion to another AT and not directly to sinus rhythm, indicating that the majority of patients do have a substrate for multiple rather than a single AT. Interestingly, the majority of these subsequent ATs was localized to relatively atypical anatomical regions and distributed throughout both atria. Finally, catheter ablation guided exclusively by conventional mapping techniques is an effective treatment with high immediate and long-term success rates in patients with recurrent ATs.

Assessment of Mechanisms and Localization of ATs using Conventional Mapping Techniques

In previous studies that evaluated mechanisms of subsequent ATs and the efficacy of catheter ablation, the procedures were performed under the guidance of a three-dimensional mapping system. Although the use of an electroanatomical mapping system may help to understand the arrhythmia by its visualization on a virtual anatomy, it has the disadvantage that individual maps are required for each AT. This can be labourious and time consuming with the result of prolonged procedure times, particularly in case of multiple ATs.
Therefore, Jais and co-workers\textsuperscript{11} proposed a new mapping algorithm for subsequent ATs occurring immediately after CAF termination. With a deductive mapping strategy, the mean diagnostic time could be reduced to an impressive 10 minutes per AT. In their approach, AT cycle length stability was assessed as the first step, followed by a detailed activation map to delineate the propagation direction of the AT. Afterwards, entrainment mapping at the deduced critical site of the AT was used to confirm the diagnosis as the final step before ablation was attempted. In our study, the combination of a detailed entrainment and activation mapping was used. The additional detailed entrainment mapping has the potential to gain information on the arrhythmia mechanism, e.g. by observing inconsistent post-pacing responses at a single pacing site in case of focal AT. This is particularly useful when focal AT appear to have stable cycle lengths. Furthermore, the length and range of variation of the post-pacing interval provides information about the distance of the pacing site to the AT focus\textsuperscript{13}. Although these pacing maneuvers have the potential to terminate or convert the interrogated AT, such counterproductive entrainment is rarely observed in clinically stable ATs and is more likely in ATs occurring immediately after termination of CAF by ablation\textsuperscript{11}.

\textbf{Mechanisms of Subsequent and Recurrent ATs}

The electrophysiological complexity of CAF is also reflected in the multiple and multifaceted types of recurrent ATs occurring late after ablation. Its incidence ranges from 4 to 40 \% after a single procedure for CAF\textsuperscript{2,3,18}. It is plausible that the mechanism of AT is a direct consequence of the greater amount and location of ablation applied during CAF ablation compared to ablation for shorter-lived AF. This is evidenced by the observation that AT occurring after circumferential PVI for paroxysmal AF are predominantly LA macro-reentry and rarely focal, whereas those occurring after CAF ablation involve several mechanisms including focal, macro- and micro-reentry\textsuperscript{2,3,9,11,15-17}. 
One observation in this study was that tachycardia cycle lengths did not depend on mechanism or, in case of reentrant ATs, on the diameter of the tachycardia circuit. Thus, the accurate identification of the AT mechanism can be challenging and usually requires detailed and sophisticated mapping, particularly in case of non-macro-reentrant AT that can be localized to virtually all aspects of the atria and thoracic veins. Although “common types” of left ATs are often easier to identify, its appropriate treatment with the achievement of bidirectionally blocked lines may be more demanding for the operator\textsuperscript{19,20}, whereas focal and microreentrant ATs usually requires discrete ablation at a critical spot\textsuperscript{12,21}.

**The Electrophysiological Genesis of Subsequent and Recurrent ATs**

The wealth of data describing different approaches to CAF ablation universally report that ATs are an inevitable consequence of ablation in a large subset of patients\textsuperscript{5,11,12,15-18,22}. Two mechanistic hypotheses have evolved to explain this phenomenon. The proarrhythmic hypothesis suggests ablation lesion and scar formation form the substrate for subsequent and recurrent AT, while on the other hand, it is hypothesized that successful ablative treatment of AF only “unmasks” the underlying culprit AT. While the proarrhythmic theory may be more intuitive and perhaps the more widely favored, there is growing evidence for a contributing role of AT in the AF process\textsuperscript{5,21,22}.

A recent study investigating a potential pathophysiological link between CAF and ATs using spectral analysis to characterize CAF and ATs, demonstrated that the frequency of subsequent ATs often overlapped with spectral components of the baseline AF periodogram\textsuperscript{5}. Also in keeping with the “unmasking” process is the observation that subsequent ATs also occur at areas that have not been targeted for any ablation previously\textsuperscript{21,22}.

In the present study, the majority of patients converted to relatively uncommon types of ATs after termination of the clinical AT. These ATs mainly constituted of small reentrant
circuits and were distributed throughout the atria, particularly in the LA. Although this observation may favor the proarrhythmic hypothesis, it is also plausible that the diseased substrate in CAF provides the milieu for such ATs which are clinically masked by a co-existing or consequential fibrillatory process. We therefore compared epidemiological parameters and the procedural data of the index CAF ablation of patients with a single AT (mainly common macroreentry) with patients who had multiple ATs (mainly localized reentry and focal). If the latter depend on a more diseased and progressed AF substrate (favoring the AF contributor hypothesis), one would expect more severe changes of echocardiographic parameters and a longer history of AF in this group. Conversely, if multiple ATs was the result of a more extended ablation (favoring the AF proarrhythmic hypothesis), this group should have been exposed to greater RF ablation time. We found no differences in epidemiological parameters or ablation time between groups and could not favor one hypothesis over another.

Regardless of cause, the appropriate treatment of AT is required for an arrhythmia-free follow-up. Whether all subsequent and/or induced ATs occurring after successful ablation of the clinical AT should be targeted for further ablation as part of “appropriate treatment” remains an open question. However, two important findings in the current study are in favor of treating all encountered ATs: first, patients who have had stepwise ablation for CAF are susceptible to multiple (rather than single) ATs, and secondly, targeting all ATs for ablation leads to very favorable results.

**Conclusions**

The majority of recurrent ATs occurring late after stepwise ablation of CAF have a reentrant mechanism including macroreentry and small localized circuits. Although widely distributed throughout the atria and thoracic veins, these recurrent ATs can be treated effectively by conventional mapping and ablation with excellent acute and long-term success
rates. Further studies are needed to extrapolate the electrophysiological and substrate basis of the cause of subsequent and recurrent ATs which would also enhance our understanding of the mechanisms operating in chronic atrial fibrillation.

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**Conflict of Interest Disclosures:** None

**References:**


Table 1

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<table>
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<tr>
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<tbody>
<tr>
<td>Age, years</td>
<td>63 ± 10</td>
</tr>
<tr>
<td>Female, n (%)</td>
<td>14 (23)</td>
</tr>
<tr>
<td>AF History, months</td>
<td>61 ± 29</td>
</tr>
<tr>
<td>LA Diameter, mm</td>
<td>48 ± 5</td>
</tr>
<tr>
<td>LVEF, %</td>
<td>58 ± 9</td>
</tr>
<tr>
<td>Hypertension, n (%)</td>
<td>51 (84)</td>
</tr>
<tr>
<td>Coronary Artery Disease, n (%)</td>
<td>9 (15)</td>
</tr>
<tr>
<td>Valvular Heart Disease, n (%)</td>
<td>5 (8)</td>
</tr>
<tr>
<td>Congestive Heart Failure, n (%)</td>
<td>4 (7)</td>
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</table>

Patients’ baseline characteristics.

AF = atrial fibrillation, LA = left atrium, LVEF = left ventricular ejection fraction.
Table 2

<table>
<thead>
<tr>
<th>Mechanism</th>
<th>Number (n, [%])</th>
<th>Mean CL (ms)</th>
<th>CL range (ms)</th>
<th>Mechanism</th>
<th>Number (n, [%])</th>
<th>Mean CL (ms)</th>
<th>CL Range (ms)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Roof Flutter</td>
<td>19 [43]</td>
<td>272 ± 35</td>
<td>220 - 360</td>
<td>Recovered PV</td>
<td>7 [41]</td>
<td>270 ± 42</td>
<td>220 - 320</td>
</tr>
<tr>
<td>Perimimal Flutter</td>
<td>15 [34]</td>
<td>278 ± 40</td>
<td>230 - 340</td>
<td>Coronary Sinus</td>
<td>4 [23]</td>
<td>300 ± 45</td>
<td>240 - 350</td>
</tr>
<tr>
<td>RA Septum</td>
<td>2 [12]</td>
<td>292 ± 50</td>
<td>220 – 350</td>
<td></td>
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</tbody>
</table>

Mechanisms, incidence and electrophysiological characteristics of clinical atrial tachycardias (AT). Of note, localized reentrant ATs are categorized as “Reentrant ATs”, while ATs presenting with electrophysiological features consistent with a focal mechanism are summarized as “Focal ATs”. 

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### Table 3

<table>
<thead>
<tr>
<th>Mechanism</th>
<th>Number (n, [%])</th>
<th>Mean CL (ms)</th>
<th>CL range (ms)</th>
<th>Mechanism</th>
<th>Number (n, [%])</th>
<th>Mean CL (ms)</th>
<th>CL Range (ms)</th>
</tr>
</thead>
</table>

Data of subsequent ATs to which the patients converted after termination of the clinical atrial tachycardias. CL = cycle length, CTI = cavotricuspid isthmus, LAA = left atrial appendage, LA = left atrium, CS = coronary sinus, RA = right atrium, SVC = superior vena cava.
Table 4

<table>
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<tr>
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<th>Patients with 1 AT</th>
<th>Patient &gt; 1 AT</th>
<th>p-value</th>
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<tbody>
<tr>
<td>Age, years</td>
<td>65 ± 10</td>
<td>62 ± 10</td>
<td>.329</td>
</tr>
<tr>
<td>LA Diameter, mm</td>
<td>49 ± 7</td>
<td>48 ± 4</td>
<td>.626</td>
</tr>
<tr>
<td>LVEF, %</td>
<td>60 ± 10</td>
<td>57 ± 9</td>
<td>.121</td>
</tr>
<tr>
<td>AF History, months</td>
<td>56 ± 32</td>
<td>63 ± 28</td>
<td>.175</td>
</tr>
<tr>
<td>Proc-Interval, months</td>
<td>10 ± 4</td>
<td>7 ± 4</td>
<td>.021</td>
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<tr>
<td>IP Number of RFC, n</td>
<td>64 ± 27</td>
<td>61 ± 12</td>
<td>.686</td>
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<tr>
<td>IP Duration, min</td>
<td>286 ± 85</td>
<td>265 ± 57</td>
<td>.743</td>
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<tr>
<td>IP Fluoro, min</td>
<td>81 ± 26</td>
<td>75 ± 13</td>
<td>.832</td>
</tr>
</tbody>
</table>

Statistical comparison of epidemiological characteristics and procedural data of the index procedure for long-lasting persistent atrial fibrillation between patients with a single atrial tachycardia (AT) and patients with multiple ATs. LA = left atrium, LVEF = left ventricular ejection fraction, AF = atrial fibrillation, Proc-Interval = interval between the index procedure for long-lasting persistent atrial fibrillation and the first redo procedure for atrial tachycardia, IP = index procedure for long-lasting persistent atrial fibrillation, * = p < 0.05.
Figure Legends:

**Figure 1:** Entrainment mapping of *a perimitral flutter* with the demonstration of the post-pacing response at the given pacing sites. AT CL = atrial tachycardia cycle length.

**Figure 2:** The tracings show an atrial tachycardia (AT) with a cycle length of 250 ms that is characterized by high-fractionated potentials covering almost the entire AT cycle length in the two bipoles of the mapping catheter which is placed at the anterior ridge of the left atrial appendage (LAA) (A). Of note, the local fractionated potentials do not have a low voltage and demonstrate rather normal (healthy) electrogram amplitudes. (B) Entrainment mapping at the anterior ridge of the LAA is reproducibly associated with a perfect return cycle. Importantly, the first fractionated potential after the last stimulus does not reflect the post-pacing interval but represents the last paced electrogram that occurs with a long local activation delay. (C) Results from entrainment mapping at the given pacing sites in the atria. (D) A single RF application at the presented site resulted in progressive prolongation of the AT cycle length and termination to sinus rhythm after 16 seconds.

**Figure 3:** Flowchart of the mapping protocol.

**Figure 4:** Atrial tachycardia of a patient with a bidirectionally blocked mitral isthmus line during the index procedure for CAF. The AT is characterized by negative inferior P waves and a positive P wave in V1. The posterior and superior aspect of the LIPV is activated passively with a Wenckebach-periodicity (LS 6/7 – LS 9/10). The anterior part of the vein (LS 1/2 – LS 5/6) shows 1:1 conduction, but is also passively activated as indicated by an earlier activation of the LAA far-field potential in relation to the PV potential. Isolation of the anterior aspect of the LIPV results in termination of the AT. LAA pacing performed after AT
termination confirmed isolation of the LIPV at the anterior aspect and the remaining anterior potential as a farfield potential originating from the LAA. Thus, along with a post-pacing interval of + 10 ms at the anterior aspect of the LIPV, the diagnosis of a peri-mitral flutter with the LIPV serving as the critical conduction gap could be confirmed. Notably, the posterior and superior part, which was activated with a functional conduction block, is still conducted but shows a long activation delay with the PV potentials activated at the time of the ventricles.

**Figure 5**: Flowchart of the course of the index procedure for atrial tachycardia (AT).
Step 1: PV Mapping / Re-Isolation

Step 2: Characterization of P-Wave

Step 3: Entrainment Mapping

Step 4: Activation Mapping

Re-Induction

SR → End

New AT → Ablation → Validation of Endpoint (Lines)
Characterization, Mapping and Catheter Ablation of Recurrent Atrial Tachycardias Following Stepwise Ablation of Long-Lasting Persistent Atrial Fibrillation

Thomas Rostock, Imke Drewitz, Daniel Steven, Boris A. Hoffmann, Tushar V. Salukhe, Karsten Bock, Helge Servatius, Muhammet Ali Aydin, Thomas Meinertz and Stephan Willems

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