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Treatment of macro-re-entrant atrial tachycardia based on electroanatomic mapping: identification and ablation of the mid-diastolic isthmus

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Aims This multicentre prospective study evaluated the ability of electroanatomic mapping (EAM) using a specific parameter setting to identify clearly the mid-diastolically activated isthmus (MDAI) and guide ablation of macro-re-entrant atrial tachycardia (MAT).

Methods and results Consecutive patients with MAT, different from typical isthmus-dependent atrial flutter, were enrolled. EAM was performed using a specific setting of the window of interest, calculated to identify the MDAI and guide ablation of this area. Sixty-five patients exhibiting 81 MATs (mean cycle length 308 ± 68 ms) were considered. Thirty-two (49.2%) had previous heart surgery. In 79 of 81 morphologies (97.5%), EAM reconstructed 95.9 ± 4.3% of the tachycardia circuit and identified the MDAI; 23 of the 79 morphologies (29.1%) were double-loop re-entry. Mapping of two morphologies was incomplete due to MAT termination after catheter bumping. In 73 of 79 mapped morphologies (92.4%), abolition of the MAT was obtained by 13.2 ± 12.4 applications. During the 14 ± 4 month follow-up, MAT recurred in 4 of the successfully treated patients (6.8%).

Conclusion EAM using a specific parameter setting proved highly effective at identifying the MDAI in MAT, even in patients with previous surgery and multiple re-entrant loops. Ablation of the MDAI yielded acute arrhythmia suppression with low rate of recurrence during follow-up.

Introduction

Unlike the majority of supraventricular tachycardias, tachycardias sustained by atypical atrial macro-re-entry still pose difficulties for diagnosis and successful ablation, especially when multiple re-entrant loops and/or relevant heart disease are present. In previous studies,[1–4] which mostly involved single centres, the best results were obtained using three-dimensional electroanatomic mapping combined with entrainment mapping to identify and transect an isthmus that was functionally or anatomically critical for re-entry. However, in some cases, electroanatomic activation mapping may provide ambiguous data,[5] and the feasibility of entrainment, when systematically attempted, can be limited.[1,6,7,8]

The current prospective study, named SWEET (Setting the Window of interest in macrorEEntrant Tachycardia), has been undertaken to evaluate the effectiveness of electroanatomic mapping, using a specific parameter setting to identify and ablate the mid-diastolically activated isthmus (MDAI) in macro-re-entrant atrial tachycardia (MAT), different from typical atrial flutter. We hypothesized that this method would simplify the procedure by clearly enhancing

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the MDAI of re-entry on the activation map and guiding ablation of this area. Multiple centres were involved in order to assess the broader applicability of this method and consistency of outcome.

Methods

Patient selection criteria

This was designed as a descriptive study involving 10 Italian centres. From 1 January 2004 to 31 December 2004, consecutive patients with recurrent, drug-refractory, sustained MAT showing stable tachycardia cycle length (TCL) were enrolled. Patients were included irrespective of their age, sex, presence of structural heart disease, and prior cardiac surgery. Both spontaneous and induced MAT morphologies were considered. Stable TCL was defined as TCL with beat-to-beat variations not exceeding 10% and 30 ms. Assessment of the macro-re-entrant nature of the tachycardia was based on commonly used electrophysiologic criteria.[1,3] Only patients exhibiting a single-arrhythmia morphology with a surface electrocardiographic pattern of typical or reverse atrial flutter, in whom a peritricuspid isthmus-dependent circuit was confirmed by intracavitary conventional criteria, were excluded, since ablation strategy is obvious in these cases. Patients with typical isthmus-dependant atrial flutter associated with atypical re-entry forms were included, but mapping and ablation data of the typical forms were excluded from this analysis to avoid the bias of an increased mapping and ablation success rate, easily obtained in typical isthmus-dependent atrial flutter. Other exclusion criteria were (i) evidence of intracavitary thrombus detected by transoesophageal echocardiography and (ii) any comorbidity that rendered anticoagulant and heparin administration impossible before, during, and after the procedure. The study was approved by each centre's institutional review board, and all patients gave written informed consent.

Electroanatomic mapping

The Carto system (Biosense-Webster Inc., Diamond Barr, CA, USA) was used, with a coronary sinus atriogram as reference signal. In this system, the duration of the window of interest corresponds to the interval of the arrhythmia cycle considered for activation and voltage mapping. For each MAT morphology, the backward and forward intervals of the window of interest (*Figure 1*) were calculated according to the following formulas:

$$\text{Backward interval} = \frac{\text{TCL} - \text{DUR}^{\text{PW}}}{2} + \text{Interval}^{\text{PWonset-ref}}$$

$$\text{Forward interval} = (\text{TCL} - \text{Backward interval}) \times 0.90,$$

where DUR^{PW} is the duration of the surface P-wave during MAT (interval 'a' in *Figure 1*), and $\text{Interval}^{\text{PWonset-ref}}$ is the interval between the onset of the P-wave and the reference signal (interval 'b' in *Figure 1*), which has a negative value when the reference signal precedes the P-wave onset. The P-wave onset and duration were measured on the 12 leads synchronously displayed at a sweep speed of 100 mm/s, as shown in *Figure 2*. Using this formula, the onset of the window of interest is set in mid-diastole, and its duration spans between 90 and 95% of the TCL. As a consequence, in the colour-coded activation map, red and orange identify mid- and late-diastolic activations, respectively, and dark blue and purple identify early- and mid-diastolic activations, respectively. The remaining colours identify areas of systolic activation. The interface between the red area and the purple area identifies the MDAI of the re-entrant circuit. Since clear identification of the surface P-wave is the key point when using this method, carotid sinus massage or intravenous adenosine bolus was used to obtain a temporary atrioventricular conduction block.

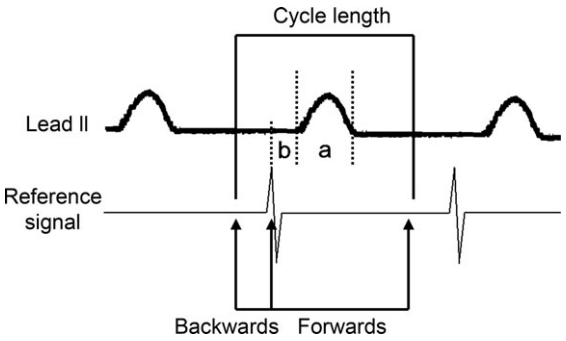

Figure 1 Method for calculating the backward and forward intervals of the window of interest during MAT. The top tracing shows the P-wave on lead II, whereas the second tracing is a schematic representation of the reference signal. Interval 'a' is the duration of the P-wave during tachycardia, and interval 'b' is measured from the P-wave onset to the reference signal.

Mapping was commenced in the right atrium and was continued in the left if a right-sided circuit was excluded. Mapping was continued until a complete electroanatomic reconstruction was obtained, and the interval between the earliest and latest activated sites was $\geq$90% of the TCL. In case of multicomponent or fragmented potentials, the first sharp deflection was annotated. Signals inscribed in the part of the tachycardia cycle outside of the limits of the window of interest (that spans 90–95% of the TCL) were annotated with respect to the reference signal as early if the first sharp deflection preceded the window of interest limits or late if it followed. Regions with no signal distinguishable from baseline noise (0.05 mV) were defined as electrically silent and were displayed in grey. Sites with double potentials separated by $\geq$50 ms, indicative of conduction block with activation detour, were tagged with blue dots. After completion of electroanatomic mapping, the MDAI was identified; its extension and the value of bipolar voltage were measured. Conduction velocity across the MDAI was also calculated and compared with the conduction velocity of three segments on the outer loop, in the direction of the propagating wavefront. To better identify the course of re-entry, especially when multiple re-entrant loops were suspected, the propagation map was carefully analysed. During mapping, if the TCL became prolonged or the MAT was interrupted and no longer inducible due to catheter bumping in a critical area, point acquisition was terminated. If mapping was insufficient to precisely define the course of re-entry and the MDAI, the morphology was classified as incompletely mapped.

Validation by concealed entrainment

Entrainment was used to validate the mid-diastolic isthmus identified by electroanatomic mapping as the critical area of re-entry. Tachycardia was entrained at 90% of its cycle length. Concealed entrainment with a difference of $<$10 ms between the TCL and the post-pacing interval confirmed a critical protected isthmus. If no capture occurred at 10 mA output, the catheter was repositioned at at least three different sites in the MDAI. In the case of discordance between entrainment mapping and electroanatomic mapping as to the definition of the critical area, extensive entrainment mapping had to be performed to redefine the re-entrant circuit. Entrainment validation was amended when any of the following events related to electrical stimulation was considered likely: (i) degeneration into atrial fibrillation (especially in morphologies with TCL $<$ 220–240 ms), (ii) conversion into another morphology, or (iii) termination of arrhythmia.

Ablation strategy

Ablation was aimed at the MDAI, with linear ablation of the whole extension of the MDAI. If MAT terminated during radiofrequency

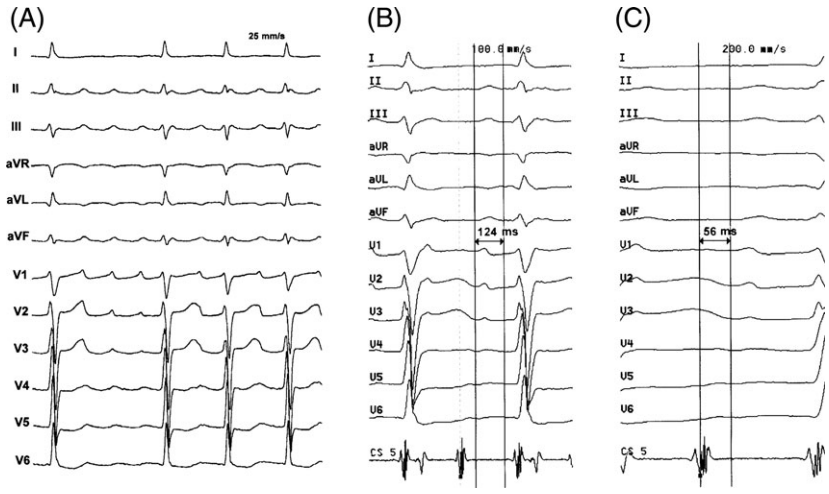

Figure 2 Surface 12-lead ECG of a MAT and an example of measurement of intervals 'a' and 'b'. (*A*) The 12 leads are synchronously displayed at 25 mm/s, showing an atypical P-wave pattern with a cycle length of 254 ms. (*B*) Interval 'a' is calculated as the maximum P-wave duration in all leads (124 ms) at a sweep speed of 100 mm/s. This sweep speed may represent an optimal compromise between lower and higher speeds, at which definition of the surface P-wave duration may be more difficult or less accurate. (*C*) Keeping the first caliper on the P-wave onset and moving the second caliper to the intracavitary reference, interval 'b' is calculated after sweep speed has been increased to 200 mm/s to better evaluate the intracavitary signal.

energy delivery, ablation was continued until disappearance of electrical signals and/or development of an activation pattern consistent with a continuous line of block in the target area during sinus or paced rhythm. In case of double-loop re-entry, the propagation sequence was carefully analysed to assess whether both loops shared the same MDAI. In case of shared MDAI, ablation was performed at this site, with the aim of abolishing both loops. When mapping was incomplete, the ablation strategy was developed by combining activation and voltage mapping data.

According to operators' preference at each centre, a 4 mm, 8 mm, or irrigated-tip catheter (Navistar, Biosense-Webster Inc., Diamond Barr, CA, USA) was used. The use of non-irrigated-tip catheters in the left atrium and in enlarged atrial chambers was strongly discouraged. When 4 mm or 8 mm tip catheters were chosen, the preset maximum temperature was 65°C and the maximum power was 65 and 80 W, respectively, unless contraindicated by the location of the ablation site. Ablation with irrigated-tip catheter was performed in temperature control mode, with a maximum preset temperature of 43°C and a maximum preset power of up to 50 W. The flow rate was set at 20 mL/s for power settings below 30 W, and 30 mL/s for power settings between 30 and 50 W. The maximum application duration for all catheters was preset at 60 s.

After arrhythmia interruption and ablation completion, induction attempts by programmed electrical stimulation with multiple extrastimuli and bursts were performed. If an organized arrhythmia was not inducible 30 min following termination of ablation, the procedure was terminated and considered acutely successful. If a new morphology with stable TCL was reproducibly inducible or converted from the index morphology during ablation, the arrhythmia was mapped and targeted for ablation, regardless of the fact that it was non-clinical. If MAT was not suppressed by ablation, accurate remapping was performed in the case of significant TCL prolongation to rule out even minor variations in the re-entrant circuit. Then, the procedure was considered unsuccessful and sinus rhythm restored by DC-shock.

Follow-up

Patients were evaluated at follow-up visits and by Holter monitoring for at least 6 months, even if asymptomatic. Patients with symptom recurrence were invited to report to the physician in charge at each centre. If concomitant atrial fibrillation or ventricular arrhythmias were present, antiarrhythmic drugs that had been ineffective in preventing MAT recurrence were continued. A second ablation procedure was offered to the patient if MAT recurred.

Table 1 Type of previous heart surgery

Type of surgical intervention	Number of patients
Repair of congenital heart disease	14
Mitral valvuloplasty	6
Mitral valvuloplasty and surgical pulmonary vein ablation	1
Mitral valve replacement	3
Aortic valve replacement	1
Mitral and aortic valve replacement	5
Myocardial revascularization	1
Cardiac transplantation	1
Total	32

Statistics

Continuous variables are expressed as mean $\pm$ SD, whereas categorical variables are expressed in percent. The Friedman ANOVA test was used to compare conduction velocities. Correlations were computed by means of Spearman's test. A one-sample *z*-test was used to analyse the difference between the percent value of the backward interval of the window of interest and the 50% value of the TCL. Mann–Whitney *U* test was used to compare the MDAI extension between patients with successful and those with unsuccessful ablation.

Results

Patient characteristics

Sixty-five patients participated in the study; 41 were male. The mean age was 56.7 $\pm$ 16.7 years (range 9–83 years). Twenty patients (30.8%) showed no evidence of structural heart disease. The remaining 45 (69.2%) showed congenital or acquired heart disease as follows: congenital heart disease (16), mitral valve disease alone (11), aortic valve

disease alone (1), mitral and aortic valve disease (5), hypertensive heart disease (6), ischaemic heart disease (3), dilated cardiomyopathy (2), and previous myocarditis (1). Thirty-two patients (49.2%) had previously undergone heart surgery with single or multiple interventions; the types of interventions are reported in *Table 1*.

This patient population showed 81 MAT morphologies with mean TCL of 308 $\pm$ 68 ms (range 200–515 ms). Other coexisting arrhythmias, which, following protocol, were not considered in the present analysis were atrial fibrillation (10 patients), typical isthmus-dependent atrial flutter (9 patients), coexistence of these two arrhythmias (5 patients), and focal atrial tachycardia (4 patients). Of the 15 patients with atrial fibrillation, 6 had previously undergone ablation in the left atrium; in the others, the arrhythmia was prevented by antiarrhythmic drugs. All of the patients with typical isthmus-dependent atrial flutter underwent ablation of the cavo-tricuspid isthmus in either the present procedure or a previous one. In patients with focal atrial tachycardia, this arrhythmia was reproducibly inducible after ablation of the index MAT. In all these cases, electroanatomic mapping using this setting of the window of interest identified a centrifugally spreading activation pattern from the site of earliest activation, enabling correct diagnosis and successful ablation.

Mapping data

In all the MATs, the surface P-wave morphology was clearly identified, and the window of interest could be correctly set. The absolute value of the backward interval ranged from 3 to 280 ms. It represented a highly variable percentage of the TCL, ranging from 1–80%. The percent value of the backward interval significantly ($P < 0.001$) differed from the 50% value of the TCL. In the right and left atria, a mean of 100 $\pm$ 56 (range 44–354) and 101 $\pm$ 63 (range 50–305) mapping sites were acquired, respectively.

In 79 of 81 MAT morphologies (97.5%), electroanatomic mapping was completed. Activation mapping covered 95.9 $\pm$ 4.3% (range 90–100%) of the TCL. For all 79 morphologies, the electroanatomic map visualized the entire arrhythmia circuit, showing a typical 'head-meets-tail' pattern and clearly identifying the MDAI between anatomic boundaries, lines of double potentials, and/or electrically silent areas (*Figure 3*). In 23 of 79 (29.1%) morphologies, electroanatomic mapping identified a double-loop re-entry, with both loops sharing the same MDAI, configured in a typical 'figure eight' pattern (*Figures 4* and *5*). The MAT circuit was located in the right and left atria in 47 and 32 morphologies, respectively (*Table 2*). The MDAI extension ranged from 5 to 69 mm with a mean of 22.7 $\pm$ 14.2 mm. *Figures 5–7* show examples of the variable location and extension of the MDAI. In the MDAI, bipolar voltage was 0.31 $\pm$ 0.32 mV on average (range 0.06–1.7 mV); 82.4% of the sites had a voltage $<$ 0.5 mV, 11.8% between 0.5 and 1 mV, 5.8% $>$1 mV. Conduction velocity across the MDAI was low (27 $\pm$ 13 cm/s) and significantly ($P < 0.00001$) differed from the ones measured along the outer loop (85 $\pm$ 33, 76 $\pm$ 18, and 82 $\pm$ 33 cm/s).

In 22 of 79 MAT morphologies (27.8%), entrainment stimulation was not performed owing to the likelihood of degeneration into atrial fibrillation (16 morphologies) or development of unstable TCL after positioning the mapping catheter on the MDAI (6 morphologies). In the remaining 57 morphologies (72.2%), entrainment stimulation was attempted in the MDAI. In 40 of these 57, it resulted in concealed entrainment, with post-pacing interval equal to the TCL; in 12, stimulation did not result in capture; and in 5, it terminated the arrhythmia, which was subsequently re-inducible.

In 2 of 81 morphologies (2.5%) in 2 centres, mapping was incomplete (77 and 81% of TCL, respectively) due to termination and inducibility prevention by catheter bumping.

Ablation data

A 4 mm tip, 8 mm tip, and irrigated-tip catheters were used in 7, 4, and 54 patients, respectively. In the two morphologies terminated by catheter bumping, ablation was performed in sinus rhythm, and no arrhythmia was

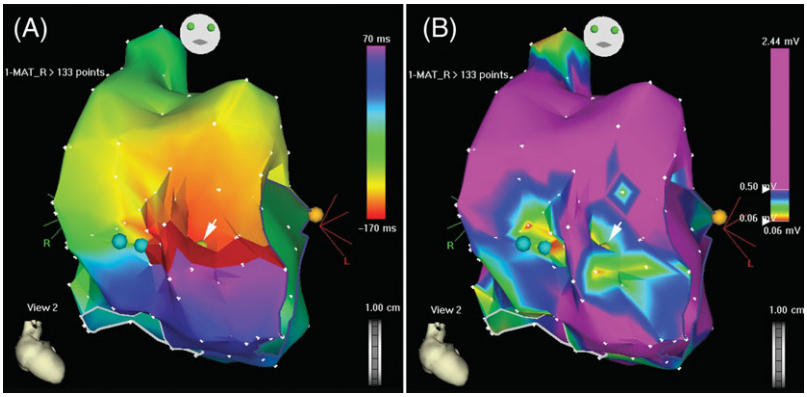

Figure 3 Electroanatomic mapping of the right atrium during MAT with TCL of 240 ms (12-lead ECG in *Figure 8A*). In this as well as in the following figures, map view is indicated by the heart icon in the lower left corner. On the activation map, a colour-coded scale from red to purple represents earliest-to-latest activation, whereas on the bipolar voltage map, the scale indicates low-to-preserved voltage. Blue dots indicate a line of double potentials, whereas yellow dots marked by an arrow show the site where concealed entrainment with a post-pacing interval equal to the TCL was obtained. (*A*) In the activation map, the entire TCL is reconstructed with an evident 'head-meets-tail' pattern and an MDAI (enhanced by the red band) between a double-potential line and the tricuspid annulus. (*B*) The bipolar voltage map shows predominantly preserved voltage, with the exception of the area of the MDAI, which exhibits minimal voltage amplitude with only a narrow band of preserved voltage, close to the site where concealed entrainment was obtained.

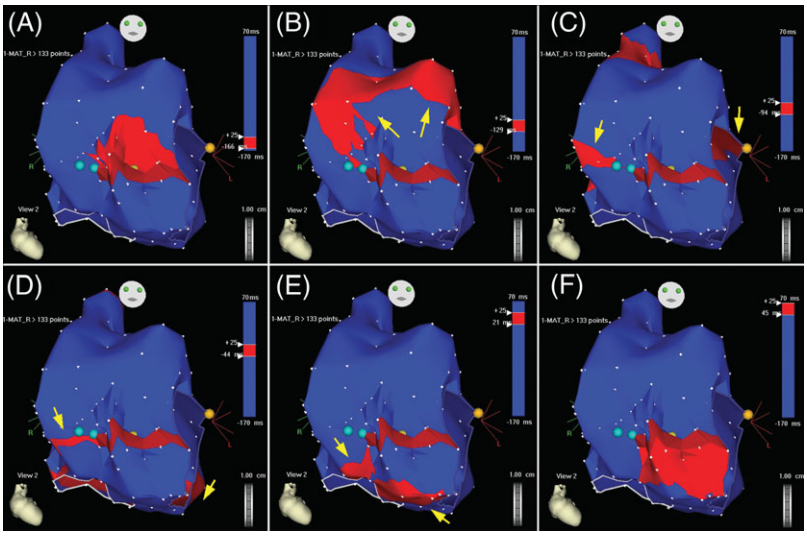

Figure 4 Sequential frames of a propagation map of the tachycardia shown in *Figure 3*. The propagation wavefronts are represented by the red bands, and the timing in the tachycardia cycle is indicated by the scale on the right-hand side of each frame. It is evident that the tachycardia is sustained by two loops, both sharing the same MDAI in a typical 'figure eight' pattern. After exiting the MDAI (*A*), the propagation wavefronts separate (*B*), propagate clockwise around the tricuspid annulus and counter-clockwise around the atriotomy (*C*–*E*), respectively, and collide at the entrance of the MDAI (*F*). Ablation aimed at the common isthmus abolished both loops and terminated the tachycardia, as reported in *Figure 8B*.

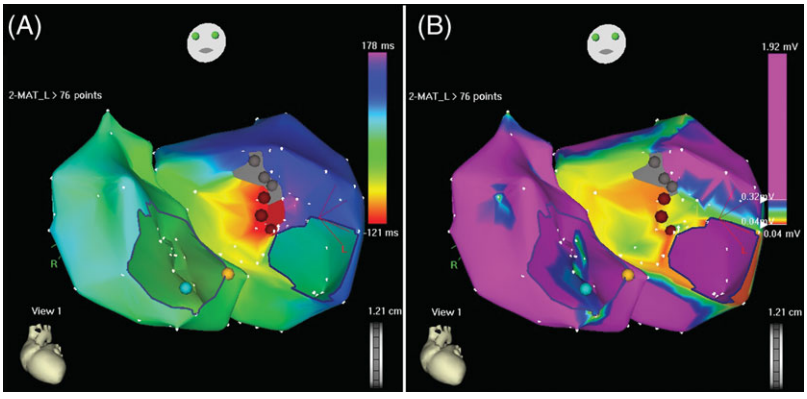

Figure 5 Electroanatomic mapping of both atria during MAT with TCL of 300 ms in a patient with dilated cardiomyopathy. In this and in the following figures, red dots indicate ablation sites and grey dots electrically silent areas. (*A*) In the activation map, the MDAI is a narrow channel in the anterior wall of the left atrium, between an area of scar and the mitral annulus. Equal colour distribution counter-clockwise around the mitral annulus and clockwise around the scar indicates, as in *Figure 3A*, two re-entrant loops with a shared MDAI. Ablation of this area easily abolished the tachycardia. (*B*) The voltage map shows very low voltage in the MDAI and anterior left atrial wall, with relatively preserved voltage in the remaining left atrium and right atrium.

subsequently inducible. Among the remaining 79 morphologies, 73 (92.4%) were abolished by radiofrequency energy delivery at the MDAI (*Figure 8*). In 6 of these 73 morphologies (8.2%), following MDAI ablation, the index morphology converted into another MAT without an intervening pause. Mapping of the second morphology identified another MDAI elsewhere located, and its ablation resulted in termination of arrhythmia, with no inducible arrhythmia at the end of the procedure. The mean number of radiofrequency energy applications was 13.2 $\pm$ 12.4, median 10, range 3–45. The number of applications significantly correlated to the isthmus extension ($r = 0.64$; $P < 0.05$); the *r*-value increased to 0.69 ($P < 0.05$) when only irrigated-tip ablation was considered.

In the remaining 6 of 79 MATs (7.6%), in 6 patients in 4 centres, arrhythmia could not be terminated by ablation. Irrigated-tip and 4 mm tip catheters were used for 5 and 1 of these patients, respectively. Five of these patients had associated heart disease and 4 had previously undergone open-heart surgery. For all six morphologies, the critical role of isthmus identified by electroanatomic mapping was confirmed by entrainment mapping. The average extension of the MDAI was significantly longer (40.6 $\pm$ 15.2 mm, range 20–56 mm) than that for the patients with successful ablation (21.7 $\pm$ 13.5 mm, range 5–69 mm; *P*-value = 0.032).

During radiofrequency energy delivery on the left aspect of the atrial septum, transient mild prolongation of the A-H interval in sinus rhythm was observed in one patient, with prompt resumption of baseline conduction after application discontinuation. One patient required surgical repair of a small femoral arterio-venous fistula. No other complications were reported.

Follow-up

During a mean 14±4 month follow-up, 7 of the 59 patients successfully treated (11.8%) experienced arrhythmia recurrence, whereas 4 patients of the 6 unsuccessfully treated (66.6%) had recurrence of the same MAT. Among successfully treated patients experiencing recurrence, 3 (5%) had recurrence of atrial fibrillation responsive to antiarrhythmic drug therapy; the remaining 4 (6.8%) had recurrence of the previously treated MAT (1 patient) or another MAT (3 patients) morphology. In the first patient, a second procedure showed the same arrhythmia circuit and MDAI, previously ablated by a 4 mm tip catheter. In the other three cases, the tachycardia circuit and MDAI differed from the index MAT. Irrigated-tip ablation was successful in abolishing arrhythmias in all four patients.

Discussion

Main findings

This study prospectively evaluates the efficacy of an approach based on electroanatomic mapping to identify and target the MDAI in consecutive MAT patients with and without structural heart disease and previous surgery. Importantly, typical isthmus-dependent atrial flutter was excluded, since its ablation strategy is obvious. This method proved highly effective in reconstructing the whole arrhythmia circuit and unambiguously identifying the MDAI, whose location and extension may be quite variable both in the right and left atria. A single-ablation procedure aimed at the MDAI was successful in the vast majority of cases at abolishing and permanently suppressing the tachycardia circuit. Previous single-centre studies[1,3,7] reported good results with ablation of atypical macro-re-entry using electroanatomic mapping and entrainment mapping with a primarily anatomically based approach so that both systolically and diastolically activated isthmi could be targeted for ablation. Our study shows comparable results in a larger, multicentre series, with a trend towards a higher rate of definition of the re-entry circuit and its target area and less need for repeated procedures, likely due also to the extensive use of irrigated-tip catheters (in 83% of the patients).

Table 2 Location of MDAI in the right and left atria

Location	Number of morphologies
Right atrium	47
Anterolateral wall	30
Posterior wall	4
High right atrium–SVC	4
Medial CTI	3
Septum	6
Left atrium	32
Anterior wall, roof	14
Area around left PVs	8
Area around right PVs	3
LIPV–MA isthmus	4
Left septum	3
Total	79

CTI, cavo-tricuspid isthmus; LIPV, left inferior pulmonary vein; MA, mitral annulus; PVs, pulmonary veins; SVC, superior vena cava.

Advantages of this approach

Although this method requires the specific measurement of intervals for each morphology following the identification of the surface P-wave, the window of interest was correctly set in all the MAT morphologies in this patient series. The value of the backward interval of the window of interest, expressed as a percentage of the TCL, was highly variable and significantly different from the 50% value of the TCL, reflecting variability in the activation time of the reference signal during MAT. This implies that an empirical definition of the window of interest, such as 50% of the TCL for both backward and forward intervals, may not be the best method to chronologically analyse the course of re-entry and identify and target the MDAI, using electroanatomic mapping. Conversely, the setting of the window of interest used in this study allows reconstruction of an unambiguous activation map, in which each colour identifies a given activation time, regardless of the TCL and re-entry course. This results in a clear 'head-meets-tail' pattern with precise identification of the MDAI, which may present a variable extension, but invariably shows low amplitude potentials and slow

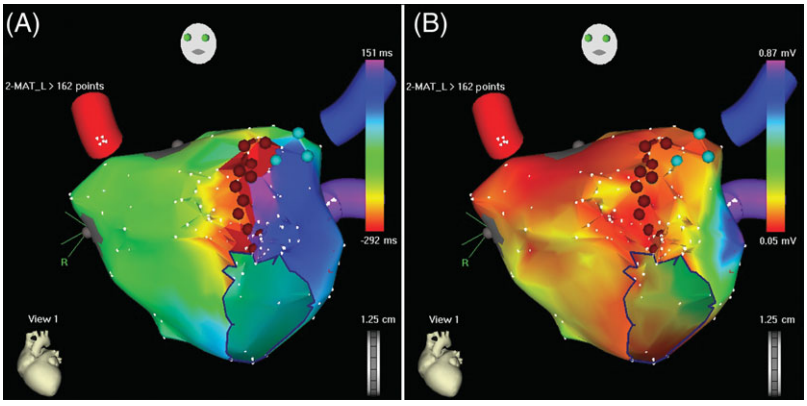

Figure 6 Electroanatomic mapping of the left atrium during MAT with TCL of 450 ms in a patient with previous surgical mitral valvuloplasty. Tubes indicate left superior (blue), inferior (purple), and right superior (red) pulmonary veins. (*A*) The activation map shows a clear 'head-meets-tail' pattern, with the MDAI localized between the left superior pulmonary vein and the mitral annulus. The extension of this area is 37 mm. (*B*) Voltage mapping shows diffuse, very low voltage, especially in the anterior wall.

conduction. This characterizes the MDAI as the weakest part of the re-entrant circuit, and, therefore, a potentially ideal site for ablation. Moreover, if not ablated, the MDAI may serve as slow conduction area for other MATs, once the index morphology has been abolished by resecting an isthmus, anatomically critical for re-entry. Finally, since in the present study there was a correlation between the number of applications required for isthmus ablation and isthmus extension, it can be generally assumed that difficulty ablating a given morphology can be estimated after mapping and before the ablation by measuring the extension of the MDAI. In our series, unsuccessfully treated morphologies showed a significantly longer extension of the MDAI.

The second advantage of this approach is that the identification of the ablation target is not strictly dependent on entrainment mapping. In fact, even in the cases for which entrainment mapping was not possible or was not performed, we found that using electroanatomic mapping with this setting of the window of interest, a protected isthmus of mid-diastolic activation could be identified and successfully ablated with tachycardia termination in the vast majority of the cases. In any case, concealed entrainment confirmed the critical role of the MDAI, identified as the target by electroanatomic mapping, in all the cases in which it could be tested (51% of the morphologies). In the remaining morphologies, electrical stimulation resulted in no capture (15%) or arrhythmia termination (6%), or was

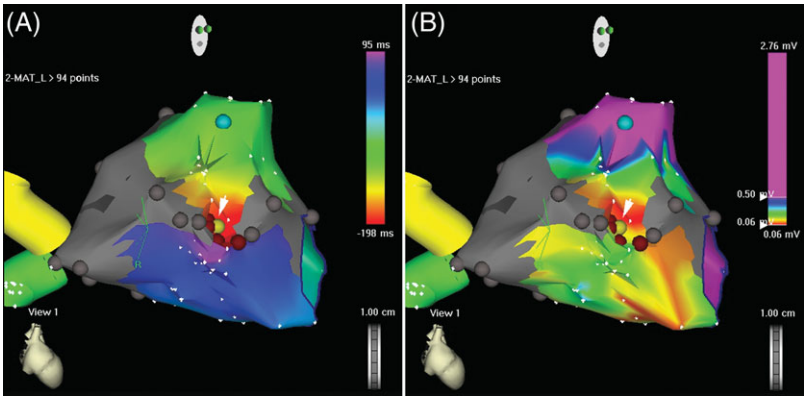

Figure 7 Electroanatomic mapping of the left atrium during MAT with TCL of 300 ms in a patient with prior catheter ablation in the left atrium for atrial fibrillation. Tubes indicate right pulmonary veins. (*A*) In the activation map, the MDAI corresponds to the conduction gap in a linear lesion (grey dots) between the mitral annulus and the right superior vein. Although this area was difficult to reach, only four applications of radiofrequency energy were needed to permanently abolish the MAT. (*B*) Voltage mapping shows very low amplitude in the MDAI.

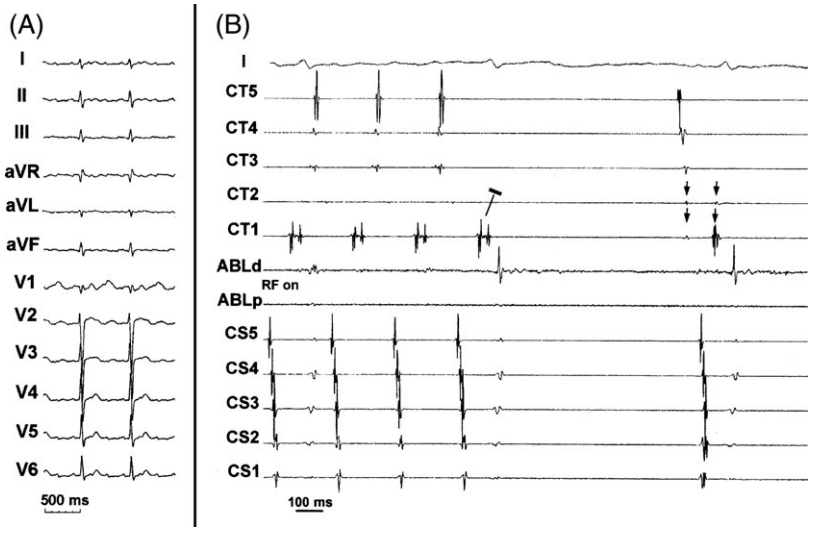

Figure 8 (*A*) Twelve-lead ECG of the MAT shown in *Figures 3* and *4*. P-wave is positive in the inferior, lateral, and V2–V6 leads and negative in V1. (*B*) Intracavitary signals upon tachycardia termination by ablation. From top to bottom, lead I, bipolar signals from the crista terminalis catheter (CT5–CT1, from the upper to the lower part), from the distal and proximal electrode pairs of the ablation catheter (ABLd and ABLp), and from the coronary sinus catheter (CS5–CS1, from proximal to distal) are displayed. During tachycardia, the activation sequence is from the lower to the upper part of the crista terminalis and from proximal to distal coronary sinus. After sequential radiofrequency energy application from posterior to anterior along the MDAI (*Figure 3*), the ablation catheter is positioned close to the tricuspid annulus (ventricular deflection is evident in ABLd) to ablate the remaining area of MDAI. During energy application, re-entry is terminated in the lower part of the crista terminalis (blocking propagation from CT1 to CT2–5), and a line of transverse conduction block in the crista terminalis is confirmed by double potentials separated by >100 ms (indicated by arrows) in CT4 and CT5 in the first sinus beat.

not performed (28%). This limitation of entrainment mapping for MAT is in agreement with previous studies,[1,6,7,8] in which, owing to arrhythmia degeneration or termination, the technique could not be systematically used to electrophysiologically define the critical isthmus.

The third advantage of this approach is represented by the finding that in double-loop, re-entry identification and ablation of an MDAI shared by both loops led to arrhythmia abolition with a single ablation line. This strategy appears more parsimonious, sparing ablation and mapping time compared with separate targeting of each loop. In fact, typical isthmus-dependent atrial flutter, in half of the cases[9] archetypal of dual-loop re-entry (around the tricuspid annulus and the inferior cava, respectively) with a shared MDAI, is targeted only at the cavo-tricuspid isthmus. In our series as in a previous study,[8] double-loop re-entry accounts roughly for one-third of all morphologies, although the reported prevalence of multiple re-entrant loops has varied from 6 to 74% in different studies.[3,4,7] Although the case of two independent loops, each one with its own MDAI, cannot be excluded, our data suggest that double-loop re-entry with a shared MDAI is by far more common.

Clinical implications

This data suggest that in MAT, an approach based on identification and target of the MDAI simplifies mapping and ablation of atrial atypical macro-re-entrant circuit, resulting in a favourable outcome after a single procedure, even in cases with relevant heart disease and prior surgery.

The prevalence of concomitant atrial fibrillation in our patient series was $\sim$25%, comparable with that observed in other studies.[3,4] Since atrial fibrillation was responsive to prior ablation or antiarrhythmic drug therapy, the clinical benefit of MAT ablation persists.

Finally, although both previous studies and this one reported findings from a limited patient series, it can be presumed from current clinical practice that atypical MAT is not as rare as it might seem. Improved clinical observation, extensive use of antiarrhythmic drugs, and left atrial ablation for atrial fibrillation could together increase the incidence of documented MAT and with it the need for treatment.

Limitations

Electroanatomic mapping requires sequential sampling of a considerable number of sites. During the course of the procedure, the arrhythmia might change or become unstable. Nevertheless, occurrence of such modifications was observed in very few cases in our study and with limited consequence. Occurrence can be further limited by making mapping of the MDAI the last step of chamber mapping. Sequential electroanatomic mapping is not possible in MAT with irregular cycle, but this type of arrhythmia was not observed in our patient series, even as an induced morphology after ablation of the index MAT.

In this study, the extensive use of an irrigated-tip catheter may have contributed to successful ablation and the good clinical outcome. In fact, the use of irrigated-tip catheters proved highly effective for ablation of MAT in congenital heart disease patients.[10] However, the prevalence of congenital heart disease patients in our series is limited (24.6%), and irrigated ablation may be of less dramatic benefit than expected when evaluated in a prospective randomized way.[11]

The mean MAT cycle length in our series, as in previous studies,[1,3,7] was $>$300 ms (308 $\pm$ 68 ms), and the lower value (200 ms) of the range of the TCL was encountered only in one morphology. Therefore, difficulties in calculating intervals 'a' and 'b' and therefore in setting the window of interest in arrhythmias with a very short cycle length and atrial activation configuring in a continuous waving pattern on surface ECG in multiple leads cannot be excluded.

The present study does not assess the superiority of the presented method to the previously described method on the basis of electroanatomic mapping using an arbitrary setting of the window of interest, entrainment mapping, and ablation of an anatomically or functionally defined isthmus. To this purpose, a randomized study should be undertaken. The present study was designed as a descriptive study to evaluate in a multicentre way the applicability and the clinical impact of the presented method, expected to simplify the procedure in MAT patients, mainly because (i) re-entry is invariably targeted at the MDAI, the weakest part of the circuit in the vast majority of the cases on the basis of the findings in this area of delayed conduction velocity and low amplitude potentials observed in our series, (ii) the use of entrainment mapping is not strictly necessary to identify the course of re-entry and the target area, and (iii) dual-loop re-entry is targeted at the shared MDAI with a single ablation line.

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Appendix

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