

Left Atrial Tachycardia After Circumferential Pulmonary Vein Ablation for Atrial Fibrillation

Electroanatomic Characterization and Treatment

Cézar E. Mesas, MD, Carlo Pappone, MD, PhD,* Christopher C. E. Lang, MB, CHB, Filippo Gugliotta, BENG, Takeshi Tomita, MD, PhD, Gabriele Vicedomini, MD, Simone Sala, MD, Gabriele Paglino, MD, Simone Gulletta, MD, Amedeo Ferro, MD, Vincenzo Santinelli, MD

Milan, Italy

OBJECTIVES	The purpose of this study was to evaluate the electroanatomic characteristics of left atrial tachycardia (AT) in a series of patients who underwent circumferential pulmonary vein ablation (CPVA) and to describe the ablation strategy and clinical outcome.
BACKGROUND	Circumferential pulmonary vein ablation is an effective treatment for atrial fibrillation. A potential midterm complication is the development of left AT. There are only isolated reports describing mapping and ablation of such arrhythmias.
METHODS	Thirteen patients (age 57.4 ± 8.9 years, five female) underwent mapping and ablation of 14 left ATs via an electroanatomic mapping system a mean of 2.6 ± 1.6 months after CPVA.
RESULTS	Three patients were characterized as having focal AT (cycle length: 266 ± 35.9 ms). Of 11 macro-re-entrant tachycardias studied in the remaining 10 patients (cycle length: 275 ± 75 ms), 5 showed single-loop and 6 dual-loop circuits. Re-entrant circuits used the mitral isthmus, the posterior wall, or gaps on previous encircling lines. Such gaps and all three foci occurred anterior to the left superior pulmonary vein or at the septal aspect of the right pulmonary veins. Thirteen of 14 tachycardias (93%) were successfully ablated.
CONCLUSIONS	Left AT after CPVA can be due to a macro-re-entrant or focal mechanism. Re-entry occurs most commonly across the mitral isthmus, the posterior wall, or gaps on previous ablation lines. Such gaps and foci occur most commonly at the anterior aspect of the left superior pulmonary vein and at the septal aspect of the right pulmonary veins. These arrhythmias can be successfully mapped and ablated with an electroanatomic mapping system. (J Am Coll Cardiol 2004;44:1071-9) © 2004 by the American College of Cardiology Foundation

Catheter ablation of the left atrium (LA) to encircle the pulmonary veins (PVs) is a highly effective treatment for patients with atrial fibrillation (AF) (1). A possible midterm complication is the development of left atrial tachycardia (AT), mostly related to re-entry around incomplete lesions or involving the mitral isthmus (2). With an increasing number of centers performing AF ablation, a higher incidence of this complication can be anticipated. However, there are only isolated reports describing mapping and ablation of such arrhythmias (3,4). In the present study, we characterize the electrophysiologic and electroanatomic features of left AT in a consecutive series of patients with previous circumferential pulmonary vein ablation (CPVA). We also describe the ablation strategy and clinical outcome.

METHODS

Patient population. Between October 2003 and January 2004, 30 patients underwent a repeat procedure after previous ablation for AF. Of these, 13 (43.3%) were determined as having left AT. Data on the index AF ablation for these patients were obtained. In the period between the first and last index procedure for these 13 patients, we performed

CPVA in 276 patients, giving an approximate incidence of AT after CPVA of 4.7%. The details of CPVA have been described elsewhere (1,5,6). In brief, three-dimensional maps of the LA were reconstructed through a transeptal route with an electroanatomic mapping system (CARTO, Biosense-Webster, Diamond Bar, California). Radiofrequency (RF) energy was applied in a linear fashion, aiming to encircle the pulmonary veins. At each point, a maximum power of 100 W was used for 15 to 30 s with an 8-mm tipped ablation catheter (Navistar, Biosense-Webster) to achieve a temperature of 50°C to 65°C. Additional lines were performed in the posterior LA in most patients using the same RF generator settings, connecting the encircling lines of the left and right pulmonary veins, and an ablation line was placed at the mitral isthmus between the mitral annulus and the left pulmonary vein encircling line to prevent LA flutter. In this study population, additional lines were performed in the posterior LA in all but one patient, and an ablation line was placed at the mitral isthmus in all patients. These lines were designed to prevent LA flutter (1,6) and were performed with the same power and temperature settings of the encircling lines. The continuity of the lines was assessed by demonstrating the abatement of local electrogram amplitude by $\geq 80\%$, double potentials and delay, or reversal in regional activation during coronary sinus pacing.

The mean time interval between CPVA and the occur-

From the Department of Cardiology, Arrhythmology Section, San Raffaele University Hospital, Milan, Italy. Dr. Mesas is the recipient of a research fellowship grant from the CAPES Foundation, a Brazilian Government research institute.

Manuscript received April 2, 2004; revised manuscript received May 4, 2004, accepted May 25, 2004.

Abbreviations and Acronyms

- AF = atrial fibrillation
- AT = atrial tachycardia
- CPVA = circumferential pulmonary vein ablation
- ESA = electrically silent area
- LA = left atrial/atrium
- LSPV = left superior pulmonary vein
- PV = pulmonary vein
- RF = radiofrequency

rence of left AT was 2.6 ± 1.6 months (range, 1 to 6 months), and the arrhythmia was incessant in 10 cases. Eleven patients were on antiarrhythmic drugs. Clinical data and CPVA characteristics are shown in Table 1. Written informed consent was obtained from all patients, and the study was conducted in accordance with our institutional ethical guidelines.

Electrophysiologic study. Antiarrhythmic drugs, except amiodarone, were interrupted for at least five half-lives. Quadripolar 6-F catheters were positioned in the coronary sinus and the apex of the right ventricle. Tachycardias were inducible by programmed electrical stimulation alone or during isoprenaline infusion in three patients and were incessant or occurring spontaneously in the remainder. After exclusion of right AT by activation mapping and entrainment techniques, LA mapping and ablation were performed through a transseptal approach.

Electroanatomic mapping. Three-dimensional maps of the LA were reconstructed using the same electroanatomic mapping system with a deflectable 8-mm tip catheter (NaviStar, Biosense-Webster) (5,6). Both bipolar and unipolar electrograms were recorded by the electroanatomic mapping system (filtered at 30 to 400 Hz and 1 to 240 Hz, respectively) and a separate Prucka system (Prucka Cardio EP, Version 4.1.104, GE Healthcare, Chalfont St. Giles, United Kingdom; filter at 30 to 500 Hz and 0.05 to 500 Hz, respectively). Local activation times recorded during tachycardia were processed in a color-coded map for analysis of

the activation sequence. Mapping also was performed during sinus rhythm in patients with paroxysmal or self-terminating tachycardias. Electrically silent areas (ESAs), defined by local electrograms ≤ 0.05 mV, were considered as fixed obstacles to propagation and displayed in gray, whereas areas with adjacent earliest and latest activation, encompassing a critical isthmus, were automatically displayed in dark red. Points showing double potentials and representing local conduction block were tagged in blue.

Characterization of the tachycardias and re-entrant circuit.

Atrial tachycardias were defined as either: 1) macro-re-entrant: continuous propagation sequence with earliest and latest activation adjacent to each other and range of activation times encompassing most of the tachycardia cycle length, or 2) focal: radial spread of activation not covering most of the tachycardia cycle length, with ablation of the focus interrupting the tachycardia (7).

The re-entrant circuit was characterized as single or dual loop (8,9), rotating around natural anatomic barriers, ESA, or previously ablated areas, which were demonstrated by activation mapping and characterized by the presence of double potential along the lines. Gaps were defined as breakthroughs in a previously ablated area, evidenced by activation mapping and by the absence of double potentials. A critical isthmus was defined as a narrow passage between fixed obstacles, demonstrated to be a crucial component of the circuit by activation mapping. Termination of the tachycardia with ablation across the isthmus confirmed its critical role. Anatomic positions were arbitrarily defined as lateral, septal, or posterior wall. Septal and lateral LAs were subdivided in inferior, middle, and superior (roof) segments.

RF catheter ablation. Ablation of macro-re-entrant tachycardias was initially attempted across the critical isthmus, using an 8-mm tipped ablation catheter with pulses at a target temperature of 60°C and a maximum power output of 100 W. The catheter was dragged after 15 to 30 s, after causing an effective lesion, demonstrated by a reduction of the local electrogram amplitude by $\geq 80\%$ or a splitting into

Table 1. Clinical Data and Previous CPVA Design

Patient	Age/ Gender	Heart Disease	Previous Arrhythmia	CPVA Design	Time to Left AT (Months)	Left AT Presentation
1	56/M	None	CAF + AFL	Double post lines + MI	6	Incessant
2	63/M	None	CAF	Double post lines + MI	2	Incessant
3	55/F	DCM	PAF	Single post line + MI	5	Incessant
4	51/M	None	CAF	Single post line + MI	1	Paroxysmal
5	52/M	None	CAF	No post line + MI	3	Incessant
6	58/M	LVH	PAF + AFL	Single post line + MI	1	Incessant
7	61/F	None	CAF	Double post lines + MI	2	Incessant
8	70/M	None	PAF	Single post line + MI	4	Incessant
9	51/M	None	CAF	Double post lines + MI	1	Incessant
10	39/F	None	PAF	Double post lines + MI	2	Paroxysmal
11	74/M	MR	CAF + AFL	Single post line + MI	2	Paroxysmal
12	57/F	None	CAF	Single post line + MI	3	Incessant
13	59/F	None	CAF	Double post lines + MI	2	Incessant

AFL = typical atrial flutter; AT = atrial tachycardia; CAF = chronic atrial fibrillation; CPVA = circumferential pulmonary vein ablation; DCM = dilated cardiomyopathy; LVH = left ventricular hypertrophy due to primary hypertension; MI = mitral isthmus; PAF = paroxysmal atrial fibrillation; post = posterior; MR = mitral regurgitation.

Table 2. Arrhythmia Characteristics, Ablation, and Outcome Data

Patient	TCL (ms)	Mechanism/ Re-Entry Circuit	Isthmus/ Focus Location*	RF Pulses	Fluoroscopy Time (min)	Procedure Time (min)†	Ablation Successful	Follow-Up (months)/ Recurrence
1	270	MCR/dual loop	Septal-inferior/PW	13	17.4	87	Yes	1/AF
2	214	MCR/single loop	Lateral-superior	22	16.4	99	No	3
3	360	MCR/single loop	Lateral-superior/MI	2	35.1	74	Yes	2
4	269	MCR/dual loop	Septal-superior	1	12.4	68	Yes	5
5	260	MCR/dual loop	Septal-inferior	5	30.8	72	Yes	4
6	307	Focal	Septal-superior	1	18.5	93	Yes	3
7	225	MCR/dual loop	Septal-superior	10	20.3	69	Yes	2
8	248	Focal	Lateral-superior	3	10.1	63	Yes	2/AT
9	235	MCR/single loop	Septal-superior	2	26.3	61	Yes	1
10	226	MCR/dual loop	Septal-superior	17	14.3	84	Yes	3
11	242	Focal	Septal-inferior	20	19.5	65	Yes	2
12	179	MCR/dual loop	MI/PW	3	9.5	74	Yes	3
13	420/368	MCR/2 single loops	MI/PW	2	51.6	96	Yes	1

*Location of the isthmuses involved and foci, according to the segments lateral or septal; subdivided in inferior, middle, and superior (roof) segments. †Time in minutes from transeptal puncture to the end of left atrial ablation.

AF = atrial fibrillation; AT = atrial tachycardia; MCR = macro-re-entrant; MI = mitral isthmus; PW = posterior wall; RF = radiofrequency; TCL = tachycardia cycle length in ms.

double potentials. If the tachycardia was not interrupted, ablation was directed to close identifiable gaps involved in the circuit, or an outer loop between fixed obstacles, such as the posterior wall or the mitral isthmus.

Ablation of focal tachycardia was performed at the sites of earliest endocardial activity with the same RF parameters, with pulses of up to 30 s. Unipolar recordings showing negative (QS) pattern with sharp initial deflection also were considered indicative of the focus. If the tachycardia was interrupted and rendered non-inducible, a careful search for other gaps on previous lines was performed, and additional lines were performed if necessary.

Statistical analysis. Continuous variables are expressed as mean ± SD.

RESULTS

Procedural data. Thirteen patients (mean age 57.4 ± 8.9 years, 5 females) underwent mapping and ablation of 14 left ATs, after a mean follow-up of 2.6 ± 1.6 months from the previous CPVA. Baseline characteristics of the patients and previous CPVA design are shown in Table 1. Three-dimensional reconstruction of the LA was performed with an average of 122 ± 23 points. In two patients, the number of mapping points was limited by the nonsustained nature of the arrhythmia. Mean fluoroscopy exposure was 21.7 ± 11.8 min and procedural duration, from transeptal puncture to the end of LA ablation, was 77.3 ± 13 min.

Arrhythmia characterization. Mapping data are presented in Table 2. Three patients were characterized as having focal AT (mean cycle length: 266 ± 35.9 ms). The tachycardia was induced by programmed stimulation in one patient and was incessant in two. Arrhythmia foci in Patients #6 and #11 were located close to the septal aspect of the right PVs ostia (Fig. 1). The earliest activation in Patient #8 was mapped at the superior segment of the lateral LA, anterior to the left superior pulmonary vein (LSPV). Unipolar electrograms at the foci

displayed negative (QS) pattern with sharp initial deflection (Fig. 1, panel 2). All focal tachycardias originated in the vicinity of previous ablation lines.

Of 11 tachycardias characterized as macro-re-entrant (mean cycle length: 275 ± 25 ms), 5 had single loop circuits. Patient 13 had a single-loop tachycardia propagating through the mitral isthmus, with a cycle length of 420 ms (Figs. 2A and 2B). After ablation at the mitral isthmus, a new tachycardia with a cycle length of 368 ms was induced, with a single loop circuit rotating around the right PVs (Figs. 2C and 2D). This arrhythmia was eliminated with linear ablation at the posterior wall.

Patient 3 had a single-loop circuit using the mitral isthmus (cycle length: 360 ms), with earliest activation adjacent to an area of slow conduction anterior to the LSPV. Patients #2 and #9 had single loop circuit tachycardias (cycle length: 214 ms and 235 ms, respectively) using gaps on the previous encircling lines. The gaps were closely positioned at the superior segment of the lateral LA, anterior to the LSPV, and at the septal aspect of the right PVs, respectively (Fig. 3). Single, fragmented electrograms could be recorded at these propagating sites, with continuous areas of double potentials interposed, representing obstacles to activation (Fig. 3, panel A1). Voltage maps showed reversal of the electrophysiological remodeling, with peak-to-peak bipolar voltage >0.1 mV within the previous ablated areas surrounding the foci, indicating loss of continuity of the ablation lines (Fig. 3, panel B1).

Six patients were determined to have dual-loop circuit tachycardias. Patient 12 had a dual-loop tachycardia involving the mitral isthmus and the LA posterior wall. The remaining five patients had circuits rotating around incomplete lines or ESAs, with critical isthmuses and gaps located mostly at the septal aspect of the lines encircling the right PVs. Figure 4 displays the location of foci and critical isthmuses in different views.

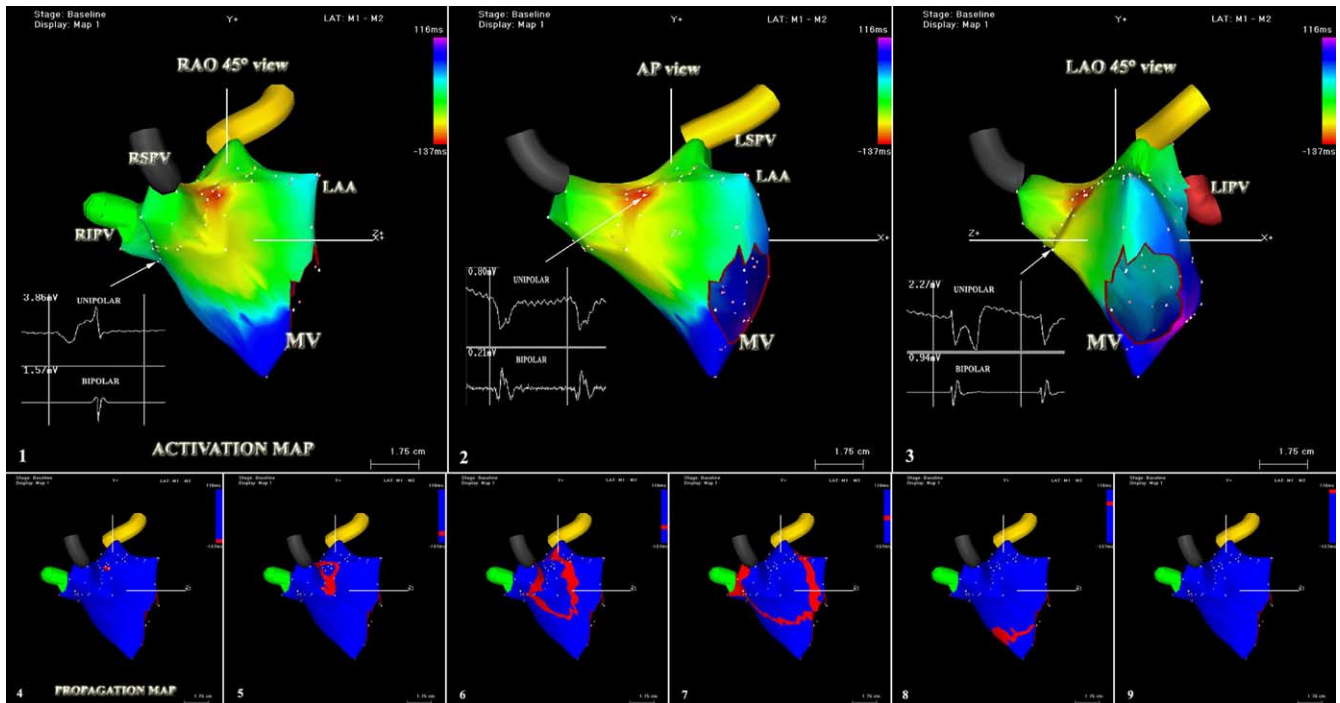


Figure 1. Three-dimensional reconstruction of the left atrium from a patient with a focal tachycardia originating on the interatrial septum anterior to the ostium of the right superior pulmonary vein (RSPV) (Patient #6). (Panels 1 to 3) Activation map during tachycardia, with earliest activation displayed in red and latest in violet. Local unipolar electrogram with a QS pattern is shown in panel 2, corresponding to the site of successful ablation. (Panels 4 to 9) Propagation map in right anterior oblique (RAO) showing centrifugal spread from site of earliest activation. AP = antero-posterior; LAA = left anterior appendage; LAO = left anterior oblique; LIPV = left inferior pulmonary vein; LSPV = left superior pulmonary vein; MV = mitral valve; RIPV = right inferior pulmonary vein.

Ablation results. Of the 14 tachycardias, 13 were successfully ablated, with a mean of 3.5 ± 3.7 RF pulses. Of the 11 macro-re-entrant tachycardias, 8 were interrupted with ablation across the isthmus, whereas 3 required ablation targeting other gaps in the CPVA lines and 2 were only interrupted with ablation at an outer loop on the posterior wall. Ablation in Patient #2 was considered unsuccessful after 22 RF applications, due in part to the self-limited behavior of the arrhythmia during the procedure, precluding more extensive mapping. No complications occurred.

After a mean follow-up of 2.5 ± 1.2 months, no recurrences were reported for 11 patients. Patient 1 developed persistent AF after 1 month of the left AT ablation. Patient #9 presented with a new incessant arrhythmia after 1 month of the left AT ablation, which proved to be a different focal LA tachycardia. This arrhythmia was successfully mapped and ablated at the anterior aspect of the left superior PV ostium.

DISCUSSION

Left AT has been reported after segmental and circumferential ablation of PVs, with an incidence ranging from 2.5% to 20% (3,10,11). However, there are only isolated reports describing re-entrant circuits in detail (3,4). This study presents the electrophysiologic findings in a consecutive series of patients with left AT after CPVA using an electroanatomic mapping system. We demonstrate focal

activity as a possible mechanism and describe a characteristic distribution of critical isthmuses and foci.

Focal tachycardia. Macro-re-entry has always been considered the only mechanism responsible for left AT after CPVA. The long linear lesions required to prevent AF also create new fixed obstacles to propagation, and eventual discontinuities represent ideal substrates for large re-entrant circuits (2,7). However, a focal mechanism has been observed in at least 1 patient after an intraoperative endocardial RF ablation (12). We describe for the first time three cases of focal left AT after a catheter-based CPVA.

Focal AT can be related to automaticity, triggered activity, or re-entry. However, there are currently no reliable means to distinguish these mechanisms in the electrophysiologic laboratory (7). The tachycardia was initiated by programmed electrical stimulation in one patient, and all arrhythmias occurred in areas surrounding previous ablation lines. These areas showed reversal of electrical remodeling caused by CPVA, with adjacent sites of slow conduction and block, the ideal conditions for re-entry. These data suggest focal re-entry on a substrate created by CPVA as the probable mechanism. Indeed, it has been demonstrated in animal models of linear ablation that gaps as small as 0.8 mm are capable of conduction at high rates (13). However, we cannot exclude an unrecognized pre-existing focus of as the primary cause of the current arrhythmia (14-16). It is possible that having modified LA substrate sufficiently to

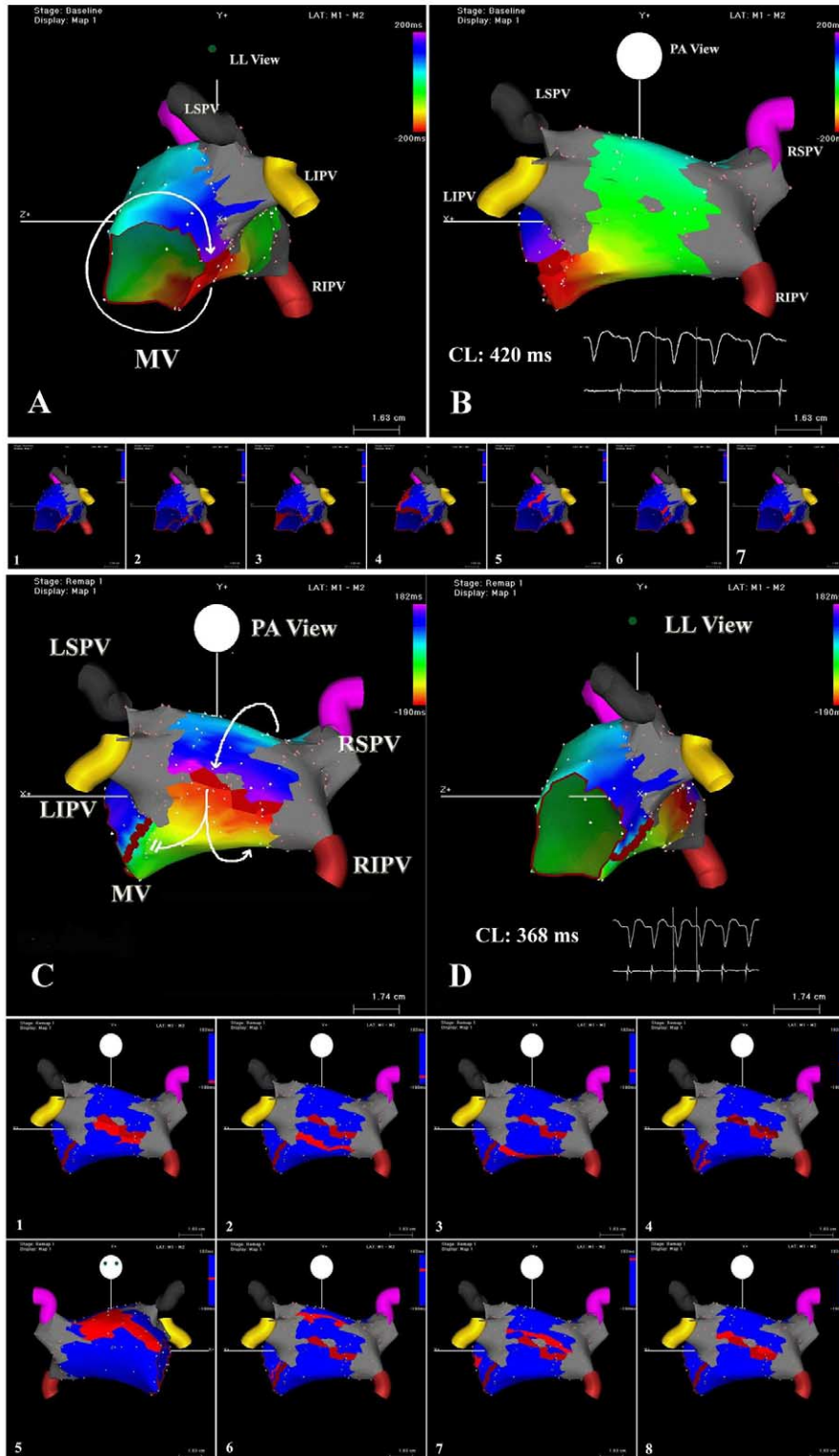


Figure 2. (A and B) Activation maps in pulmonary artery (PA) and left anterior oblique (LAO) views of Patient #13 showing re-entry using the mitral isthmus. The tachycardia cycle length (CL) was 420 ms. (C and D) After ablation and block at the mitral isthmus (dark red line), a new single loop tachycardia, now with a cycle length of 368 ms, was induced. The critical isthmus was identified at the posterior wall, between two previously ablated areas. Ablation at this site eliminated the tachycardia. Propagation maps for each of the two tachycardias are shown as an inset in the lower part of each panel. LL = left lateral; LIPV = left inferior pulmonary vein; LSPV = left superior pulmonary vein; MV = mitral valve; PA = postero-anterior; RIPV = right inferior pulmonary vein; RSPV = right superior pulmonary vein.

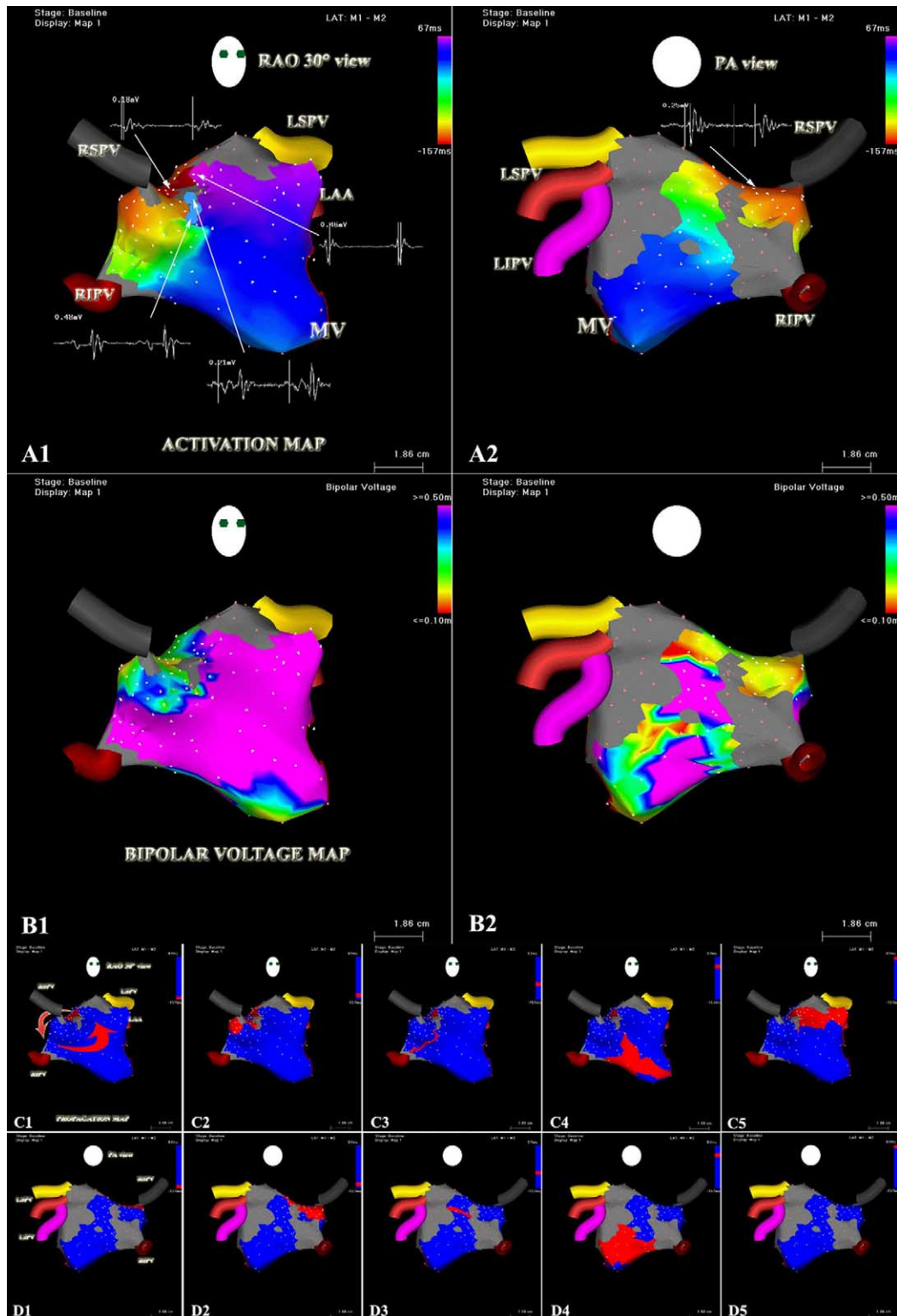


Figure 3. (A1 and A2) Activation maps illustrating a case of single-loop re-entry around a previously ablated area at the septal aspect of the right pulmonary veins (Patient #9) in right anterior oblique (RAO) and postero-anterior (PA) views. A line of block with double potentials (**blue tags**) and an electrically silent area (**gray surface**) can be seen at the superior segment of the septal line in A1. The circuit uses two gaps closely positioned at the superior and middle segments of the line. Single, fragmented bipolar electrograms can be seen at these sites. (B1 and B2) Voltage map in the same views showing recovery of the maximum bipolar voltage in the ablated areas. A wide area of high voltage (**violet**) can be seen over the septum in B1. (C1 to C5 and D1 to D5) Propagation map in RAO and PA showing the spread of activation. LAA = left anterior atrium; LIPV = left inferior pulmonary vein; LSPV = left superior pulmonary vein; MV = mitral valve; PA = postero-anterior; RIPV = right inferior pulmonary vein; RSPV = right superior pulmonary vein.

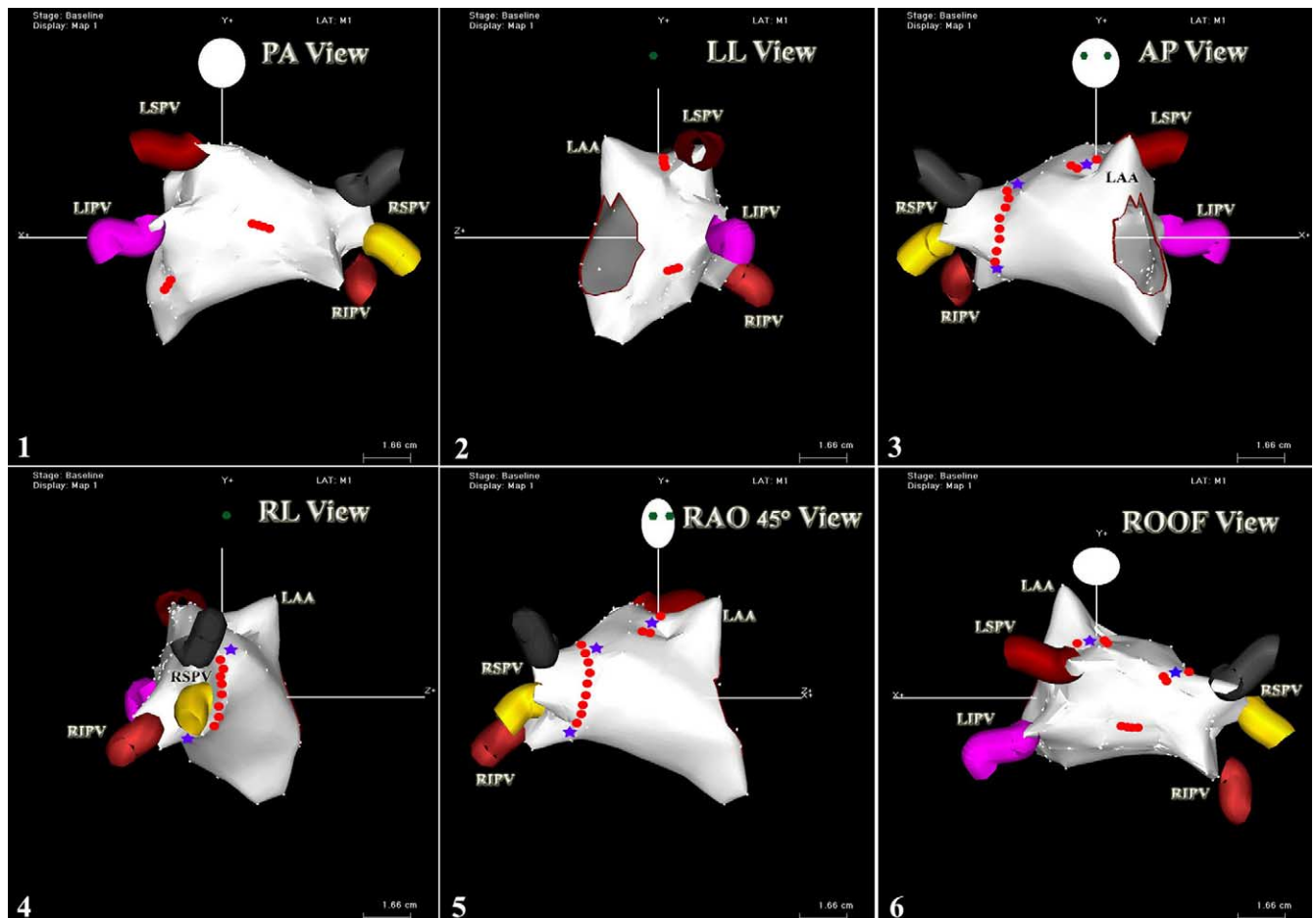


Figure 4. Three-dimensional solid geometry maps of the left atrium in different views, showing the location of the involved gaps (red points) and foci (blue stars). Most of the gaps and all three foci are located over the septal aspect of the right veins and the anterior aspect of the left superior pulmonary vein (LSPV) ostium. AP = antero-posterior; LAA = left anterior atrium; LL = left lateral; LIPV = left inferior pulmonary vein; PA = postero-anterior; RAO = right anterior oblique; RIPV = right inferior pulmonary vein; RL = right lateral; ROOF = cranial view; RSPV = right superior pulmonary vein.

prevent maintenance of AF we are unmasking rotors or foci responsible for fibrillatory conduction and, ultimately, clinical AF.

Macro-re-entrant tachycardia. Electroanatomic-guided ablation of macro-re-entrant AT has proven to be feasible and effective (8,9). The arrhythmia circuit was characterized in the majority of our patients. As expected, most of the tachycardias were related to discontinuities on previous ablation lines. Analysis of the index CPVA showed no residual gaps on the encircling lines at the end of the procedure. In four patients, the LA posterior wall or the mitral isthmus were involved in the circuit of left AT. All four patients had lines placed in these areas during CPVA to prevent re-entry. This suggests that recovery of electrical function or non-transmurality of lesions play an important role in the genesis of these arrhythmias.

The shortest cycle lengths occurred with circuits rotating around closely positioned gaps, rather than with large circuits, such as in mitral isthmus dependent re-entry. This observation emphasizes the importance of deploying con-

tinuous lesion at the encircling lines, hence avoiding these very rapid tachycardias.

Distribution of critical isthmuses and foci. Although acute electrical remodeling was achieved in all patients at CPVA, with no identifiable gaps, recovery of conduction in critical points on the lines was the basic mechanism of macro-re-entrant, and possibly focal activity.

In two patients, the arrhythmias were entirely related to gaps on lines at the mitral isthmus and posterior wall, and all but one of our patients had lines deployed at the posterior wall during CPVA. Incomplete lines or recovery of conduction at these areas can create the slow conduction necessary to sustain re-entry. Although ablation at these sites are designed to avoid macro-re-entry, it is still not clear whether they actually prevent or contribute to it (14), and further studies are needed to define their role in CPVA.

We observed a tendency of gaps and foci to occur at the septal aspect of the right PVs, and at the superior segment of the lateral LA, anterior to the LSPV (Fig. 4). Eight of 11 macro-re-entrant tachycardias had critical components at

these areas. Also, all three of the focal arrhythmias originated in these same areas. It is noteworthy that the few reports describing in detail macro-re-entrant tachycardia after catheter ablation are associated with gaps at these same locations (3,4).

Continuous linear lesions are difficult to achieve, even under direct visualization during intraoperative ablation (8). Creating transmural, permanent lines of block with a percutaneous approach can be even more challenging. Technical factors can be used to explain the propensity to discontinuities at specific areas. Frequently, only a narrow rim of atrial tissue between the LSPV and the LA appendage is present, which makes stable catheter positioning difficult to attain. In addition, the presence of dense pectinate muscle in the LA appendage, contiguous to the normal atrial wall, can make transmural lesion more difficult to achieve in this area.

Good access to the posteroseptal area is essential to perform the encircling lines of the right PVs. This is not always possible with the current approach. A very high or posterior transseptal puncture may make stable catheter positioning over the septum difficult, with the distal electrode in poor contact or inadequate orientation with the endocardial surface. Another possible explanation is the presence of muscle bundles deeply located in the epicardial fat of the posterior interatrial and atrioventricular groove (17,18). These fibers were observed merging with the atrial wall muscle and may be spared by RF ablation, giving origin to breakthroughs on the septal line due to nontransmural lesions. Thomas et al. (8) reported a high prevalence of circuits involving the interatrial septum in a series of patients with left AT following an intraoperative RF ablation procedure. Our similar results with a catheter-based approach seem to confirm these areas as critical points for recovery of conduction.

Clinical implications. Our experience suggests that the septal aspect of ablation lines encircling the right PVs and the area anterior to the left superior PV are particularly vulnerable to recovery of conduction after CPVA, predisposing patients to the development of left AT. Particular attention should be paid at these sites to ensure continuous, transmural lesions.

Meticulous mapping to define the LA anatomy, particularly between the LSPV and the LA appendage, is necessary to guide linear ablation. Further research is needed to define the role of lines in the mitral isthmus and the posterior wall.

The recognition of focal tachycardia as a possible mechanism can further assist the treatment because detailed mapping with high density of points is necessary only at the sites of early activation, shortening the procedure time and fluoroscopy exposure. The impact of other image methods or new catheter designs on the occurrence and characteristic of these arrhythmias is unclear.

Study limitations. The small number of patients limits the scope of our observations and, in particular, because the data regarding the incidence of AT after CPVA were collected

retrospectively, this may not be an accurate reflection of the incidence. Complete elucidation of the electrophysiologic mechanism of focal tachycardias was not attempted. However, this is considered difficult with current techniques and of limited value from a therapeutic point of view.

The purpose of this article was to describe the features and management of patients returning with AT after AF ablation. The period of follow-up is short, and we cannot be certain that patients will not have recurrences of AT at this stage.

We use high power and temperature settings in our laboratory, both for AF ablation and for ablation of focal or macro-re-entrant AT. These settings may not be appropriate in centers with less experience or lower volumes of cases. We have recently experienced a case of atrio-esophageal fistula occurring as a consequence of RF ablation in the posterior wall of the LA and have subsequently recommended lower settings of 50 W and 55°C in direct response to this (19).

Conclusions. Left AT is an important midterm complication after CPVA. This arrhythmia can be due to either a macro-re-entrant or a focal mechanism. Re-entry occurs most commonly using gaps on previously continuous ablation lines. Such gaps have shown a tendency to occur at the septal aspect of the right PVs and between the LSPV and the LA appendage. Macro-re-entrant circuits frequently involved the mitral isthmus and posterior wall of the LA. Particular attention is required to ensure that linear lesions are continuous and transmural at these sites. Electroanatomic mapping is effective in characterizing such arrhythmias and guiding ablation.

Reprint requests and correspondence: Dr. Carlo Pappone, Department of Cardiology, San Raffaele University Hospital, Via Olgettina 60, 20132, Milan, Italy. E-mail: carlo.pappone@hsr.it.

REFERENCES

1. Pappone C, Rosanio S, Augello G, et al. Mortality, morbidity, and quality of life after circumferential pulmonary vein ablation for atrial fibrillation: outcomes from a controlled nonrandomized long-term study. *J Am Coll Cardiol* 2003;42:185-97.
2. Packer DA, Asirvatham S, Munger TM. Progress in nonpharmacologic therapy of atrial fibrillation. *J Cardiovasc Electrophysiol* 2003;14:S296-309.
3. Villacastin J, Perez-Castellano N, Moreno J, González R. Left atrial flutter after radiofrequency catheter ablation of focal atrial fibrillation. *J Cardiovasc Electrophysiol* 2003;14:417-21.
4. Oral H, Knight BP, Morady F. Left atrial flutter after segmental ostial radiofrequency catheter ablation for pulmonary vein isolation. *Pacing Clin Electrophysiol* 2003;26:1417-9.
5. Pappone C, Oreto G, Rosanio S, et al. Atrial electroanatomic remodeling after circumferential radiofrequency pulmonary vein ablation. Efficacy of an anatomic approach in a large cohort of patients with atrial fibrillation. *Circulation* 2001;103:2539-44.
6. Pappone C, Santinelli V, Manguso F, et al. Pulmonary vein denervation enhances long-term benefit after circumferential ablation for paroxysmal atrial fibrillation. *Circulation* 2004;109:327-34.
7. Saoudi N, Cosio F, Waldo A, et al. A classification of atrial flutter and

- regular atrial tachycardia according to electrophysiological mechanisms and anatomical bases: a Statement from a Joint Expert Group from the Working Group of Arrhythmias of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. *Eur Heart J* 2001;22:1162-82.
8. Ouyang F, Ernst S, Vogtmann T, et al. Characterization of re-entrant circuits in left atrial macro-re-entrant tachycardia critical isthmus block can prevent atrial tachycardia recurrence. *Circulation* 2002;105:1934-42.
 9. Peichl P, Kautzner J, Cihak R, Vancura V, Bytesnik L. Clinical application of electroanatomical mapping in the characterization of "incisional" atrial tachycardias. *Pacing Clin Electrophysiol* 2003;26:420-5.
 10. Seshadri N, Marrouche NF, Wilber D, Packer D, Natale A. Pulmonary vein isolation for the treatment of atrial fibrillation: recent updates. *Pacing Clin Electrophysiol* 2003;26:1634-40.
 11. Ernst S, Ouyang F, Lober F. Catheter-induced linear lesions in the left atrium in patients with atrial fibrillation: an electroanatomic study. *J Am Coll Cardiol* 2003;42:1271-82.
 12. Duru F, Hindricks G, Kottkamp H. Atypical left atrial flutter after intraoperative radiofrequency ablation of chronic atrial fibrillation: successful ablation using three-dimensional electroanatomic mapping. *J Cardiovasc Electrophysiol* 2001;12:602-5.
 13. Thomas SP, Wallace EM, Ross DL. The effect of a residual isthmus of surviving tissue on conduction after linear ablation in atrial myocardium. *J Interv Card Electrophysiol* 2000;4:273-81.
 14. Kistler PM, Sanders P, Hussin A, et al. Focal atrial tachycardia arising from the mitral annulus: electrocardiographic and electrophysiologic characterization. *J Am Coll Cardiol* 2003;41:2212-9.
 15. Kistler PM, Sanders P, Fynn SP, et al. Electrophysiological and electrocardiographic characteristics of focal atrial tachycardia originating from the pulmonary veins: acute and long-term outcomes of radiofrequency ablation. *Circulation* 2003;108:1968-75.
 16. Takahashi Y, Iesaka Y, Takahashi A, et al. Re-entrant tachycardia in pulmonary veins of patients with paroxysmal atrial fibrillation. *J Cardiovasc Electrophysiol* 2003;14:927-32.
 17. Ho SY, Sanchez-Quintana D, Cabrera JA, Anderson RH. Anatomy of the left atrium: implication for radiofrequency ablation of atrial fibrillation. *J Cardiovasc Electrophysiol* 1999;10:1525-33.
 18. Platonov PG, Mitrofanova LB, Chireikin LV, Olsson SB. Morphology of inter-atrial conduction routes in patients with atrial fibrillation. *Europace* 2002;4:183-92.
 19. Pappone C, Oral H, Santinelli V, et al. Atrio-esophageal fistula as a complication of percutaneous transcatheter ablation of atrial fibrillation. *Circulation* 2004;109:2724-6.