A typical macroreentrant atrial tachycardias are frequently encountered in patients after cardiac surgery, correction of congenital heart disease, or atrial fibrillation ablation. Ablation of one circuit can produce an abrupt change in atrial activation sequence giving rise to another macroreentrant circuit. In this report, we present a unique case of a bilateral septal macroreentrant atrial tachycardia, which developed after perimital flutter ablation in a patient with mitral valve replacement.

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A 59-year-old man with a mitral valve replacement (bioprosthesis) in 1983 for a rheumatic mitral stenosis and a rereplacement in 1999 (right-sided approach with a vertical incision of the interatrial septum) with a mechanical prosthesis attributable to the degeneration of the former prosthesis was referred for the ablation of a sustained atrial tachycardia with a stable cycle length of 260 ms. The echocardiogram demonstrated a left ventricular ejection fraction of 45% to 50%, a moderately dilated right atrium (RA) as well as left atrium (LA) without signs of a prosthetic malfunction. The baseline surface 12-lead ECG showed a atypical atrial flutter with a regular, monomorphic atrial activity and an irregular atrioventricular conduction (Figure 1A). With broad positive flutter waves in unipolar lead V1 and inferior limb leads II, III and avF but negative flutter waves in limb leads I and avL, the ECG morphology suggested LA localization. Any significant 12-lead isoelectric interval was absent. Endocardial activation mapping with a quadripolar catheter in the RA and a steerable octopolar catheter in the coronary sinus (CS, Bard Medical, Covington, GA) demonstrated a distal to proximal activation sequence in the CS with a baseline cycle length of 260 ms, suggestive of a lateral to septal activation of the LA posterior wall (Figure 1B). An irrigated tip ablation catheter (Navistar Thermocool, Biosense Webster, Diamond Bar) was advanced into the LA via a transseptal puncture. Return cycle mapping at a pacing cycle length of 240 ms demonstrated a postspacing interval (PSI) of 430 ms in the RA free wall, 320 ms in the posterior LA wall, 290 ms in the LA roof, and finally 260 ms in the distal CS, allowing the confirmation of an LA localization of the macroreentrant tachycardia (Figure 1C). Subsequent electroanatomical mapping (CARTO XP, Biosense Webster, Diamond Bar) of the LA during tachycardia revealed a clockwise perimital flutter, but with a relatively narrow isthmus (12 mm) in the anterior LA wall, bounded above (and anteriorly to the RSPV ostium) as well as below (to the anterior mitral annulus) by a line of block, consistent with a surgical approach scar for the mitral valve replacement surgery (Figure 1D). Ablation of this isthmus was started targeting single, healthy voltage potentials choosing this more anteriorly located narrow isthmus over the classic mitral isthmus. The tachycardia slowed progressively (CL 330 ms) during radiofrequency energy delivery, and a pause followed by an abrupt change in CS activation was observed after the seventh radiofrequency application, whereas the RA activation continued unchanged (Figure 2A). The 12-lead ECG of the second tachycardia clearly revealed an isoelectric interval (130 ms) across all 12 leads, suggesting the presence of a residual narrow isthmus of slow conduction as the differential diagnosis of a focal atrial tachycardia (Figure 2B). Repeat return cycle mapping from the LA septum around the fossa ovalis, where multicomponent diastolic potentials were recorded, showed a PSI which equaled the tachycardia cycle length (Figure 2C). Furthermore, pacing dissociated the LA from the RA free wall (Figure 2D). Because ablation at the LA septal site with a PPI=TCL had no effect, the decision was taken to perform another electroanatomical map of the LA. However, despite careful mapping, the latter accounted only for 75% of the full tachycardia cycle (Figure 3A, left). However, mapping the RA septal accounted for nearly the entire remaining 25%, showing an activation wavefront completing a bilateral macroreentrant circuit around the fossa ovalis (Figure 3A, right). Here, return cycle mapping depicted a PPI equaling the TCL, where radiofrequency delivery finally terminated the tachycardia after 8 seconds (Figure 3B). No tachycardia was inducible by burst pacing immediately after tachycardia termination and 20 minutes thereafter. Of note, the final surface 12-lead ECG in sinus rhythm showed a block of the Bachmann bundle (P-wave duration ≥110 ms with a terminal deep negative component in II, III, & avF) correlating with the ablation of the second tachycardia at the site of residual conduction across the Bachmann bundle.

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From the Hôpitaux Universitaires de Genève, Service de Cardiologie, Geneva, Switzerland.

Correspondence to Dipen Shah, MD, Service de Cardiologie, Hôpitaux Universitaires de Genève, Rue Gabrielle-Perret-Gentil 4, CH - 1211 Genève 14, Switzerland. E-mail dipen.shah@hcuge.ch

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LA flutter is frequently related to a history of open heart surgery or an ablation procedure performed for atrial fibrillation and has become an increasingly prevalent arrhythmia in many patients. However, its diagnosis as well as catheter ablation remains a challenge, as can be demonstrated in the herewith presented case, where a reentrant circuit involving the mitral annulus was identified as, notably the left-sided, component of a biatrial, septal macroreentrant tachycardia in a patient who underwent 2 surgical interventions for a mitral valve replacement. Such dual-loop macroreentrant atrial tachycardias, defined as the concomitance of 2 simultaneous circuits, each meeting the definition of a reentrant tachycardia, have already been reported to be frequent in patients after cardiac surgery (mimicking the ECG pattern of a common atrial flutter in some patients after a surgical atriotomy), correction of congenital heart disease, or atrial fibrillation ablation. A single, interesting case report represented an early description of an atypical atrial flutter using the myocardium of the CS as a critical isthmus in a patient without any structural heart disease. However, clinical descriptions of biatrial circuits involving the interatrial septum are scarce. The following distinct clinical as well as intracardiac findings were useful in directing toward a certain differential diagnosis. Generally, one of the first steps in confirming the suspicion of a left atrial flutter is excluding a cavotricuspid isthmus–dependent flutter, typically by demonstrating interatrial dissociation, long PPIs, or complete cavotricuspid isthmus block in sinus rhythm. PPI gradients (PPIs from multiple sites at the same pacing cycle length) allow the localization of the circuit to a region, in this case the LA. Perimital atrial flutters in particular are more common in patients with a history of iatrogenic or noniatrogenic structural heart disease, yet, scar or low-voltage areas on the posterior wall of the LA as a posterior boundary of this circuit have also been described by means of electroanatomic voltage mapping in patients without obvious structural disease. Another mentionable observation is the dissociation of the CS and posterior LA from the RA free wall (here during return cycle pacing in the LA aspect of the interatrial septum), a frequently seen phenomenon in patients with extensive scars after open heart surgery. Nevertheless, it has to be kept in mind as a first clue that the appearance of macroreentrant
circuits may not be limited to the LA, although the associated structural heart disease or related interventions are essentially left sided because the interventional access may, for example, involve the right-sided aspect of the interatrial septum. As a matter of fact, the second surgical mitral valve replacement in our patient required a right-sided approach with a vertical incision of the interatrial septum at the fossa ovalis to access the left-sided annulus. Accordingly, we were able to identify a second, right-sided component of the biatrial macroreentrant tachycardia with a circuit around the fossa ovalis, which was only demasked after having blocked the critical isthmus of the perimitral flutter in the anterior mitral annulus; thereby changing activation sequence and cycle length of the tachycardia and necessitating yet another left-sided mapping procedure, which, however, did not account for the full tachycardia cycle. The latter circumstance offers another valuable clue (in awareness of the clinical history and surgical technique) to deliberate about whether a chosen strategy focusing on only 1 chamber of origin should be reconsidered. It is well conceivable that not both circuits were actually present in the beginning of the procedure and that the second component only developed with slowing/ablation of the perimitral circuit. However, the latter remains elusive because no right septal return cycle mapping was performed in the beginning. Finally, our report emphasizes the importance of taking into account the pathophysiological as well as electrophysiological importance of the interatrial conduction, which normally occurs via anatomically distinct connections (already described by Bachmann, Wenckebach, and Thorel in the beginning of the twentieth century) such as the Bachmann bundle (60%), around the fossa ovalis and rarely in the coronary sinus. These considerations may give direction to a definite therapeutic success as shown with the final electroanatomical map identifying the critical isthmus of the second macroreentrant tachycardia.

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References


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Mehdi Namdar, Pascale Gentil-Baron, Henri Sunthorn, Haran Burri and Dipen Shah

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