A 63-year-old male patient was admitted for catheter ablation of persistent atrial fibrillation (AF). On echocardiography, the left atrial (LA) size was 48 mm and the left ventricular function was normal. After electric cardioversion, ipsilateral pulmonary veins (PVs) were circumferentially ablated in sinus rhythm using double-Lasso (Lasso, Biosense Webster, Diamond Bar, CA) technique.1–3 Both ipsilateral PVs were simultaneously isolated. Two years later, the patient was admitted for the ablation of recurrent, drug-resistant atrial tachycardia (AT) (Figure 1A).

The baseline tachycardia cycle length (TCL) was 350 ms. A 20-pole mapping catheter (Irvine Biomedical Inc, Irvine, CA) was inserted through the right jugular vein and positioned in the coronary sinus (CS). An 8-mm-tip ablation catheter (Japan Lifeline Inc, Tokyo, Japan) was advanced into the LA through a transseptal puncture. Mapping during AT revealed the resumption of conduction in the right PVs. On entrainment mapping, the post pacing interval (PPI-TCL) was found to be 120, 70, 56, and 10 ms at the low lateral right atrium, anterior LA, bottom of LA, and left septum near the right PVs, respectively. On activation mapping using a 3D electroanatomic mapping system (Carto, Biosense Webster), earliest atrial activation was observed on the roof of right superior vein and the latest at the bottom of right inferior vein. Successful entrainment from the superior and inferior veins revealed that both sites were within the tachycardia circuit (Figure 1B and 2A). Entrainment from the LA septum near the right PVs demonstrated that septal myocardium was also a part of the reentrant circuit. Furthermore, P-wave morphology during entrainment pacing from near the roof of the right superior vein was identical to that observed during tachycardia (Figure 2B). Mapping near the site of the earliest atrial activity on the 3D map identified a 140-ms-long, fractionated and low-amplitude signal (0.044 mV, Figure 3A and 3B). After 4.5 seconds of radiofrequency (RF) delivery at that site, the tachycardia terminated (Figure 3C). Induction attempts from the right PVs were negative. In sinus rhythm, a gap in the circumferential PV lesion was observed at the site that showed latest activation during tachycardia (Figure 4A and 4B). It was successfully ablated, ensuing in simultaneous right inferior and superior PVs isolation (Figure 4C). The patient has remained free from any arrhythmia for 3 years.

**Discussion and Teaching Points**

Electric PV isolation is the cornerstone of AF ablation, which is now widely undertaken in symptomatic, drug-refractory patients.4,5 PV antrum isolation has higher success rate than segmental isolation.6–8 After ablation, gaps in circumferential lesions may result in a variety of recurrent ATs. These ATs are often poorly tolerated and difficult to manage using pharmacological rate- and rhythm-control strategies, necessitating ablation for long-term maintenance of sinus rhythm.9–13

In the practical context of diagnostic mapping and ablation, these ATs could be classified into focal, localized reentry and macroreentrant types.10–12,14 Macuareentry is characterized by activation that can be recorded over the entire AT cycle length (CL) and generally encircles a “large” central obstacle of several centimeters diameter such that the entrainment from several separate segments yields PPI equal to CL. Localized reentry is diagnosed when a small area (≤2 cm diameter) contains the entire AT CL and where the wave front spreads centrifugally to the rest of atria. In focal AT, atrial activity originating from a single focus spreads out centrifugally.

Activation and entrainment mapping are useful diagnostic techniques.15–19 A stimulus that captures the tissue in the excitable gap of the reentrant circuit resets the tachycardia during entrainment pacing. The resulting orthodromic wave front returns to the pacing site after completing one revolution along the circuit. Thus, the interval from the last stimulus to the subsequent depolarization recorded at the pacing site lying within the circuit is equal to the revolution time along the circuit, which, in fact, equals (within 30 ms) the TCL. However, there are some caveats. Activation mapping alone may not be able to differentiate a focal tachycardia, mimicking macroreentry from true macroreentry. In other words, tachycardia arising from a focus near a completely blocked ablation line can mimic the activation sequence of macroreentry. Additional entrainment mapping is required to differentiate these 2 different tachycardias. For rapid and accu-
rate identification of postablation ATs, a deductive approach including these 2 mapping techniques should be divided into 3 diagnostic steps: (1) assessing the stability of cycle length, (2) a search for macroreentry, and (3) if macroreentry is excluded, a search for a focal-source with centrifugal activation of the atria.10,11 If the cycle length variability is greater than 15%, and particularly in the rare cases in which the AT has a start-stop pattern, a focal AT is deemed highly likely. When there was less than 15% cycle length variability, both focal and macroreentrant AT were deemed plausible.10,11

Figure 1. A, Twelve-lead electrogram of the clinical atrial tachycardia. B, Entrainment mapping at right inferior pulmonary vein (RIPV) showed that postpacing interval tachycardia cycle length was 10 ms. RA indicates right atrium; CS, coronary sinus.

Figure 2. A, Entrainment mapping at right superior pulmonary vein (PV) showed that postpacing interval tachycardia cycle length was 10 ms. B, P-wave morphology during right superior PV entrainment (red arrow) is identical to that observed during tachycardia (blue arrow). RA indicates right atrium; CS, coronary sinus; and RF, mapping catheter (p, proximal; d, distal).
The electrophysiological mechanism of AT depends on the previous ablation lesions and the type of AF.\textsuperscript{5,10,12,20–23} Although extensive atrial ablation frequently leads to the occurrence of AT, it can also occur after PV isolation alone. In addition, in a study comparing segmental ostial PV isolation with circumferential PV ablation, symptomatic AT was observed more frequently in the patients with large circumferential lesions.\textsuperscript{7,8} Perimtrial AT, LA roof–dependent AT, and cavotricuspid isthmus–dependent AT comprise of the majority of the macroreentrant tachycardia circuits.\textsuperscript{20,21} These ATs utilize regions of incomplete or recovered ablation lesions or anatomic obstacles as the critical isthmuses to sustain the arrhythmia. Both activation and entrainment mapping should be undertaken to determine and confirm macroreentrant activation patterns.

Several prior studies have shown that the PVs can reconnect late after the isolation procedure and can lead to the recurrence of atrial tachyarrhythmia.\textsuperscript{22,23} The mechanism of AT associated with PV reconnection is focal in most of the cases wherein the tachycardia originates from the muscle sleeve of the reconnected PV.\textsuperscript{22,23} The LA activation occurs with 1-to-1 or Wenckebach conduction through the gap in the circumferential PV lesion set. Rarely, LA-PV reentrant tachycardia is observed in patients with arrhythmia recurrence associated with PV reconnection.\textsuperscript{24}

In the present case, activation mapping demonstrated a reentrant pattern and entrainment mapping showed that the right PVs and the LA septum were the integral parts of the tachycardia circuit. Exiting the PV from near the roof of right superior vein, the activation spread to the LA septum anteriorly and reentered the PV through a gap in the circumferential lesion at the bottom of the right inferior vein; where the electric contiguity between the 2 PVs affected the wave front’s return to the right superior vein (Figure 3A, dotted arrow). The gap between right superior vein and the LA was the site of critical isthmus with slow conduction. First, RF
delivery at that site immediately terminated the tachycardia (Figure 3C). The second RF delivery, now in sinus rhythm, at the bottom of right inferior vein, simultaneously isolated both the right PVs from the LA (Figure 4C), confirming that the 2 veins were mutually connected and the conduction gap in right inferior vein was critical for reentry of the tachycardia wave front from the LA into the PVs. These findings confirm that the mechanism of this AT was LA-PV reentrant tachycardia with active involvement of 2 gaps in the previously deployed circumferential ablation line. This tachycardia could have been potentially misdiagnosed as focal AT arising from the right PVs in the absence of entrainment mapping.

In conclusion, multiple PV reconnections after electric PV isolation could result in PV-LA reentrant AT. For a precise diagnosis of such an AT, it is important to evaluate PVs for reconnection with the possibility of gap-mediated PV-LA reentry.

Disclosures
None.
Atrial Tachycardia after Atrial Fibrillation Ablation

Miyazaki et al present an interesting case of a reentrant atrial tachycardia (AT) after atrial fibrillation (AF) ablation and provide many instructive educational points for our readers in their discussion.

Atypical flutterers (reentrant atrial tachycardias) after AF ablations are among the most challenging of arrhythmias encountered in contemporary interventional electrophysiology. Often they are seen at the end of an already complex and arduous ablation, the arrhythmias may be multiple, and the operator has a definite end point to meet of flutter termination and inability to reinude.

To maximize the chance of success without complication for these difficult arrhythmias, the operator needs to fully understand the value, exceptions to rules, and execution of various apparently simple maneuvers, including activation mapping, and catheter ablation of recurrent atrial tachycardias after stepwise ablation of long-lasting persistent atrial fibrillation. Circ Arrhythm Electrophysiol. 2010;3:160–169.

Key Words: atrial tachycardia  macroreentry  catheter ablation  pulmonary vein isolation

References
mapping, electrogram annotation, and entrainment. Perhaps even more important is understanding what information cannot be used in the usual method of interpretation when dealing with post-AF ablation reentry. Added to this, a thorough appreciation of the relevant anatomy and knowledge of where prior ablations were done is critical for success. To further bring out the teaching points from this representative case, let us examine some of the salient features.

**What Can the Surface Lead Tell Us?**

The 12-lead electrogram of the persistent arrhythmia is shown in Figure 1A. If this were an automatic AT, we can directly deduce from the P-wave vector the approximate site of origin often with significant accuracy. The P wave is positive in the inferior leads and mostly negative in aVR and aVL, suggesting a high (superior) origin. Candidate sites could include the roof of the superior pulmonary veins, atrial roof, or left-sided Bachmann bundle region. However, it is important to note that with reentrant arrhythmia, the P-wave morphology has much less predictive value for the likely successful site of ablation. The P wave for reentrant AT, as is the QRS for reentrant ventricular tachycardia, signifies the exit. The concept of the exit for a tachycardia itself is not straightforward to comprehend but perhaps is best thought of as the transition from the abnormal tissue housing the critical components of the circuit to relatively normal myocardium. At times there can be a significant distance between the critical components of a reentrant circuit and the exit site. From this ECG, perhaps all we can say is the right atrium is probably normal and a left atrial circuit exits and depolarizes the right atrium in a superior to inferior direction.

The authors have importantly pointed out that when pacing within the slow zone of the flutter circuit and when entraining at similar sites, the surface P-wave morphology may be reproduced.

**Choice of Mapping/Ablation Catheter**

The authors used an 8-mm-tipped ablation catheter for mapping the AT. As is explained below, detailed mapping with meticulous care in interpreting the electrograms obtained is essential when dealing with reentrant tachycardias and diseased/previously ablated hearts. The 8-mm-tipped catheter has a wide antenna, and, especially in regions of complex geometry such as between pectinate muscles, pulmonary vein junctions, septum (where both right and left atrial myocardial signals are present), and in the region of the vein of Marshall, accurate signal diagnosis and interpretation may not be possible with a large catheter. In general, for these cases, smaller electrode tips (such as 3.5 or 4 mm) are preferred for mapping when possible. Large lesions can be created with 8-mm electrodes, but with open irrigation, myocardial signals are present), and in the region of the vein of Marshall, accurate signal diagnosis and interpretation may be difficult to confirm. A useful point to remember is that at 2 or more locations the atrial lesion size and transmurality is typically adequate.

**Entrainment Mapping**

The authors note that with entrainment mapping, the δ postspacing interval (PPI-TCL) was found to be 120 ms in the low lateral right atrium and 10 ms near the right pulmonary veins. This technique is sometimes referred to as “pinging,” where an approximate idea of where the circuit is housed can be defined. In general, the greater the δ-PPI, the further away the pacing site is located from the critical limbs of the circuit. However, in very diseased hearts and after prior ablation, regions of conduction block may separate a pacing site from the circuit such that a site close to the critical components of the circuit to relatively normal myocardium. At times there can be a significant distance between the critical components of a reentrant circuit and the exit site. From this ECG, perhaps all we can say is the right atrium is probably normal and a left atrial circuit exits and depolarizes the right atrium in a superior to inferior direction.

Do we routinely find sites with a δ-PPI of zero? Because of prior ablation and disease, pacing faster than the tachycardia may cause decrement within the circuit and the PPI may be longer than the TCL even when inside the circuit. Pacing as slow as possible, but still fast enough to entrain the tachycardia may help, but then entrainment can be difficult to confirm. A useful point to remember is that if at 2 or more locations the δ-PPI is of the same value (small but not zero), it is highly likely that all of these points are within the circuit.

The authors state that they were able to demonstrate concealed entrainment from the pulmonary veins. It is likely that this was done at the ostium or just atrial to the pulmonary veins. When pacing within the pulmonary veins, there is invariably exit delay to the atrium even without prior ablation at the ostium and would have been more so in this case, making a δ-PPI of zero unlikely unless the entire circuit was itself within the pulmonary veins with an exit to the remainder of the atrium (this was not the case in this instance).

We should remember that entrainment is not only for defining where we need to ablate but is a powerful maneuver for defining the mechanism of the tachycardia. As was explained with the relation to ECG interpretation and with regard to activation mapping below, there is a major difference when approaching a reentrant versus an automatic focal source tachycardia. The mechanism of reentry is readily identified when entrainment occurs with fusion evident in the atrial activation sequence and/or P wave, and the last beat after pacing is entrained but not fused. This response is distinctly different from the overdrive suppression without fusion seen with an automatic tachycardia.
Activation Mapping

The authors found that the “earliest atrial activation was observed on the roof of the right superior vein and the latest at the bottom of the right inferior vein.” With reentrant tachycardia, what do the activation sites signify? In a circuit, there is always some region that has electric activity earlier and later than any other electrogram. In other words, there will be electrograms to be found that span the cycle length of the tachycardia. The designations of sites as early (or late) are entirely dependent on what was chosen as a reference electrogram and the “windows” that we choose to set up in mapping the tachycardia. For example, in this instance, if the low right atrium was taken as the reference point and three-quarters of the cycle length taken before and a quarter after the reference electrogram, we would have found early and late sites as much as several cm away from that recorded in the map.

Therefore, the operator should focus on mapping the entire circuit. That is, accounting for the entire cycle length with the recorded electrograms, and, if this is done, it is likely that the entire circuit can be visualized by following the color and activation time sequences with a 3-dimensional map.

How do we deal with the fragmented signals that are common and similar to what the authors found near the pulmonary veins? If we include these sites in our activation map, which component do we take? Or if we choose not to include it (location-only fragmented signal), there will be a large chunk (sometimes more than 50% of the tachycardia cycle length) that will not be included in the map. To deal with this, the target percentage of the cycle length of the flutter to be mapped should be the cycle length of the flutter minus the fragmented signal duration. If, however, the fragmented signal duration added to the otherwise mapped flutter cycle length significantly exceeds the mapped flutter cycle length, at least a portion of the fragmented signals are likely to be bystanders.

The importance of defining tachycardia mechanism can be readily appreciated from the above. If this were a focal atrial tachycardia, then the early site means everything (identifying the tachycardia origin), and the late site means little and simply represents the latest site to get activated in the atrium during the automatic tachycardia. In some tachycardias, however, an apparent pattern of focal activation is found, but there are also characteristics of reentry. These findings indicate a so-called microreentrant atrial tachycardia and are increasingly recognized in the post–atrial fibrillation ablation scenario. The clue, however, is the observation that at the site of “early” activation, markedly fragmented signals explaining nearly all of the cycle length of the tachycardia are identified. Targeting these sites for ablation is often successful.

One Gap Versus Two Gaps Versus … Gaps

The authors report an interesting and frequently seen finding. When we are mapping a reentrant tachycardia and find that the pulmonary veins have not been isolated and we locate and ablate one of the gaps—often with fragmented signals—the flutter terminates but there is still conduction into the vein and then a second gap is identified and ablated to isolate the vein. Why does this happen? What is the difference between 1 versus 2 gaps, and so on? This scenario can occur in 1 of 2 situations.

When creating a line during AF ablation, we must be cognizant of what anchors the end of the line to prevent conduction around the line. The anchor is as important as making sure that the line is complete and transmural. A frequent anchor is the wide area encircling ablation lesion line around the pulmonary veins. For example, consider a line created between the mitral annulus and the ablation lesion circle around the left-sided pulmonary veins. If there are 2 gaps in the ablation circle, then the mitral isthmus flutter can continue with conduction proceeding into and outside the vein to complete the circuit. Similarly, when a roof line is created between the 2 pulmonary vein circles, if there are 2 gaps in the right vein circle, then conduction can go into the veins and out and thus bypass the roof line to create a flutter around the veins. Why are 2 gaps necessary? If there is only 1 gap, the line is still good because conduction can go into the vein, and unless there is extraordinarily slow conduction inside the vein creating the reentry within the vein itself, it will not be arrhythmogenic in terms of flutter. Thus, sealing one gap eliminates the flutter, and sealing the second causes conduction block into the vein. If more than 2 gaps are present, all but 1 will need to be sealed to eliminate the flutter, assuming the line itself and the other anchor point are definite. Because of this possibility, some operators approach left atrial flutter after atrial fibrillation ablation by first making sure the pulmonary veins are isolated; thus eliminating the possibility of proarrhythmic flutter from gaps in the encircling pulmonary vein ablation lines and ensuring that ablation lines anchored to the encircling lines will create conduction block.

Summary

Miyazaki et al present a highly instructive case representative of an increasingly common scenario presenting to interventional electrophysiologists today. A careful study of their discussion, figures, and the key points from the case itself will help electrophysiologists, and especially the trainee, in approaching ablation of this increasingly common and difficult arrhythmia.
How to Approach Reentrant Atrial Tachycardia After Atrial Fibrillation Ablation
Shinsuke Miyazaki, Ashok J. Shah, Atsushi Kobori, Taishi Kuwahara and Atsushi Takahashi

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