Pulmonary vein (PV) isolation is a cornerstone of atrial fibrillation (AF) ablation. This technique is now widely spread all over the world. Numerous studies have demonstrated that complete PV isolation with circular lesions has a better outcome in patients with paroxysmal and persistent AF. In contrast, it may lead to iatrogenic left atrial tachycardia (AT) as an adverse effect of the ablation. Three mechanisms of AT can develop after AF ablation, including macro-reentrant AT, focal AT and PV tachycardia. AT after AF ablation is predominantly related to the arrhythmogenicity of the PVs, lesions created by the ablation procedure and damaged atrial tissue from the persistent tachyarrhythmias. The 3-dimensional maps play an important role in clarifying the mechanism of the tachycardia and the optimal ablation site for ATs. (Circ J 2010; 74: 1051–1058)

Key Words: Atrial fibrillation; Atrial tachycardia; Electro-anatomical mapping; Pulmonary vein

Macro-AT

The incidence of regular macro-AT was reported to be 4–40% after PV isolation. The incidence depends on the type of AF, LA diameter, and ablation technique. The 3-D mapping can demonstrate a relatively large reentrant circuit and critical isthmus in these ATs. Entrainment studies found an identical post-pacing interval to the AT cycle length at several different sites in the atrium. The most common macro-AT after PV isolation is atrial flutter around the mitral annulus (Figure 2), followed by roof-dependent AT (Figure 3), and PV–left atrial appendage (LAA) ridge-dependent AT (Figure 4). These ATs utilize regions of incomplete or recovered ablation lesions or anatomical obstacles as the critical isthmuses to sustain the arrhythmia. The creation of conduction block at the tachycardia isthmus by RF deliveries can terminate the tachycardia. These data suggested that macro-AT is an iatrogenic tachycardia due to large circumferential and incomplete ablation lesions.

The identification of the critical isthmus of macro-ATs is...
Figure 1. Schematic illustration of the 3 mechanisms of atrial tachycardia (AT) developing after pulmonary vein (PV) isolation. First, large macro-reentrant tachycardias have been observed such as mitral flutter (Left). Second, focal tachycardia had been observed to originate from the left atrium (LA) near the previous circumferential lesions around the PVs (Middle). Third, The PV tachycardias originating from the PVs within a previously isolated area conducted to the LA through a reconnection gap between the LA and PV (Right).

Figure 2. (A) Surface 12-lead electrocardiogram (ECG) during atrial tachycardia (AT) that developed after a pulmonary vein isolation for persistent atrial fibrillation. (B) Activation map of the left atrium using electro-anatomical mapping (CARTO). The activation duration (234 ms) observed with the CARTO system was almost equal to the tachycardia cycle length (240 ms), indicating that this tachycardia was a macro-reentrant tachycardia and the reentrant circuit turned around the mitral annulus (MA) in a counter-clockwise fashion. Irrigated radiofrequency delivery (red dots) was applied between the left inferior pulmonary vein (LIPV) and MA, and successfully terminated the AT and created bi-directional block between the LIPV and MA. AP, antero-posterior.
relatively easy based on electro-anatomical mapping. A few applications can eliminate most types of ATs, but catheter ablation of atrial flutter around the mitral annulus (mitral flutter) is sometimes challenging. In most cases, creation of a block line is attempted between the left inferior PV and mitral annulus. Complete block of the mitral isthmus has not always been completed even when using irrigated RF energy and epicardial RF applications within the coronary sinus (CS). This combined epicardial and endocardial approach allows for a variable success rate (76–92%) of achieving bidirectional isthmus block, resulting in a higher cost of the risk of complications—including cardiac tamponade and damage to the circumflex artery.

Previous studies have demonstrated that the shape and depth of the atrial myocardium vary greatly around the mitral isthmus and that the depth of the tissue may be the limiting factor in achieving bidirectional block by endocardial ablation alone. Also, the presence of blood flow in the coronary vessels may act as a heat sink, thereby preventing transmural RF lesion formation.

**Focal AT**

Focal tachycardia has a focal origin and the activation of this tachycardia spreads in a concentric manner to the atrium. The development of focal AT originates from around the PVs and is most likely related to the ablation procedure and arrhythmogeneity of the PV antrum. The slow conduction around the PV ostia or antrum is related to the prior ablation procedure. It has been also reported that the myocardium within the PVs in patients with AF has exhibited short effective refractory periods and decremental conduction, which favors the occurrence of reentry around the PVs (Figure 5).

In contrast, some ATs are generally focally driven arrhythmias that are either unmasked following the AF ablation or arise from segments of atrial or venous tissue, that is, the crista terminalis, superior vena cava or CS. This form of tachycardia is uncoupled from the surrounding atrial myocardium by previous RF applications.

**PV Tachycardia**

PV tachycardia is another type of arrhythmia demonstrating an identical atrial activation sequence and P wave morphology (Figure 6). This type of AT may be seen transiently a few weeks following the AF ablation with a repetitive form and may resolve without intervention. This AT typically originates from incomplete lines of ablation and the focal mechanism of the AT is predominantly related to the reconnection of the conduction from PV to the LA.

This tachycardia originates from the myocardium within the PVs and activates the LA through a recovered conduction gap with 1-to-1 or Wenckebach conduction after the PV isolation. Therefore an identical P wave morphology and intracardiac atrial activation sequence are observed even during different cycle lengths in some cases. In such cases the PV activity is faster than the atrial activity due to the slow

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**Figure 3.** (A) Surface 12-lead electrocardiogram (ECG) during atrial tachycardia (AT) that developed after a pulmonary vein (PV) isolation for paroxysmal atrial fibrillation. (B) Activation map of the left atrium using electro-anatomical mapping (CARTO) demonstrating that the isthmus of this AT was in the region between the previous circumferential ablation lines around both the left and right PVs. Both the left and right PVs were completely isolated during the second session and play a role as an anatomical obstacle for the reentrant circuit of the AT. A few radiofrequency applications (red dots) between both the right and left PVs terminated the AT, and no AT could be induced thereafter with programmed stimulation.
Figure 4.  (A) Surface 12-lead electrocardiogram (ECG) during an atrial tachycardia (AT) induced after a pulmonary vein (PV) isolation of paroxysmal atrial fibrillation. (B) Activation map of the left atrium using electro-anatomical mapping (CARTO) during the tachycardia demonstrates that the isthmus of this AT was located between the left-sided PVs and left atrial appendage (LAA), the so called “ridge”. One radiofrequency application (the red dot) between the left superior PV and LAA terminated the AT and no further AT could be induced thereafter by programmed stimulation. MA, mitral annulus.

Figure 5. Activation map of the left atrium using electro-anatomical mapping (CARTO) during an atrial tachycardia that developed after pulmonary isolation exhibiting a centrifugal activation pattern. The earliest activation site of the AT is an anterior site near the right superior pulmonary vein. AP, anteroposterior; PA, posteroanterior.
Figure 6. (A) Surface electrocardiogram (ECG) leads II and V1, and the intracardiac recordings from an ablation catheter (Mp) and diagnostic catheters inside the left superior pulmonary vein (LPV) and coronary sinus (CS) during atrial tachycardia (AT) that developed during pulmonary vein (PV) isolation of persistent atrial fibrillation. Note that (1) the tachycardia persisted within the PV with a cycle length of 220 ms (asterisk); (2) the left atrium (LA) is passively activated with 2-to-1 conduction through a conduction gap between the PV and LA (Mp d or Cs d). (B) The AT was terminated by radiofrequency application at the conduction gap and the surface ECG shows that sinus rhythm was restored; the tachycardia within the PV (PV tachycardia) spontaneously terminated after achievement of the PV isolation. This phenomenon indicated that a PV tachycardia was the dominant tachycardia maintaining this AT. The PV tachycardia itself, however, required both PV and LA conduction in order to persist.
Figure 7. (A) Surface 12-lead surface electrocardiogram (ECG) leads (Left), and intracardiac recordings from an ablation catheter (Mp) and diagnostic catheters inside the left superior pulmonary vein (LSPV), left inferior pulmonary vein (LIPV), and coronary sinus (CS) at the termination of the tachycardia (Right). Note that (1) the tachycardia was terminated by the application at the anterior–superior region of the LSPV; (2) the tachycardia was terminated just after the left atrium (LA) potential with conduction block from the LA to the PV by radiofrequency (RF) application (arrow); this shows that the ablation catheter was located at the entrance of the PV; and (3) PV conduction with a significant delay (▼) was still present after termination of the tachycardia. The earliest activation site of the PV was at LSPV 1-2 during the tachycardia (asterisk). After the termination of the tachycardia, the earliest site of the PV activation moved from LSPV 1-2 to RIPV 8-9 (▼). This phenomenon indicates that there are different entrance sites to the left PV (assumed conduction gaps in previous circular lesions) during the tachycardia and sinus rhythm (SR). (B) The 3-dimensional reconstruction of the LA and left-sided PVs in posteroanterior view (Left). The PV ostium is tagged in white. The activation propagates from the posterior region of the left PV to the LA and back to the anterior region of the left PV in a figure of 8. Green dots, site with a post-pacing interval (PPI) identical to that of the tachycardia (ATCL). Site with a brown dot located in the left superior–anterior region along the PV ostium indicates the region of the tachycardia termination, and the other brown dot in the anterior–superior region of the PV indicates the site of the isolation of the left and right PVs (Right). Schematic illustration of the activation around the PVs during the tachycardia and SR after the termination of the tachycardia. The reentrant circuit is assumed to enter the PV at one site and exit at another site, thus including both the PV and LA, which are connected by 2 conduction gaps between the PV and LA. This tachycardia can be terminated by RF delivery at either the entrance or exit site. The PV activation sequence between that of the tachycardia and SR differed when the RF energy was delivered at the entrance site.
conduction of the conduction gap in the previous circumferential lesions. The tachycardia recovers sinus rhythm after block of the conduction gap is created between the LA and PV and sometimes persists within the PV independent of the atrial activation.

**LA-PV Reentrant Tachycardia**

Recently a new form of reentrant tachycardia after PV isolation has been reported. This reentrant circuit included the myocardium within the PV and LA via 2 conduction gaps in a previous circumferential lesion, which were widely separated (Figure 7A). This tachycardia developed within 1 month after the initial circumferential PV isolation, which is similar to the previous findings that these macro-ATs generally develop 1–2 months after ablation. The critical isthmus was located within the PV and LA myocardium via the 2 conduction gaps in the previous circumferential lesion and the RF delivery easily terminated each conduction gap (Figure 7B).

The 3-D mapping sometimes demonstrated focal tachycardia along previous circumferential lesions. Centrifugal activation can be mimicked by activation from the exit site of the slow conduction isthmus of a macro-reentrant circuit, especially when high-density mapping is not performed or the exit site from the reentry circuit is within a low-voltage area. Therefore this tachycardia could potentially be misdiagnosed as focal AT without entrainment guided by a Lasso catheter within the PV.

### AT After Termination of Long-Standing Persistent AF

An extensive ablation consisting of a PV isolation, linear ablation, and electrogram-based ablation is effective in eliminating the fibrillatory substrate of long-standing persistent AF, persisting more than 1 year. ATs, however, often occur after the procedure or after termination of AF by RF application. The mechanism of which has been shown to be macro-reentry, focal AT or localized reentry. ATs were documented within 3 months in 40% of patients who underwent a stepwise catheter ablation. Interestingly, the anatomical distribution of the focal ATs was located in the superior base of the LAA, CS ostium, and inside the CS.

In most patients with persistent AF, ablation is performed in the atria in order to target an extensive arrhythmogenic substrate. It is known that non-transmural lesions can cause slow conduction, thereby serving as a reentry substrate. Also, the atria are being remodeled progressively during persistent AF. Therefore these patients sometimes have multiple ATs and it is difficult to identify and eliminate all the circuits of the ATs.

### Conclusions

AT after AF ablation is predominantly related to the lesions created by the ablation procedure, and damaged atrial tissue due to persistent tachyarrhythmias. The 3-D maps play an important role in identifying the mechanisms of tachycardias and the optimal ablation site during stable tachycardias. Damaged atrial tissue with remodeling of the atrium due to long-standing persistent tachycardia evokes complicated ATs and requires newly developed mapping and ablation tools.

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### References


