Recovered Pulmonary Vein Conduction as a Dominant Factor for Recurrent Atrial Tachyarrhythmias After Complete Circular Isolation of the Pulmonary Veins
Lessons From Double Lasso Technique

Feifan Ouyang, MD; Matthias Antz, MD; Sabine Ernst, MD; Hitoshi Hachiya, MD; Hercules Mavrakis, MD; Florian T. Deger, MD; Anselm Schaumann, MD; Julian Chun, MD; Peter Falk, MD; Detlef Hennig; Xingpeng Liu, MD; Dietmar Bänsch, MD; Karl-Heinz Kuck, MD

Background—Atrial tachyarrhythmias (ATa) can recur after continuous circular lesions (CCLs) around the ipsilateral pulmonary veins (PVs) in patients with atrial fibrillation (AF). This study characterizes the electrophysiological findings in patients with and without ATa after complete PV isolation.

Methods and Results—Twenty-nine of 100 patients had recurrent ATa after complete PV isolation by use of CCLs during a mean follow-up of 8 months. A repeat procedure was performed in 26 patients with ATa and in 7 volunteers without ATa at 3 to 4 months after CCLs. No recovered PV conduction was demonstrated in the 7 volunteers, whereas recovered PV conduction was found in 21 patients with recurrent ATa (right-sided PVs in 9 patients and left-sided PVs in 16 patients). The interval from the onset of the P wave to the earliest PV spike was 157±66 ms in the right-sided PVs and 149±45 ms in the left-sided PVs. During the procedure, PV tachycardia activated the atrium and resulted in atrial tachycardia (AT) in 10 patients. All conduction gaps were successfully closed with segmental RF ablation. After PV isolation, macroreentrant AT was induced and ablated in 3 patients. In the 5 patients without PV conduction, focal AT in the left atrial roof in 2 patients and non-PV foci in the left atrium in 1 patient were successfully abolished; in the remaining 2 patients, no ablation was performed because of noninducible arrhythmias. During a mean follow-up of 6 months, 24 patients were free of ATa without antiarrhythmic drugs.

Conclusions—In patients with recurrent ATa after CCLs, recovered PV conduction is a dominant finding in 8% of patients and can be successfully eliminated by segmental RF ablation. Also, mapping and ablation of non-PV arrhythmias can improve clinical success. (Circulation. 2005;111:127-135.)

Key Words: atrium | arrhythmia | ablation | mapping | tachyarrhythmias

Recent studies have demonstrated that atrial fibrillation (AF) could be cured by continuous circular lesions (CCLs) around the pulmonary veins (PVs) guided by 3D mapping in the majority of patients.1–4 We have recently demonstrated that all PVs could be completely isolated from the atrium by CCLs around the ipsilateral PVs guided by 3D mapping and double Lasso technique.4 However, data on atrial tachyarrhythmias (ATa) after complete PV isolation by use of CCLs are very limited. In particular, the importance of long-term completeness of CCLs is still under debate. In the present study, we prospectively investigated electrophysiological findings after complete PV isolation in patients with and without recurrent ATa.

Methods

Patient Characteristics
One hundred highly symptomatic AF patients (88 with paroxysmal AF and 12 with persistent AF) underwent complete PV isolation by use of CCLs around the ipsilateral PVs guided by 3D mapping and double Lasso technique. All patients were kept on the previously ineffective antiarrhythmic drugs, which were discontinued after the first month if no recurrent ATa occurred after the initial ablation procedure. The ablation protocol consisted solely of 2 CCLs around the ipsilateral PVs. Structural heart disease had been diagnosed in 18 of 100 patients (coronary artery disease in 11 patients, aortic valve disease in 4 patients, hypertrophic obstructive cardiomyopathy in 2 patients, and idiopathic dilated cardiomyopathy in 1 patient).

Twenty-nine patients (25 patients with paroxysmal AF and 4 patients with persistent AF before the initial CCL) had recurrent ATa after ablation during a follow-up of 238±87 days (range, 123 to 325 days). Three patients had infrequent short episodes of recurrent ATa after the initial CCL and refused to undergo a second procedure. A repeat procedure was performed in only 26 patients with recurrent ATa (18 male; mean age, 60±9 years) at 52±59 days (range, 2 to 193 days) after the initial procedure. The patient cohort included 23 patients with paroxysmal AF and 3 patients with persistent AF before the initial procedure. The early and late recurrences were defined by an interval of less than 2 weeks or more than 2 weeks after the initial
ablation procedure, respectively. There were 21 patients with early recurrent ATa (range, 1 to 13 days) and 5 patients with late recurrent ATa (range, 30 to 135 days) (Table). All 26 patients experienced highly symptomatic ATa unresponsive to antiarrhythmic drugs. Among the 26 patients, coronary artery disease had been diagnosed in 4. The mean left atrial (LA) diameter was 44 mm (range, 35 to 63 mm). In addition, a repeat study was performed 3 to 4 months after the initial ablation procedure in 7 volunteers (5 male; mean age, 57 years; 6 with paroxysmal AF and 1 with persistent AF) out of 71 patients without ATa recurrence. The institutional review board of St Georg General Hospital approved the study protocol.

Electrophysiological Study

All patients provided written informed consent. The ablation procedure was performed with the patient taking the previous antiarrhythmic drugs under sedation with a continuous infusion of propofol. Two standard catheters were positioned: a 6F (Biosense-Webster, Inc) at the His bundle region via a femoral vein and a multipolar electrode 6F catheter in the coronary sinus (CS) via the left subclavian vein. Three 8F SL1 sheaths (St Jude Medical, Inc) were advanced to the LA via the patent previously punctured site in patients with early recurrent ATa or by use of a modified Brockenbrough technique in the patients with late recurrent ATa. In the control group, a single 8F sheath was advanced to the LA for mapping PV spikes. After transseptal catheterization, intravenous heparin was administered to maintain an activated clotting time of 250 to 300 seconds. Also, continuous infusions with heparinized saline were connected to the transseptal sheaths (flow rate of 10 mL/h) to avoid thrombus formation or air embolism.

Mapping and Catheter Ablation

Selective PV angiography was performed to evaluate all PV ostia. After PV angiography, 2 decapolar Lasso catheters (Biosense-Webster) were placed within the ipsilateral superior and inferior PVs or within the superior and inferior branches of a common PV to confirm whether there was recovered PV conduction. In the case of AF, sinus rhythm (SR) was restored by external cardioversion. If discrete PV spikes were recorded in the ipsilateral PVs, a 3.5-mm-tip

<table>
<thead>
<tr>
<th>Patient</th>
<th>Arrhythmias Before CCL</th>
<th>Arrhythmias After CCL</th>
<th>Time of Recurrent AT Before CCL, d</th>
<th>Time of Repeat Procedure After CCL, d</th>
<th>P-PV Interval in Right-Sided PVs Before CCL, ms</th>
<th>P-PV Interval in Left-Sided PVs Before CCL, ms</th>
<th>PV Tachycardia Location</th>
<th>Cycle Length, ms</th>
</tr>
</thead>
<tbody>
<tr>
<td>1*</td>
<td>Paroxysmal AF</td>
<td>AT</td>
<td>135</td>
<td>193</td>
<td>69</td>
<td>132</td>
<td>LCPV</td>
<td>170–215</td>
</tr>
<tr>
<td>2*</td>
<td>Paroxysmal AF</td>
<td>Persistent</td>
<td>42</td>
<td>90</td>
<td>128</td>
<td>187</td>
<td>LCPV</td>
<td>165–260</td>
</tr>
<tr>
<td>3*</td>
<td>Paroxysmal AF</td>
<td>AT</td>
<td>120</td>
<td>170</td>
<td>60</td>
<td>262</td>
<td>LIPV</td>
<td>110–280</td>
</tr>
<tr>
<td>4</td>
<td>Paroxysmal AF</td>
<td>Paroxysmal</td>
<td>3</td>
<td>150</td>
<td>103</td>
<td>124</td>
<td>LIPV</td>
<td>110–280</td>
</tr>
<tr>
<td>5</td>
<td>Paroxysmal AF</td>
<td>AT</td>
<td>1</td>
<td>48</td>
<td>78</td>
<td>178</td>
<td>LCPV</td>
<td>190–230</td>
</tr>
<tr>
<td>6</td>
<td>Paroxysmal AF</td>
<td>AT</td>
<td>2</td>
<td>133</td>
<td>156†</td>
<td>175</td>
<td>LSPV</td>
<td>136–138</td>
</tr>
<tr>
<td>7*</td>
<td>Paroxysmal AF</td>
<td>AT</td>
<td>30</td>
<td>92</td>
<td>60</td>
<td>147</td>
<td>LCPV</td>
<td>170–450</td>
</tr>
<tr>
<td>8*</td>
<td>Paroxysmal AF</td>
<td>AT</td>
<td>67</td>
<td>70</td>
<td>103</td>
<td>171</td>
<td>LSPV</td>
<td>170–450</td>
</tr>
<tr>
<td>9</td>
<td>Paroxysmal AF</td>
<td>AT</td>
<td>1</td>
<td>6</td>
<td>110</td>
<td>128</td>
<td>LCPV</td>
<td>165–260</td>
</tr>
<tr>
<td>10</td>
<td>Paroxysmal AF</td>
<td>AT</td>
<td>2</td>
<td>5</td>
<td>75</td>
<td>112</td>
<td>LIPV</td>
<td>110–280</td>
</tr>
<tr>
<td>11</td>
<td>Paroxysmal AF</td>
<td>AT</td>
<td>3</td>
<td>2</td>
<td>82</td>
<td>250</td>
<td>LCPV</td>
<td>110–280</td>
</tr>
<tr>
<td>12</td>
<td>Paroxysmal AF</td>
<td>AT</td>
<td>13</td>
<td>17</td>
<td>75</td>
<td>106</td>
<td>LIPV</td>
<td>190–230</td>
</tr>
<tr>
<td>13</td>
<td>Paroxysmal AF</td>
<td>AT</td>
<td>2</td>
<td>7</td>
<td>119</td>
<td>229</td>
<td>LIPV</td>
<td>120–375</td>
</tr>
<tr>
<td>14</td>
<td>Paroxysmal AF</td>
<td>AT</td>
<td>1</td>
<td>4</td>
<td>119</td>
<td>229</td>
<td>LIPV</td>
<td>110–280</td>
</tr>
<tr>
<td>15</td>
<td>Paroxysmal AF</td>
<td>AT</td>
<td>1</td>
<td>7</td>
<td>119</td>
<td>229</td>
<td>LIPV</td>
<td>110–280</td>
</tr>
<tr>
<td>16</td>
<td>Paroxysmal AF</td>
<td>AT</td>
<td>2</td>
<td>12</td>
<td>67</td>
<td>148</td>
<td>LCPV</td>
<td>170–450</td>
</tr>
<tr>
<td>17</td>
<td>Persistent AF</td>
<td>Persistent</td>
<td>2</td>
<td>12</td>
<td>67</td>
<td>148</td>
<td>LCPV</td>
<td>170–450</td>
</tr>
<tr>
<td>18</td>
<td>Persistent AF</td>
<td>Persistent</td>
<td>2</td>
<td>117</td>
<td>69</td>
<td>238</td>
<td>LCPV</td>
<td>170–450</td>
</tr>
<tr>
<td>19</td>
<td>Persistent AF</td>
<td>Persistent</td>
<td>10</td>
<td>77</td>
<td>70</td>
<td>169</td>
<td>LCPV</td>
<td>170–450</td>
</tr>
<tr>
<td>20</td>
<td>Paroxysmal AF</td>
<td>AT</td>
<td>3</td>
<td>53</td>
<td>71</td>
<td>104</td>
<td>LCPV</td>
<td>260</td>
</tr>
<tr>
<td>21</td>
<td>Paroxysmal AF</td>
<td>AT</td>
<td>13</td>
<td>34</td>
<td>85</td>
<td>110</td>
<td>LCPV</td>
<td>260</td>
</tr>
<tr>
<td>22</td>
<td>Paroxysmal AF</td>
<td>AT</td>
<td>1</td>
<td>7</td>
<td>62</td>
<td>79</td>
<td>LCPV</td>
<td>217–248</td>
</tr>
<tr>
<td>23</td>
<td>Paroxysmal AF</td>
<td>AT</td>
<td>13</td>
<td>35</td>
<td>38</td>
<td>56</td>
<td>LSPV</td>
<td>200–260</td>
</tr>
<tr>
<td>24</td>
<td>Paroxysmal AF</td>
<td>AT</td>
<td>2</td>
<td>7</td>
<td>70</td>
<td>155</td>
<td>LIPV</td>
<td>200–260</td>
</tr>
<tr>
<td>25</td>
<td>Paroxysmal AF</td>
<td>AT</td>
<td>8</td>
<td>13</td>
<td>92</td>
<td>142</td>
<td>LIPV</td>
<td>200–260</td>
</tr>
<tr>
<td>26</td>
<td>Paroxysmal AF</td>
<td>AT</td>
<td>1</td>
<td>2</td>
<td>92</td>
<td>142</td>
<td>LIPV</td>
<td>200–260</td>
</tr>
</tbody>
</table>

*Patient with late recurrent atrial tachyarrhythmias.
†Interval after segmental PV ostial isolation.

Clinical and Mapping Data in Patients With Recurrent Atrial Tachyarrhythmias After CCL

<table>
<thead>
<tr>
<th>Patient</th>
<th>Arrhythmias Before CCL</th>
<th>Arrhythmias After CCL</th>
<th>Time of Recurrent AT Before CCL, d</th>
<th>Time of Repeat Procedure After CCL, d</th>
<th>P-PV Interval in Right-Sided PVs Before CCL, ms</th>
<th>P-PV Interval in Left-Sided PVs Before CCL, ms</th>
<th>PV Tachycardia Location</th>
<th>Cycle Length, ms</th>
</tr>
</thead>
</table>
catheter (Thermo-Cool, Biosense Webster) was advanced close to the site with the earliest PV spikes and was then withdrawn to the LA and rotated to the area near the earliest PV spikes during SR. An additional conduction gap was considered if the PV activation sequence changed after one conduction gap had been closed. 3D mapping with a 3.5-mm-tip catheter (Thermo-Cool Navi-Star, Biosense Webster) was used to identify conduction gaps. The location of conduction gaps was arbitrarily defined as the roof, anterosuperior, anteroinferior, inferior, posteroinferior, or posterosuperior region of the previous CCL. In the control group, one decapolar Lasso catheter (Biosense-Webster) was placed within all PVs to check whether there was recovered PV conduction during SR.

Irrigated RF energy was delivered as previously described by use of a target temperature of 45°C, a maximal power limit of 40 W, and an infusion rate of 17 mL/min. RF energy was applied in the conduction gaps of the previous CCL or the critical part of non-PV atrial tachycardia (AT).

The end point of the CCLs was defined as (1) the absence of all PV spikes documented with the 2 Lasso catheters within the ipsilateral superior and inferior PVs at least 30 minutes after isolation, (2) no recurrence of the PV spikes within all PVs after intravenous administration of 9 to 12 mg of adenosine during SR, and (3) no inducible AT after ablation.

Postablation Care and Follow-Up
After the procedure, intravenous heparin was administered for 3 days, followed by warfarin for at least 3 months in all patients. All patients continued the previously ineffective antiarrhythmic drugs for 1 month after the ablation. A surface ECG, transthoracic echocardiography, and 24-hour Holter recording were performed 1 day after the procedure and repeated after 1, 3, 6, and 12 months by the referring physician or in the ablation center. All patients had a telemetry ECG recorder (Philips Telemedizin) for 6 months to document symptomatic arrhythmias or to transfer a 30-second ECG once per week if asymptomatic.

Statistical Analysis
All values are expressed as mean±SD and/or range. Mean changes of the interval from the onset of the P wave to the earliest PV spikes before and after CCLs were compared by use of the Wilcoxon test. Mean values of the LA diameter between patients with AT and AF were compared by use of the Mann-Whitney test. A probability value of P<0.05 was considered significant.

Results
Before the initial CCLs, 25 of the 26 patients presented with AF alone, and patient 19 presented with AF and left macro-reentrant AT (Macro-AT), which was identified and dissociated from CCLs during the initial procedure. Twenty-one patients presented with only AT and 5 patients presented with AF after the initial procedure. The clinical arrhythmias before and after the initial CCL are shown in the Table. The LA dimension in 5 patients with AF was significantly larger than that in 21 patients with AT (52.8±5.9 versus 42.1±4.3 mm, P<0.01). During the repeat procedure, no PV narrowing was demonstrated in all patients with AT and in the 7 volunteers. In 4 patients with persistent AF, recovered PV spikes during SR were demonstrated after external cardioversion.

Recovered PV Conduction During SR
During the procedure, recovered PV conduction was observed in 17 of 21 patients (81%) with early recurrent AT and in 4 of 5 patients (80%) with late recurrent AT (Table). Recovered PV conduction was also observed in 17 of 21 patients (81%) with recurrent AT and in 4 of 5 patients (80%) with recurrent AF (Table). Recovered PV conduction was found only in the right-sided PVs in 5 patients, only in the
left-sided PVs in 12 patients, and in the left-sided and right-sided PVs in 4 patients. During SR, the recovered PV spikes were widely separated from the far-field atrial activation (Figure 1); the interval from the onset of the P wave to the earliest PV spike was significantly longer than that at baseline before CCL (157 ± 66 versus 71 ± 24 ms in the right-sided PVs; 149 ± 45 versus 92 ± 25 ms in the left-sided PVs; \( P < 0.01 \)). In the 5 patients with only recovered right-sided PV spikes, pacing within the PV resulted in fast conduction into the LA via the recovered conduction gaps despite a significant delay (Figure 1).

The conduction gaps were identified by use of 3D mapping and double Lasso in the initial 8 patients and only by double Lasso in the last 13 patients. The locations of all 32 conduction gaps are shown in Figure 2. The electrograms at the successful site presented as fractionated potentials in 19, double potentials in 11, and a single potential in 2 conduction gaps. All electrograms were of low amplitude without PV spikes, except in 3 anterior gaps with tiny “far-field” PV spikes. All conduction gaps were successfully closed with segmental RF applications. After PV isolation, PV spikes recurred in the right-sided PVs in patient 23 after intravenous administration of 12 mg adenosine. After further RF applications, the PV spikes did not reappear after a repeat intravenous administration of adenosine. The mean RF duration was 393 ± 176 seconds for closing the conduction gaps in the previous left CCL and 405 ± 205 seconds for closing those in the previous right CCL. In the 7 volunteers, no recovered PV conduction was demonstrated during SR.

**Figure 3.** Tracings A to D are ECG leads II, III, and V1 and intracardiac electrograms recorded from 2 Lasso catheters within left superior and inferior PVs (LSPV, LIPV), a mapping catheter (Map), and a catheter inside CS in patient 24, with incessant AT during procedure. A, Tachycardia originates from LIPV with a CL of 120 to 130 ms and activates LSPV with 3:2 or 2:1 conduction. LA activation is propagated from LIPV via conduction gaps. B, After a spontaneous change in CL of tachycardia, there are CL changes with alternating short and long CL and 1:1 conduction between left-sided PVs. PV spikes in LSPV follow spikes in LIPV with a fixed interval (marked by PV), which indicates passive activation in LSPV. Atrial activation recorded from CS (marked by A) is relatively stable because of decremental conduction from LIPV to LA via a conduction gap. C, Fluoroscopic right and left anterior oblique views (left) show mapping catheter in inferior gap of previous CCL, 2 Lasso catheters within LSPV and LIPV, a catheter within CS, and a catheter at His bundle region (His). Note that P-wave morphology during PVT changes to a different P-wave morphology (marked by asterisk) when irrigated RF energy is delivered in inferior gap during second RF application. D, Fluoroscopic right and left anterior oblique views (left) show mapping catheter in inferoposterior gap of previous CCL, 2 Lasso catheters within LSPV and LIPV, a catheter within CS, and a catheter at His bundle region (His). Note that tachycardia terminates when fourth RF delivery is applied in posteroinferior region of previous CCL.

**PV Tachycardia Responsible for Clinical AT During the Procedure**

During the procedure, a spontaneous or catheter-induced PV tachycardia (PVT) activated the atria, resulting in clinical AT in 10 patients who had only documented AF before the initial CCL. The cycle length (CL) and location of the PVTs are also shown in the Table. There were 2 patients with relatively constant CL and 8 patients with a variable CL during PVT. These PVTs were located within the left common PV in 4, the left superior PV (LSPV) in 3 and the left inferior PV (LIPV) in 3 patients. These PVTs presented as a repetitive pattern in 8 patients and an incessant pattern in 2 patients during the procedure. The surface ECG showed a constant P-wave morphology in 9 patients and 2 different morphologies in 1 patient.

In patient 24, with incessant PVT, after 2 Lasso catheters were placed within the left-sided PVs, mapping demonstrated that the tachycardia from the LIPV propagated into the LSPV with alternating Wenckebach 3:2 or 2:1 conduction and activated the atrium with an irregular CL via conduction gap.
During continuous recording, the tachycardia CL spontaneously prolonged with alternating short and long CLs, which resulted in 1:1 conduction from the LIPV to the LSPV and relatively stable CL in the atrial activation because of decremental conduction through the conduction gap (Figure 3B). Interestingly, RF delivery in the inferior gap resulted in a change of P-wave morphology, which indicated LA activation via a different conduction gap of the previous left CCL (Figure 3C). Further RF delivery in the inferoposterior gap terminated the tachycardia (Figure 3D). After the termination, discrete PV spikes after the P wave were observed in both PVs during SR and were successfully abolished by a single RF application in the third conduction gap located in the inferoanterior part of the previous CCL.

In patient 22, with catheter-induced incessant PVT within the LSPV, the tachycardia propagated into the LIPV with 1:1 or 2:1 conduction and into the LA with 2:1 conduction (Figure 4A). A single RF application closed the anterosuperior gap of the previous CCL, resulting in SR and no change of PVT within the LSPV (Figure 4B). Burst pacing from the mapping catheter within the isolated LSPV was unable to terminate the tachycardia, which was finally terminated by external cardioversion with 200 J (Figure 4C) 30 minutes after complete PV isolation. After the cardioversion, recovered PV conduction was not found in the left-sided PVs.

(Figure 3A). Other Atrial Arrhythmias

After complete PV isolation, 3D mapping demonstrated that clinical Macro-AT presented with an isthmus in the left posterior wall constrained between the 2 CCLs in patient 2 and with an isthmus between the left CCL and the lateral mitral annulus in patient 20. Clinical Macro-AT in the right free wall was also induced in patient 22. All Macro-ATs were successfully ablated with a linear lesion across the isthmus.

In the 5 patients without recovered PV conduction (Table), mapping demonstrated that focal AT originated from the LA roof in patients 8 and 16. Both ATs had repetitive patterns with an earliest atrial activation preceding the onset of the P wave by 34 and 31 ms, respectively. Interestingly, patient 16 presented with AT and dissociated repetitive fast PVT within the isolated right-sided PVs (Figure 5, A and B). Both tachycardias were successfully abolished by 2 to 6 irrigated RF applications at the site with the earliest atrial activation. In patient 4, with recurrent paroxysmal AF, intravenous isoproterenol provoked a short episode of AF initiated by atrial extrasystoles, which were located in a tiny area (1 cm anterior to the lateral PVs) and successfully abolished by a linear lesion from this area to the lateral mitral annulus. In patients 15 and 25, with recurrence of documented AT, no AT was initiated by programmed stimulation and intravenous isopro-
terenol during the procedure. No ablation was performed in those 2 patients.

**Follow-Up**

No complications occurred in all 26 patients with ATa and in the 7 volunteers. No recurrence of ATa was observed in the 7 volunteers in a follow-up period of 164±17 days (range, 132 to 184 days). During a follow-up of 175±55 days (range, 100 to 264 days), no ATa recurred in 20 patients after reconducting PVs had been isolated; short episodes of recurrent AF occurred infrequently within the first 3 months after the ablation and later disappeared spontaneously in patient 10, with recovered PV conduction. In the 5 patients without recovered PV conduction, no ATa recurred after ablation in patients 4, 8, and 16; in the 2 remaining patients, AT resolved spontaneously immediately after the procedure in patient 25.
and 1 month after the procedure in patient 15. In summary, after repeat procedures, no episode of ATa recurred in 84 of 88 patients (95.5%) with paroxysmal AF and 11 of 12 patients (91.7%) with persistent AF before CCL. The procedure time was 183 ± 58 minutes, with a fluoroscopy time of 23.1 ± 13.2 minutes.

**Discussion**

The present study describes electrophysiological findings after CCLs: (1) recovered PV conduction in patients with and without recurrent ATa, (2) identification and segmental ablation of the conduction gaps, (3) CCL-induced change of AF substrate in the LA, and (4) non-PV ATa.

**Recovered PV Conduction in Patients With and Without Recurrent ATa After CCLs**

Recovered PV spikes have been demonstrated in almost all patients with recurrent AF after segmental PV ostial isolation, even in asymptomatic patients on antiarrhythmic drugs after successful isolation. However, published data about the incidence of recovered PV conduction are very limited in symptomatic patients after complete PV isolation by use of CCLs and are not available at all in asymptomatic patients after complete PV isolation by use of CCLs. In the present study, no PV conduction was demonstrated in the 7 volunteers without antiarrhythmic drugs 3 to 4 months after the CCLs, whereas recovered PV conduction in 21 patients with recurrent ATa after CCLs was demonstrated in the left-sided PVs in 16 patients and in the right-sided PVs in 9.

An interesting finding in this study was that the recovered PV spikes presented as discrete potentials during SR. They were widely separated from the far-field atrial potentials and were easily identified. The interval from the onset of the P wave to the earliest recovered PV spike was significantly longer compared with baseline before CCLs. However, pacing within the PV and PVT could rapidly activate the atria via the conduction gaps. Twenty patients were free of ATa after the recovered PVs had been isolated. These data strongly suggest that only complete and permanent PV isolation but not incomplete isolation with delayed activation can prevent ATa recurrence in the majority of patients with paroxysmal and persistent AF.

**Identification of Conduction Gaps in the Previous CCLs**

In the present study, the conduction gaps were identified by use of a 3D mapping system and double Lasso technique in
the initial 8 patients and only by double Lasso technique in the last 13 patients. All conduction gaps were individually distributed in the previous CCLs. In our experience, local electrograms of the conduction gaps presented as low amplitude with fragmented or double potentials in most patients. The most important proof of the conduction gap in the previous CCLs was successful isolation of the reconducting PVs with a few RF applications. This result represents a nearly perfect paradigm of incomplete CCLs around the ipsilateral PVS that can be completed by specifically targeted segmental lesions. Therefore, this information can provide a simple online method to identify the conduction gaps by use of only double Lasso catheters in patients undergoing CCLs around the ipsilateral PVS. Our data are not in accordance with a previous study, which suggested that PV isolation by CCLs may not be necessary to prevent paroxysmal AF recurrence. However, the inability to capture the LA while pacing within the PV after CCLs and an amplitude <0.1 mV within the CCLs are no established criteria for PV isolation in AF ablation by use of a single transseptal approach and no Lasso catheter within the PV.

**CCL-Induced Change of AF Substrate in the LA**

Previous studies have demonstrated that CCLs in the left posterior wall may result in atrial electroanatomic remodeling by eliminating triggered activity and/or mother waves near the PV ostium that drive AF, isolating relatively large areas that are not available for perpetuating AF, as well as vagal denervation. In the present study, double Lasso catheters within ipsilateral PVSs demonstrated that a fast PVT with very irregular CL activates the atria via decremental conduction gaps. These PVTs clinically resulted in a focal AT with constant P-wave morphology and the same atrial activation sequence in 10 patients with only AF before CCLs. PVTs with variable CL are similar to focal ATs with irregular CL in the LA roof or the base of the LA appendage near the PV ostia, which were mentioned in a recent study. However, the authors did not provide an anatomic location of the PV ostia by use of selective PV angiography or used a Lasso catheter within the PV to record low-amplitude PV spikes. This methodology may underestimate the true incidence of PVTs. Also, the 10 patients after CCLs clinically resemble the group of patients previously described as a focal source of AF, but several features differentiate them. In the present study, all patients had consistently presented as having AF before CCLs. On the basis of our experience with the double Lasso catheter technique, understanding the PVT can facilitate catheter ablation in patients with recurrent AT and after CCLs.

In 2 patients with incessant PVT, mapping showed that PVTs are most likely because of a reentrant mechanism, because (1) PVT within the isolated LSPV is terminated by DC cardioversion only after the conduction gap has been closed in patient 22, and (2) the atrial myocardium may be involved in the PVT mechanism, on the basis of the fact that RF delivery in the posterior wall terminated the tachycardia from LIPV before complete isolation of left-sided PVSs in patient 24. This observation is consistent with the findings of Oral and coworkers that atrial input is often required for intermittent PVT perpetuation. On the basis of previously published data and our findings from the repeat procedure, all facts strongly suggest that previous CCLs in the posterior LA eliminate the substrate of multiple reentry critical for AF perpetuation and allow PVT to result in focal AT via conduction gaps with a decremental conduction property in patients with nearly normal LA.

In the present study, an important electrophysiological finding during the repeat procedure was the recovered PV conduction demonstrated in 81% of patients with recurrent AT and in 80% of patients with recurrent AF. Also, complete PV isolation prevented recurrent ATs in 16 of 17 patients with recurrent AT and in 4 of 4 patients with recurrent AF. Our data strongly support the notion that recurrent AF and AT share similar mechanisms after complete PV isolation by use of only CCLs around the ipsilateral PVSs. Conversely, the 5 patients with recurrent AF after CCLs had enlarged LAs. This suggests that some additional lines in the enlarged LA may be necessary to prevent AF perpetuation.

**Non-PV ATa**

Previous studies have demonstrated that Macro-AT can coexist with AF or occur after CCLs. In the present study, clinical Macro-AT was documented in 3 patients with recovered PV conduction after CCLs. 3D mapping demonstrated a critical isthmus dissociated from CCLs in 1 patient and associated with CCLs in the LA in the other 2 patients. All tachycardias were successfully ablated by linear lesions by use of 3D mapping. Conversely, focal AT can recur even after complete PV isolation. These ATs were located in the left roof in 2 patients and in the LA anterior to the left-sided PVSs in 1 patient. All ATs were successfully abolished by focal or linear lesions. These data support the fact that previous linear lesions performed in the left roof and in the left isthmus can improve clinical outcome. However, we recommend that reablation be performed only in patients with recurrent ATa, because the incidence of focal ATa from the roof and left isthmus is very low in our series.

**Study Limitations**

This study has several limitations, as follows. (1) The number of volunteers is small. Our data do not prove that all asymptomatic patients have no recovered PV conduction after CCLs. (2) The recurrence rate after the initial procedure was 29% in our series, and it might have been lower if the reablation procedure had been performed more than 4 weeks after the initial ablation. (3) Although our patients had highly symptomatic AF before the initial CCLs and asymptomatic short episodes of paroxysmal AF were not documented, it is very difficult to rule out the occurrence of asymptomatic AF in these patients. (4) A small number of persistent AF patients were included in this study. However, no ATa recurred in 11 of 12 patients (91.7%), including 3 patients with a repeat procedure during follow-up. This information strongly supports the proposition that complete PV isolation by CCLs can prevent AF recurrence in patients with persistent AF.

**Conclusions**

Recovered PV conduction is a dominant finding in approximately 80% of patients with recurrent ATa after complete PV isolation by CCLs and could be successfully eliminated by...
segmental RF applications. Complete and permanent PV isolation by CCLs can provide high success rate in patients with paroxysmal and potentially in patients with persistent AF. Also, mapping and ablation of non-PV ATa can improve clinical outcome in patients without recovered PV conduction.

References
Recovered Pulmonary Vein Conduction as a Dominant Factor for Recurrent Atrial Tachyarrhythmias After Complete Circular Isolation of the Pulmonary Veins: Lessons From Double Lasso Technique

Feifan Ouyang, Matthias Antz, Sabine Ernst, Hitoshi Hachiya, Hercules Mavrakis, Florian T. Deger, Anselm Schaumann, Julian Chun, Peter Falk, Detlef Hennig, Xingpeng Liu, Dietmar Bänsch and Karl-Heinz Kuck

_Circulation_. 2005;111:127-135; originally published online December 27, 2004; doi: 10.1161/01.CIR.0000151289.73085.36

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2004 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/111/2/127

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Circulation_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Circulation_ is online at:
http://circ.ahajournals.org//subscriptions/