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Pulmonary Vein Stenosis After Catheter Ablation of Atrial Fibrillation

Ivan M. Robbins, MD; Edward V. Colvin, MD; Thomas P. Doyle, MD; W. Evans Kemp, MD; James E. Loyd, MD; William S. McMahan, MD; G. Neal Kay, MD

Background—This report describes the complication of pulmonary vein stenosis with resultant severe pulmonary hypertension that developed in 2 patients after successful catheter ablation of chronic atrial fibrillation.

Methods and Results—Three months after successful catheter ablation of atrial fibrillation, both patients developed progressive dyspnea and pulmonary hypertension. Both were found to have severe stenosis of all 4 pulmonary veins near the junction with the left atrium. Balloon dilation of the stenotic pulmonary veins was performed in these patients, with improvement in dyspnea and pulmonary hypertension.

Conclusions—The complication of pulmonary vein stenosis is potentially life-threatening, and the application of radiofrequency current within the pulmonary veins with standard catheter technology should be avoided. This complication can be treated with balloon dilation, although the long-term course is unknown. (*Circulation*. 1998;98:1769-1775.)

Key Words: stenosis ■ catheter ablation ■ fibrillation

Although catheter ablation of atrial fibrillation (AF) has been demonstrated to be feasible, the long-term complications and efficacy of this technique are unknown.¹⁻⁵ Successful interruption of chronic AF usually involves application of radiofrequency current within the left atrium (LA), linking the ostia of the pulmonary veins (PVs) to the mitral annulus.¹ This report describes the complication of PV stenosis resulting in severe pulmonary hypertension that developed in 2 patients after successful catheter ablation of chronic AF. The clinical course and treatment of this complication are reported.

Case Summaries

Both patients in this report were included in a multicenter feasibility trial of catheter ablation for the treatment of chronic AF. Among 18 patients enrolled in the trial, 2 patients developed a similar clinical course, characterized by progressive dyspnea and pulmonary hypertension. None of the other patients have experienced similar symptoms or developed increased pulmonary artery (PA) pressures by repeated Doppler echocardiography at 1 month, 3 months, and 6 months after catheter ablation. The clinical courses of the 2 patients who have developed pulmonary hypertension are detailed below.

Case 1

A 53-year-old man with chronic AF refractory to antiarrhythmic medications and repeated cardioversion was referred for enrollment in an investigational protocol for catheter ablation

of AF. The patient had experienced intermittent AF for 12 years, which had become chronic for the preceding 3 years (Figure 1).

After having given written, informed consent, the patient was enrolled in a multicenter protocol for catheter ablation of AF that had been approved by the Institutional Review Board for Research Involving Human Subjects at the University of Alabama at Birmingham. Echocardiography demonstrated normal left ventricular function, with an LA dimension of 38 mm and mild mitral and tricuspid regurgitation and an estimated PA systolic pressure by Doppler echocardiography of 39 mm Hg. On April 23, 1997, the patient underwent catheter ablation under general anesthesia. Standard transeptal puncture was performed with a Brockenbrough needle and a 13F outer sheath and 11F inner sheath (Daig Corp). The activated clotting time was maintained at >300 seconds with heparin. Catheter ablation involved application of radiofrequency current within both atria to create 5 lines of conduction block. The radiofrequency current was applied with a target temperature of 55°C as the ablation catheter was gradually withdrawn in increments of 2 to 3 mm every 30 seconds. The ablation lines were (1) from the right upper PV to the left upper PV, which was extended to the mitral annulus; (2) from just to the left of the posterior interatrial septum 1 cm above the mitral annulus to the ostium of the right superior PV; (3) diagonally across the roof of the LA, starting anteriorly along the interatrial septum and continued to the left superior PV; (4) from the superior vena cava to the inferior vena cava along the posterior wall of the right atrium;

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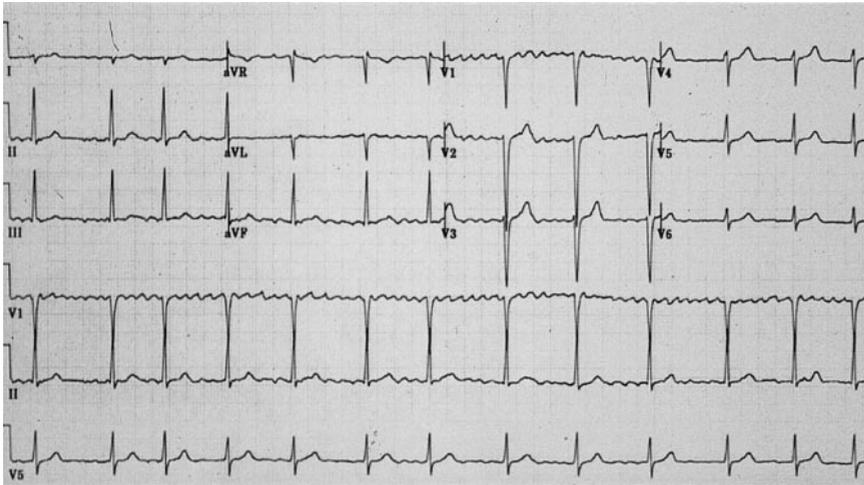


Figure 1. 12-lead ECG before ablation, demonstrating chronic AF.

and (5) from the tricuspid annulus to the Eustachian ridge, as used for ablation of typical atrial flutter. The total procedural duration was 8.0 hours. During the ablation session, the intracardiac electrograms in AF became progressively more organized, with lengthening of the average atrial cycle length and widening of the isoelectric segments in the right atrial and LA electrograms and conversion to normal sinus rhythm. The patient developed typical atrial flutter in June 1997 that was successfully ablated with radiofrequency applications in the isthmus between the tricuspid valve and the Eustachian ridge. AF has not recurred in follow-up of >12 months (Figure 2).

In July 1997, the patient noted the onset of progressive dyspnea on exertion and nonproductive cough. The ECG demonstrated normal sinus rhythm throughout the postoperative course (Figure 2). An echocardiogram performed on July 21, 1997, demonstrated an estimated PA systolic pressure of 65 mm Hg, and physical examination demonstrated an accentuated pulmonic valve closure sound. Pulmonary function tests were normal. The patient underwent right heart catheterization on September 18, 1997, with a PA pressure of 60/24 mm Hg (mean, 41 mm Hg), a pulmonary capillary wedge pressure (PCWP) of 16 mm Hg, cardiac output of 7.0

L/min, and a pulmonary vascular resistance (PVR) of 287 dynes \cdot cm²/s. Bilateral pulmonary arteriograms showed no evidence of thrombus but diffuse pruning of the small pulmonary arteries and a delayed transit time through the lungs to the LA. The patient experienced no improvement with intravenous prostacyclin (epoprostenol) or oral amlodipine. Because of worsening dyspnea on exertion and radiographic evidence of pulmonary edema, the patient was reevaluated with transesophageal echocardiography on December 12, 1997, which demonstrated an estimated PA systolic pressure of 88 mm Hg and high-velocity turbulence (2.5 m/s) within the LA near the ostia of both superior PVs, suggesting PV stenosis.

On December 18, 1997, the patient underwent right and left heart catheterization by the transeptal technique. The PA pressure was 98/36 mm Hg (mean, 58 mm Hg), with an LA pressure of 23 mm Hg, cardiac output of 4.78 L/min, and PVR of 586 dynes \cdot cm²/s. Selective cannulation of each of the 4 PV ostia and venography were performed. All 4 of the PVs demonstrated localized stenosis within 10 mm from the ostium draining into the LA. The left superior PV was severely stenotic \approx 10 mm from the ostium, with a mean pressure of 42 mm Hg and a simultaneously recorded LA

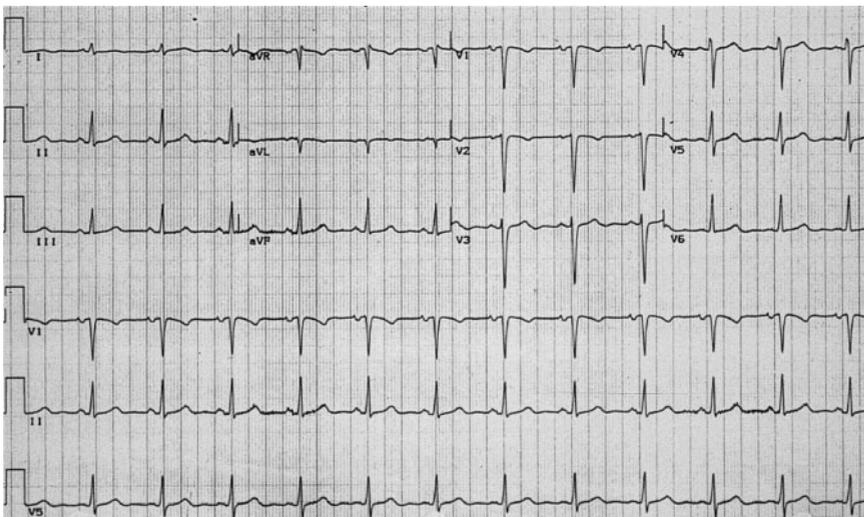


Figure 2. 12-lead ECG 8 months after catheter ablation.

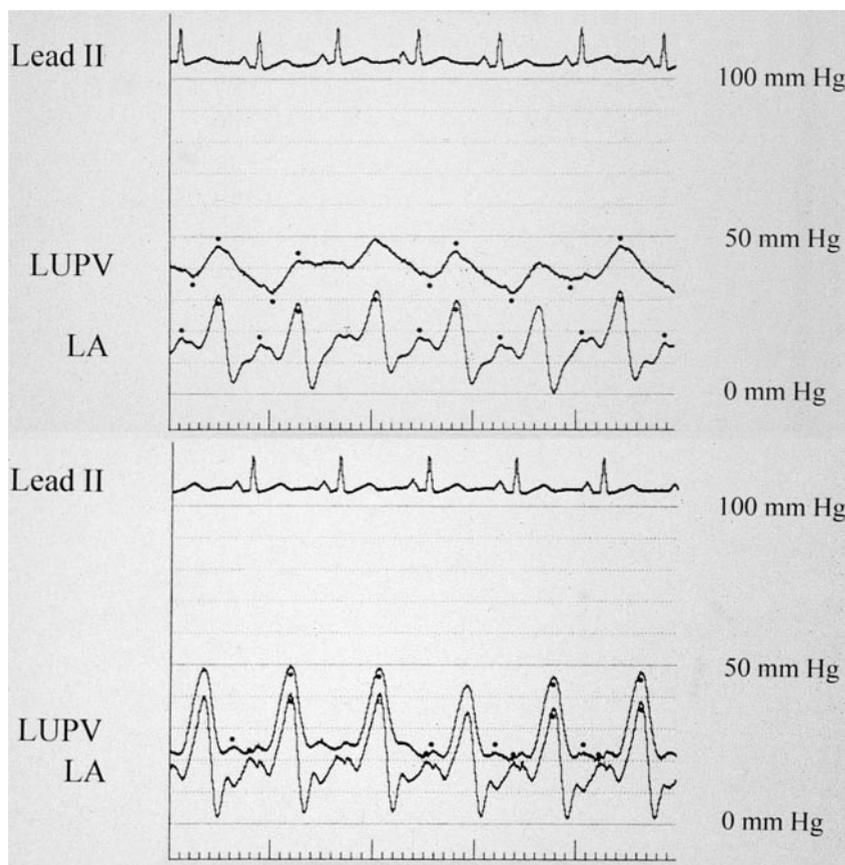


Figure 3. Top, Simultaneously recorded pressure tracings from left upper PV (LUPV) and LA. Note that mean LA pressure is 17 mm Hg, with mean LUPV pressure of 42 mm Hg. Bottom, After balloon dilation, mean pressure in LA is 21 mm Hg, with mean pressure in LUPV of 25 mm Hg.

pressure of 17 mm Hg (Figure 3A). Venous dilatation was performed with a 6-mm-diameter balloon at an inflation pressure of 16 atm, which resulted in a decrease in the gradient between the left superior PV and the LA to 5 mm Hg (Figure 3B and Figure 4). The ostium of the left superior PV was sequentially dilated with balloons having diameters of 8 and 10 mm. Balloon dilation was performed to relieve stenoses within all 4 PVs (Figures 5 through 7). At the conclusion of the procedure, the PA pressure had decreased to 60/24 mm Hg, with an LA pressure of 15 mm Hg and a PVR of 557 dynes \cdot cm²/s.

The patient noted an immediate improvement in his dyspnea and was discharged 5 days after catheterization. He was reevaluated at a clinic visit on January 2, 1998, and reported marked improvement in his dyspnea on exertion. Right heart catheterization was performed on January 2, 1998, and demonstrated a PA pressure of 54/24 mm Hg, with PCWP of 16 mm Hg, cardiac output of 5.9 L/min, and PVR of 293 dynes \cdot cm²/s.

Case 2

The second patient is a 36-year-old woman with paroxysmal AF since age 17 years that had been refractory to flecainide, propafenone, disopyramide, sotalol, dofetilide, β -blockers, calcium blockers, and digoxin. Despite multiple electrical cardioversions, AF became chronic and associated with marked fatigue, dyspnea, and palpitations. Transthoracic and transesophageal echocardiograms showed an estimated PA systolic pressure of 28 mm Hg and a mildly dilated right ventricle, with no evidence of intracardiac shunting.

After informed, written consent to the investigational protocol, she underwent radiofrequency catheter ablation by the same technique as described above. The total procedural duration was 12.5 hours. The patient developed atypical atrial flutter and returned for repeat ablation on December 31, 1996. An echocardiogram before the procedure demonstrated an estimated PA systolic pressure of 38 mm Hg. Catheter ablation was performed in the usual isthmus of tissue between the tricuspid annulus and the Eustachian ridge, with reversion to sinus rhythm. AF was induced with programmed electrical stimulation, and a linear application of radiofrequency current was delivered from the superior vena cava to the inferior vena cava along the posterior wall of the right atrium. AF reverted to normal sinus rhythm during this application and was no longer inducible with up to 4 extrastimuli. An LA flutter was then induced with programmed electrical stimulation, which was mapped and ablated in the region between the left inferior PV and the mitral annulus. She has maintained sinus rhythm for >1 year after this procedure.

Immediately after ablation, the patient complained of a persistent, nonproductive cough. Over the next several months, she gradually developed progressive dyspnea on exertion that did not respond to inhaled corticosteroids or bronchodilators. An echocardiogram on June 20, 1997, demonstrated an estimated PA systolic pressure of 85 mm Hg, with a dilated right ventricle. A radionuclide ventilation-perfusion lung scan demonstrated nearly complete absence of perfusion to the right lung and left lower lobe, with a normal pattern of ventilation. Right heart catheterization was per-

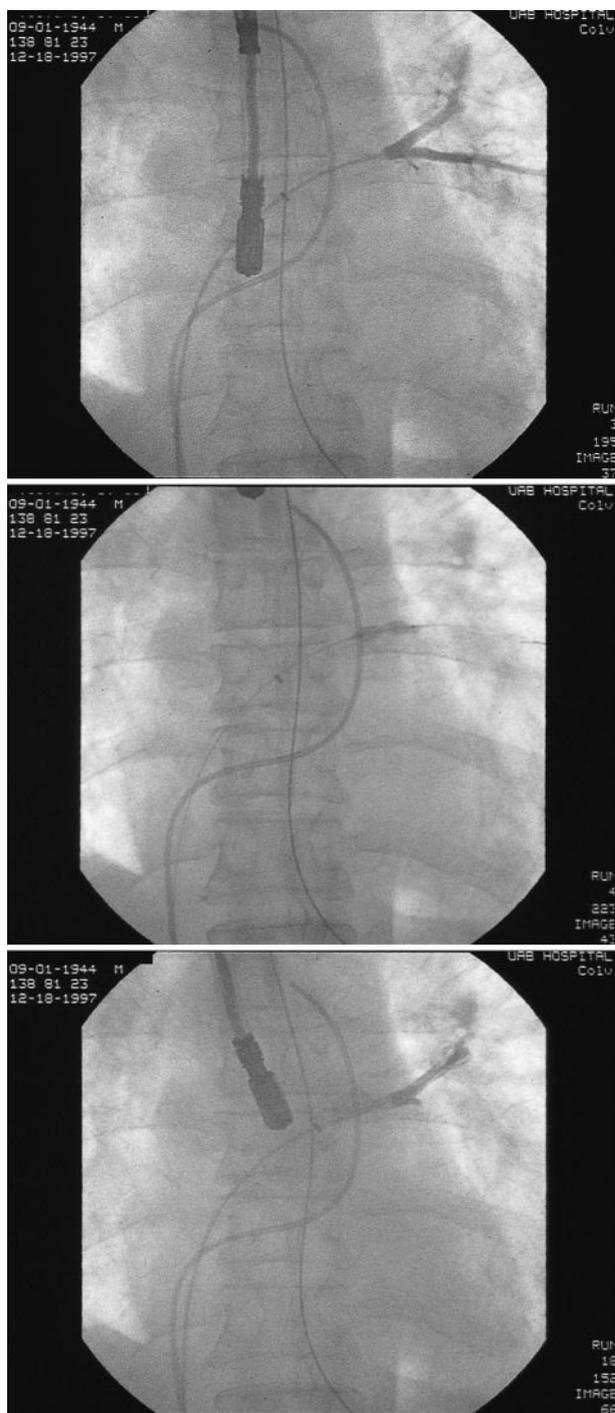


Figure 4. Top, Contrast injection into left superior PV. Note localized stenosis of vein with absence of flow into LA. Middle, A 6-mm-diameter balloon is inflated within left upper PV. Bottom, Contrast injection into left superior PV after balloon inflation, demonstrating resolution of stenosis.

formed on June 21, 1997, with a PA pressure of 81/25 mm Hg (mean, 54 mm Hg), PCWP of 16 mm Hg, PVR of 512 dynes \cdot cm²/s, and cardiac index of 3.1 L \cdot min⁻¹ \cdot m⁻². Bilateral pulmonary arteriograms demonstrated no thromboemboli but pruning of the distal pulmonary vascular bed, with delayed transit of contrast through the lungs.

On September 12, 1997, the patient was reevaluated with right heart catheterization, which demonstrated a PA pressure

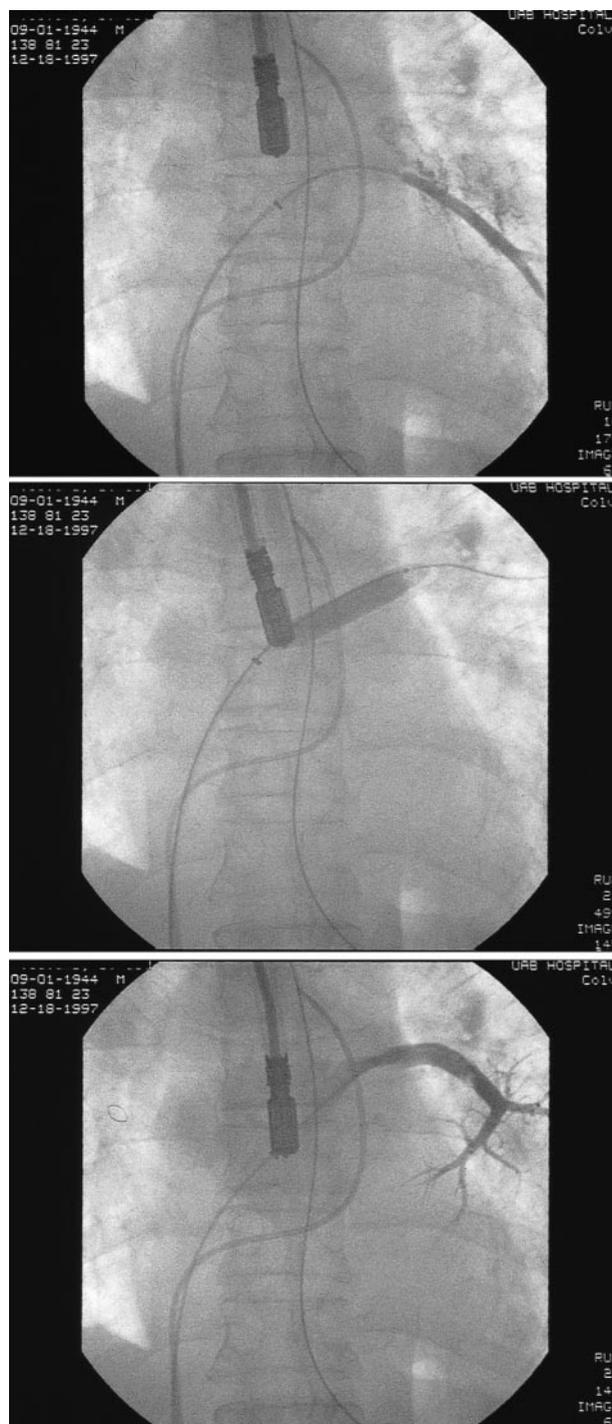


Figure 5. Top, Contrast injection into left inferior PV. Note localized stenosis of PV. Middle, A 10-mm-diameter balloon is inflated within ostium of vein. Bottom, Contrast injection into left inferior PV after balloon inflation, demonstrating mild focal stenosis.

of 99/46 mm Hg (mean, 64 mm Hg), PVR of 1152 dynes \cdot cm²/s, and cardiac output of 3.5 L/min. Inhaled nitric oxide decreased the PA pressure to 75/36 mm Hg (mean, 52 mm Hg) and the PVR to 600 dynes \cdot cm²/s. Intravenous prostacyclin (epoprostenol) afforded minimal improvement in her dyspnea. A transesophageal echocardiogram was performed in November 1997 and demonstrated high-

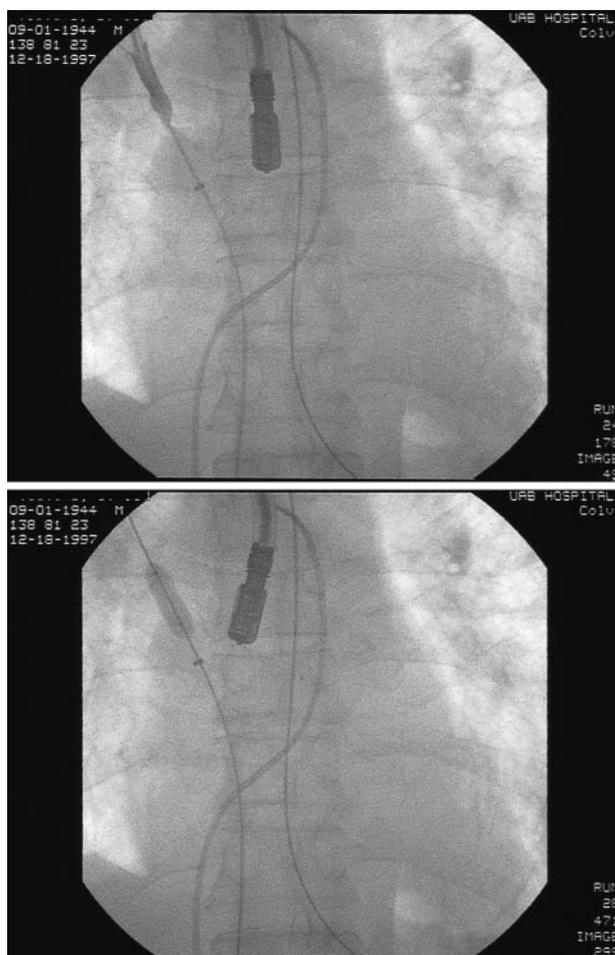


Figure 6. Top, Contrast injection into right superior PV with localized stenosis. Bottom, An 8-mm-diameter balloon is inflated within ostium of PV.

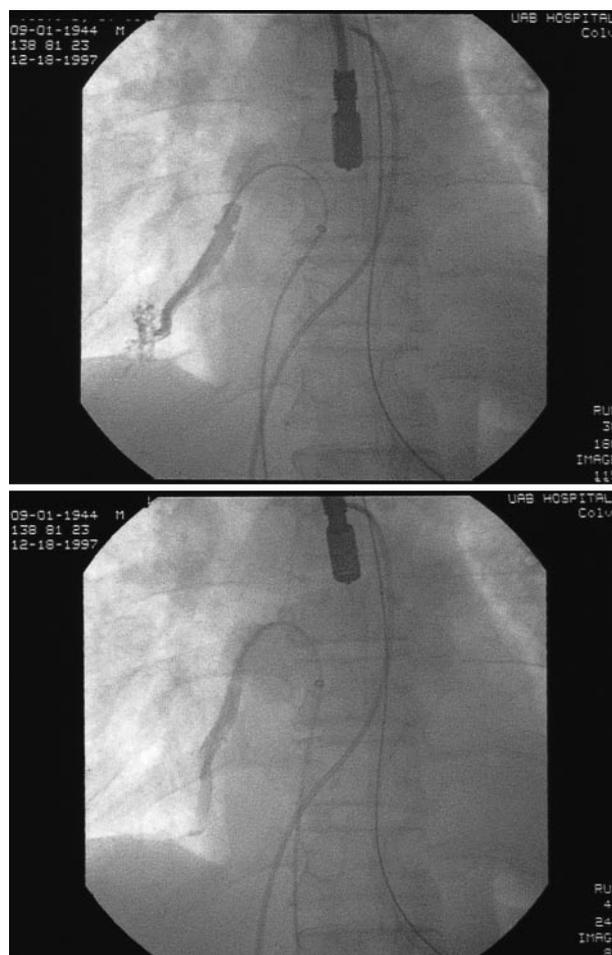


Figure 7. Top, Contrast injection into right inferior PV with localized stenosis. Bottom, Contrast injection into right inferior PV after balloon inflation, demonstrating resolution of stenosis.

velocity turbulence within the LA at the junction of both superior PVs. Repeat right heart catheterization was performed on December 15, 1997, and demonstrated a PA pressure of 90/50 mm Hg (mean, 62 mm Hg), LA pressure of 3 mm Hg, and cardiac index of $1.9 \text{ L} \cdot \text{min}^{-1} \cdot \text{m}^{-2}$. Selective wedge pulmonary arteriograms demonstrated severe stenosis of the left superior and inferior PVs and the right superior PV near their junction with the LA. The right lower PV was occluded.

Transseptal catheterization and balloon dilation of the PVs was performed on January 15, 1998. Direct cannulation of the right superior, left superior, and left inferior PVs demonstrated gradients to the LA of 21, 16, and 33 mm Hg, respectively. Balloon dilation of the 2 superior PVs was performed with 12-mm-diameter balloons, and an 8-mm balloon was used to dilate the left inferior PV (Figures 8 through 10). The gradients between the PVs and the LA decreased to 3, 4, and 0 mm Hg, and the PA systolic pressure decreased to 55 mm Hg, with marked improvement in her symptoms.

Discussion

Catheter ablation of AF is in its infancy. However, the early experience with this procedure has shown that chronic AF

can be interrupted by the application of radiofrequency current within the LA in most patients, although some patients require additional applications in the right atrium.¹⁻⁵ The complications of this procedure as performed with standard catheters have been substantial, including pericardial effusion, systemic emboli, pulmonary dysfunction, and bleeding from the intense anticoagulation that is required.¹⁻⁵ This report demonstrates that PV stenosis is an additional, potentially severe complication of this procedure. During the present multicenter trial of catheter ablation for chronic AF, PV stenosis has been identified in 2 of 18 patients undergoing this procedure.

The mechanism of PV stenosis after radiofrequency ablation in our patients probably involves scarring and contraction of the venous wall as a result of thermal injury. In an analogous situation, thrombosis or stenosis of the coronary sinus has been observed after radiofrequency catheter ablation of accessory pathways.^{6,7} Whereas stenosis or occlusion of the coronary sinus is usually not associated with clinical sequelae, stenosis of a PV may produce dyspnea on exertion, orthopnea, cough, hemoptysis, and recurrent pulmonary infections. If untreated, congenital PV stenosis is usually fatal, with most patients succumbing to right heart failure.

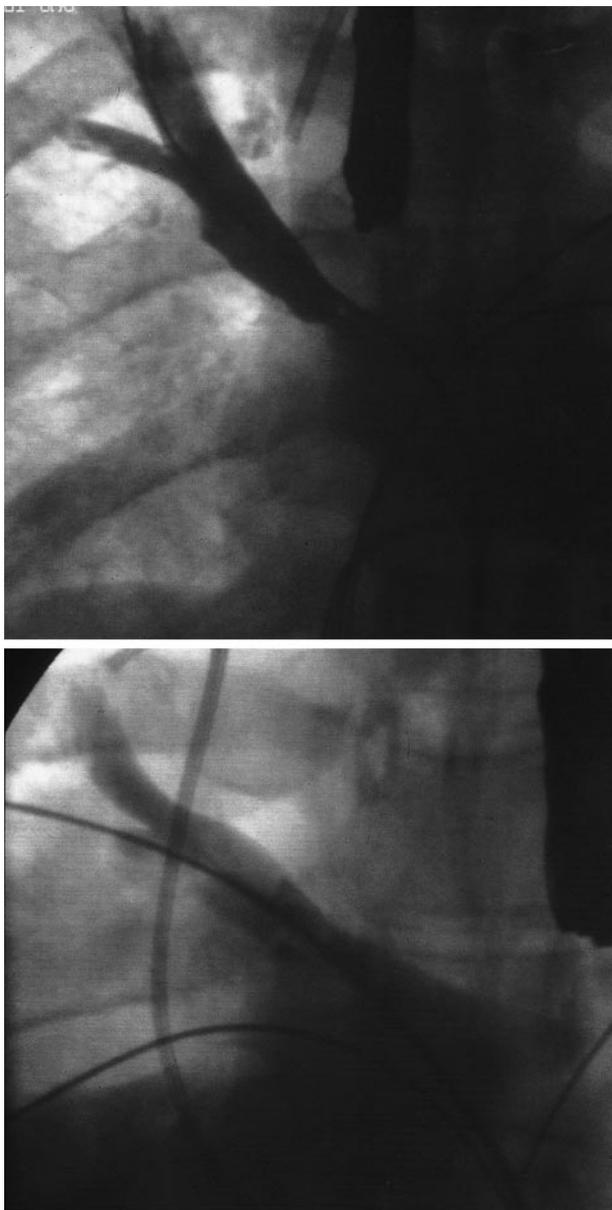


Figure 8. Top, Contrast injection into right superior PV in patient 2, with stenosis at junction of PV with LA. Bottom, After balloon dilation with a 12-mm balloon, there is resolution of stenosis.

The treatment of PV stenosis has included surgical excision of the localized stenosis,⁸ reimplantation of the PVs with direct anastomosis to the LA appendage or LA,⁹ balloon angioplasty,¹⁰ or endovascular stenting.^{11,12} The use of balloon angioplasty in congenital PV stenosis has been generally unsatisfactory, with a high incidence of restenosis and recurrent pulmonary hypertension.¹⁰ Although experience with stenting of the PVs in congenital stenosis is limited, the results have also been generally disappointing.^{11,12} Whether acquired PV stenosis demonstrates a similar course after balloon dilatation has not been determined, although PVs that are not congenitally malformed may respond better to angioplasty and stenting.¹³

The present report raises several concerns regarding catheter ablation of chronic AF. First, the exact incidence of PV stenosis

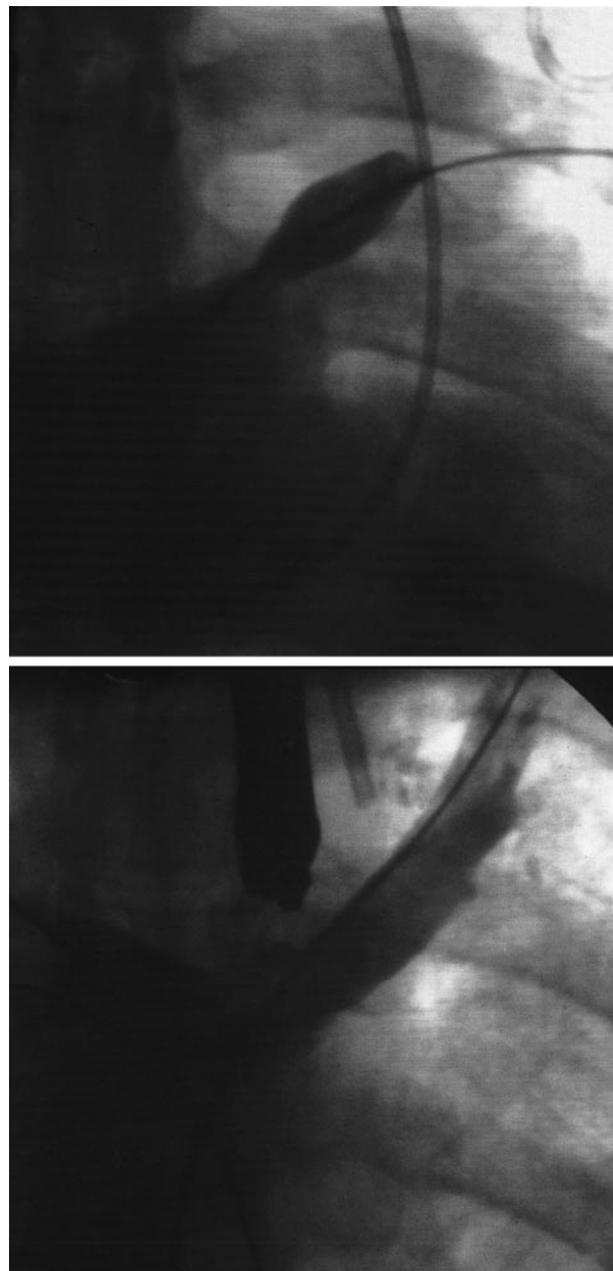


Figure 9. Top, Balloon inflation in ostium of left superior PV in patient 2. Note severe stenosis at junction of PV with LA, as demonstrated by waist on 12-mm-diameter balloon. Bottom, Contrast injection into left superior PV after balloon dilation, with resolution of stenosis.

after catheter ablation is unknown. However, it is unlikely that pulmonary hypertension would develop unless a substantial portion of the pulmonary venous drainage is affected. If only a single PV becomes obstructed, the clinical manifestations may be mild or unrecognized. Second, on the basis of the location of the PV stenoses observed in these patients, radiofrequency current probably should not be applied within a PV. Rather, the ablation lesion should probably be confined to the LA or end at the PV ostium. The consequences of not extending the ablation line into the PV on the maintenance of AF are unknown, although it is possible that reentrant LA arrhythmias may be promoted by such an approach. Third, the development of

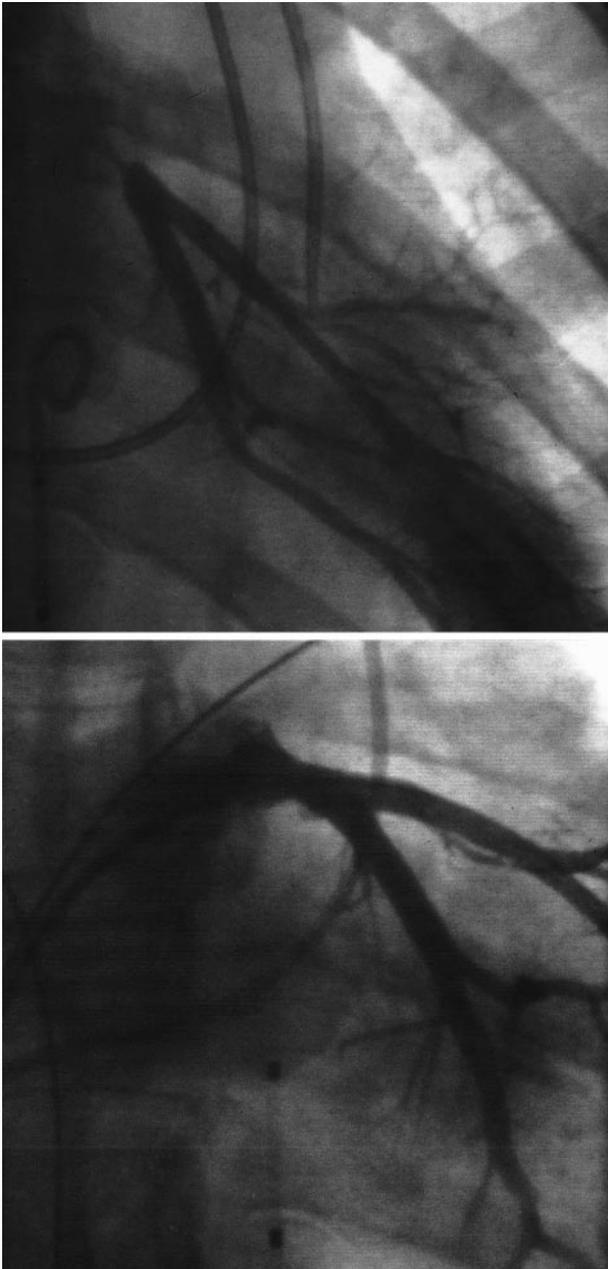


Figure 10. Top, Contrast injection into left inferior PV in patient 2. Note severe stenosis of PV \approx 1 cm from its junction with LA. Bottom, Contrast injection into left inferior PV after balloon dilation with an 8-mm balloon, with resolution of stenosis.

dyspnea or cough after catheter ablation in the region of the PVs should raise suspicion of PV stenosis. Finally, this complication can be treated acutely with balloon dilation of the PVs, although the long-term outcome is uncertain.

Conclusions

This report demonstrates that PV stenosis may occur after catheter ablation of AF, leading to severe pulmonary hypertension. Future ablation strategies must prevent damage to the PVs.

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