



Lehigh Valley Procedure: Combined Pulmonary Vein Isolation and Accessory Pathway Ablation for Atrial Fibrillation in Wolff- Parkinson-White Syndrome

CASE REPORT

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ABSTRACT

Wolff-Parkinson-White (WPW) syndrome is a congenital condition characterized by the presence of an accessory pathway (AP) that can lead to tachyarrhythmias such as atrial fibrillation (AF). Patients with WPW are at an increased risk of developing AF, with a prevalence of up to 30%. Despite successful ablation of the AP, AF recurrence remains a challenge, particularly in older patients or those with a history of AF. Concomitant pulmonary vein isolation (PVI) may help reduce the risk of recurrence by targeting atrial remodeling and pulmonary vein involvement in AF. This case describes a 70-year-old male with WPW and AF who underwent a combined PVI and AP ablation procedure. The patient remained free of AF post-procedure, suggesting that this approach may be effective in high-risk patients. Further randomized controlled trials are necessary to evaluate the efficacy of this combined approach in reducing AF recurrence compared to AP ablation alone, especially in older patients.

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KEYWORDS:

Wolff-Parkinson-White;
pulmonary vein isolation;
accessory pathway ablation;
atrial fibrillation

TO CITE THIS ARTICLE:

Sabri MS, Benedict L, Souder D, Kaugars L, Patel K, Bozorgnia B, Nazir T. Lehigh Valley Procedure: Combined Pulmonary Vein Isolation and Accessory Pathway Ablation for Atrial Fibrillation in Wolff-Parkinson-White Syndrome. Methodist DeBaKey Cardiovasc J. 2025;21(1):74-80.doi:[10.14797/mdcvj.1618](https://doi.org/10.14797/mdcvj.1618)

INTRODUCTION

Wolff-Parkinson-White (WPW) syndrome is a congenital condition affecting between 0.1% and 0.3% of the population and characterized by the presence of an accessory pathway (AP), distinctive electrocardiogram features, and tachyarrhythmias.¹ Patients with preexcitation are at an increased risk for developing atrial fibrillation (AF), with a prevalence of 30% in those with WPW.² While AP ablation reduces AF inducibility, it does not eliminate the risk of recurrence, and patients who undergo radiofrequency ablation (RFA) are at higher risk for future AF, particularly if they are over 50 years old or have a history of AF prior to the procedure.³ Therefore, concomitant pulmonary vein isolation (PVI) may be beneficial in preventing AF recurrence, especially when interatrial block is present prior to ablation.⁴ This case describes a 70-year-old male with WPW and AF who underwent a combined PVI and AP ablation procedure. The patient remained free of AF post-procedure, suggesting that this approach may be effective in high-risk patients.

CASE

A 70-year-old male with a medical history of AF and WPW pattern presented with complaints of ear fullness and bilateral lower extremity swelling. He denied chest pain, palpitations, or shortness of breath but reported a gradual worsening of lower extremity edema over the past 2 months. He never had atrioventricular reentrant tachycardia and was not evaluated for AP ablation in the past. Upon presentation, his vital signs revealed a systolic

blood pressure of 140 mm Hg, a heart rate of 130 beats per minute (bpm), and oxygen saturation of 96% on room air.

Physical examination demonstrated irregular rhythm, tachycardia, and pitting edema in the lower extremities. Laboratory results indicated a troponin level of 18 ng/L and brain natriuretic peptide of 1321 pg/L. Electrocardiogram (ECG) demonstrated AF with rapid ventricular response of 160 bpm, nonspecific intraventricular conduction block, Q waves in inferior leads, and ST and T-wave changes in the anterior, and lateral leads (Figure 1). After discussing the benefits and risks, the patient was started on amiodarone to manage rate control due to heart failure concerns and the low risk of cardioversion compared to the risk of a cerebrovascular event in the context of chronic AF without anticoagulation. A transesophageal echocardiogram (TEE) (Figure 2A, B) revealed severe dilation of both the left and right atria, with severely decreased left ventricular systolic function (ejection fraction of 20-25%). Right ventricular systolic function was mildly to moderately reduced, and mild-to-moderate mitral and tricuspid regurgitation were noted. The patient successfully cardioverted to normal sinus rhythm (NSR) via direct current cardioversion (DCCV). Post-procedure ECG (Figure 3) showed NSR with a preexcitation pattern, negative delta waves in the inferior leads, and positive delta waves in the anterior leads. The patient was also treated with intravenous furosemide. Cardiomyopathy was attributed to tachycardia, with further outpatient workup planned. A subsequent transthoracic echocardiogram (TTE) (Figure 2C, D) revealed normal left ventricular size and wall thickness, with severely decreased systolic function (ejection fraction of 25-30%) and global hypokinesis. The initial plan was to consider outpatient ablation of the accessory pathway, but the patient reverted

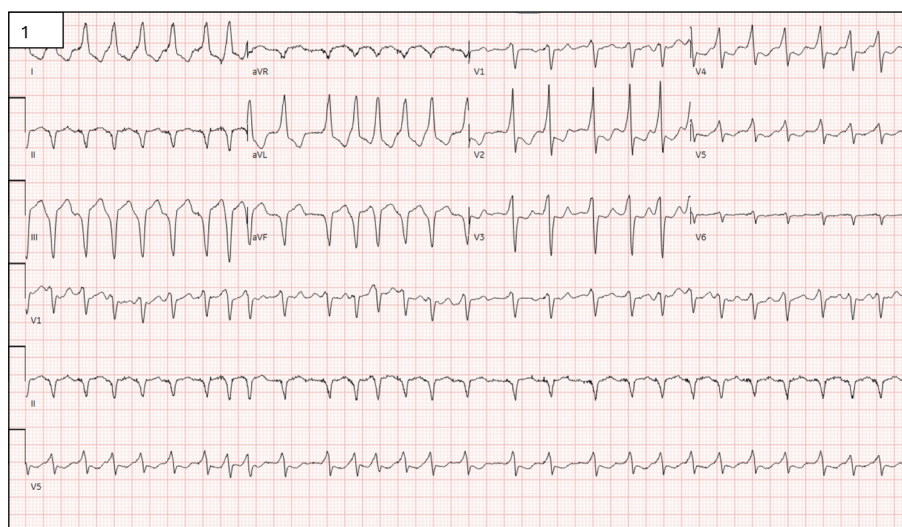


Figure 1 Electrocardiogram demonstrating atrial fibrillation with rapid ventricular rate, nonspecific intraventricular conduction block, and marked ST segment changes concerning for anterior subendocardial injury. There is also ST segment depression in lateral leads and Q waves in inferior leads.

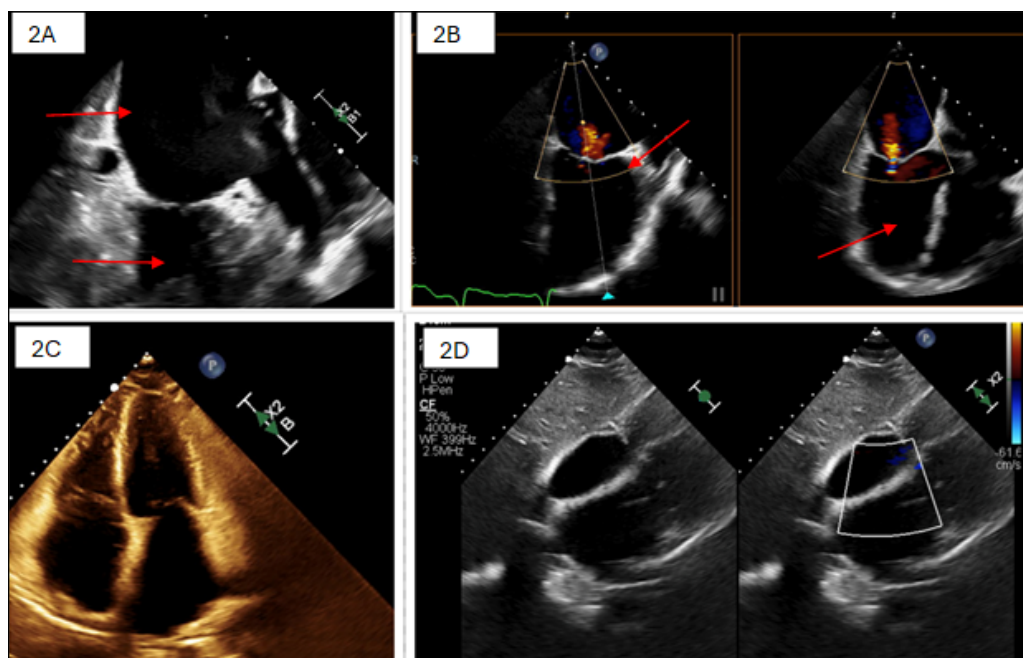


Figure 2 (A,B) Transesophageal echocardiogram demonstrates severe dilation of the left and right atria with no thrombus. The left ventricle (LV) exhibits significantly reduced systolic function (ejection fraction 20-25%), with global hypokinesis; the right ventricle shows mild to moderate systolic dysfunction. Mild mitral and moderate tricuspid regurgitation are present (2B), with central jets in both valves. **(C,D)** Transthoracic echocardiogram demonstrates persistent decreased systolic function and low ejection fraction of LV after cardioversion.



Figure 3 Electrocardiogram demonstrating normal sinus rhythm with preexcitation pattern, negative delta waves in the inferior leads, and positive delta waves in the anterior leads as depicted by the arrows.

to AF 2 days after achieving NSR. Consequently, the decision was made to perform combined PVI and AP ablation during the same hospital course. The patient was taken to the electrophysiology laboratory in a fasting, nonsedated state. Anesthesia and airway management were provided by the anesthesiology team, and the ablation procedure commenced. The groins were prepped and draped in

a sterile fashion, and sheaths were inserted using the modified Seldinger technique. Various catheters were employed, including an Abbott Livewire™ Duo-Decapolar catheter, a Biosense Webster Octopolar catheter at the HIS location, a Biosense Webster Quadripolar catheter at the right ventricular septum, a GE AcuNav ultrasound catheter, a Boston Scientific Fara wave Pulse Field Ablation

(PFA) catheter, and an Abbott Flexibility DF curve ablation catheter. The baseline rhythm was preexcited AF, and the patient underwent successful DCCV to sinus rhythm with a 200-joule synchronized shock. An intravenous heparin bolus was administered, and transseptal catheterization was performed under fluoroscopic and intracardiac echocardiography guidance, with the Faradrive sheath advanced into the left atrium.

The baseline left atrial voltage mapping post-cardioversion demonstrated a broad area of low voltage signals over the posterior wall, consistent with substrate abnormality. PVI was achieved using the FARAWAVE catheter (Boston Scientific) with a total of eight pulsed field ablation (PFA) applications per vein—four in basket and four in flower orientations, with splines rotated orthogonally. Posterior left atrial (LA) wall ablation was also performed in flower orientation with two PFA applications per site. Electroanatomic mapping using the Achieve catheter (Medtronic), and EnSite X (Abbott Cardiovascular) confirmed isolation of all four pulmonary veins and posterior LA wall, with conduction block demonstrated across the posterior wall. After posterior wall isolation, remaining LA regions showed preserved voltage, and no additional atrial scar was identified that warranted ablation. Thus, no other atrial substrates outside of the PVs and posterior wall were targeted, and the posterior wall isolation was anatomically and substrate guided, not purely empirical.

The patient had a manifest AP with bidirectional conduction; 1:1 ventriculoatrial (VA) conduction over the AP was < 290 ms, and VA effective refractory period at a drive of 600 ms was < 310 ms, with the earliest atrial activation at the proximal coronary sinus (CS). Orthodromic reentrant tachycardia was spontaneously induced, showing right bundle branch block morphology and cycle length of 550 ms, with earliest atrial activation again at proximal CS. Initial mapping during antegrade conduction localized earliest ventricular activation to both right and left posteroseptum. Although standard diagnostic pacing maneuvers were limited due to spontaneous termination of the tachycardia, the VA timing, activation sequence, and presence of manifest AP confirmed orthodromic atrioventricular reentrant tachycardia. Given the manifest pathway, short VA effective refractory period, inducible atrioventricular reentrant tachycardia, and presentation with preexcited AF, ablation of the AP was indicated to mitigate both the risk of sudden cardiac death and symptomatic supraventricular tachycardia burden.

Ablation at the mitral annulus from the left atrium using 35 watt to 40 watt radiofrequency (RF) did not eliminate AP conduction. After withdrawing the Faradrive sheath and stopping heparin, remapping during ventricular pacing showed earliest atrial activation at the right posteroseptum just outside the CS ostium (Figure 4A).

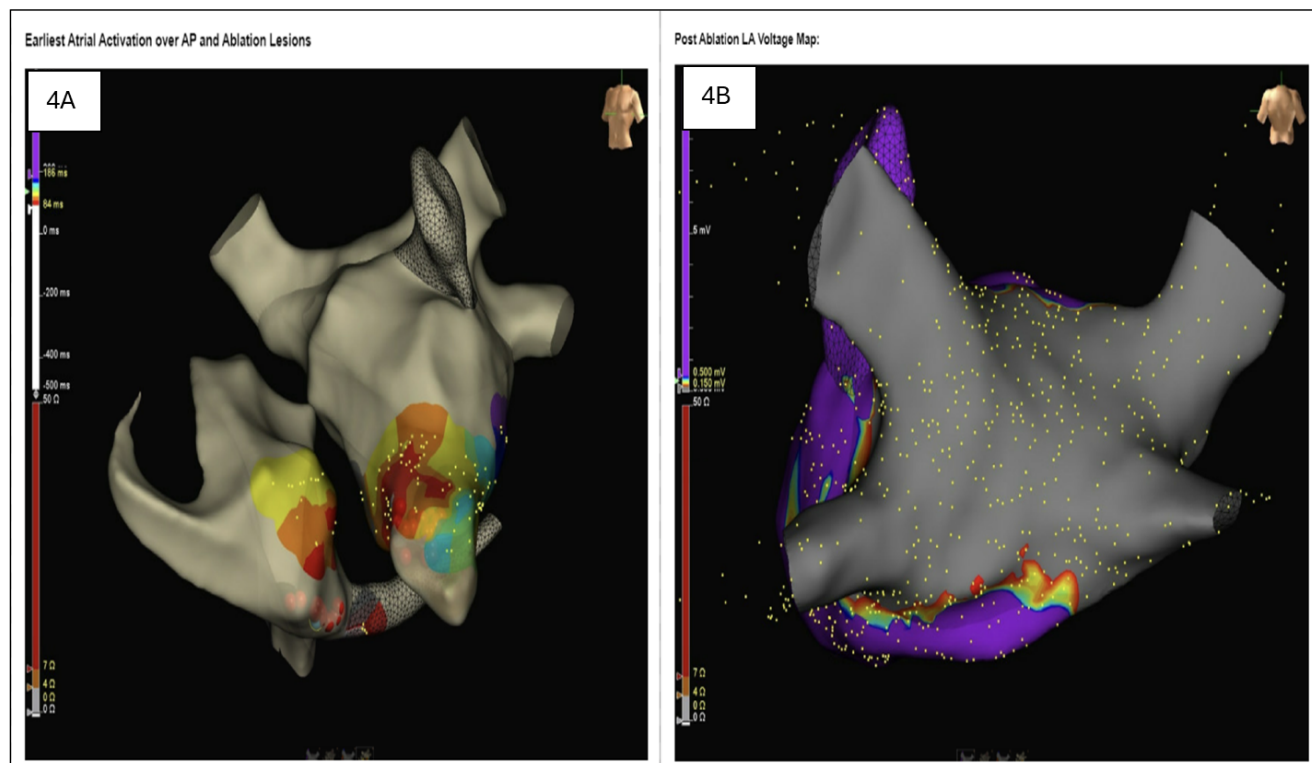


Figure 4 (A) Mapping illustrating the earliest atrial activation and potential accessory pathway. **(B)** Successful ablation of the accessory pathway in the right posteroseptum, with no remaining evidence of the pathway.

RF ablation at this site resulted in prompt elimination of retrograde AP conduction followed by additional lesions in the area. Post-ablation, there was no evidence of residual AP conduction or inducible tachycardia (Figure 4B). Intracardiac echocardiography confirmed no pericardial effusion. All catheters were removed, and a test dose of protamine was followed by full-dose administration without an allergic response.

An ECG (Figure 5) after the procedure showed NSR with first-degree atrioventricular block, right bundle branch block, QT interval prolongation, and T-wave inversion in the anterior, lateral, and inferior leads. The patient did not experience any episodes of AF following the procedure. At the 2-month follow-up, the ECG continued to show persistent NSR despite the patient not being on any antiarrhythmic medications (Figure 6).

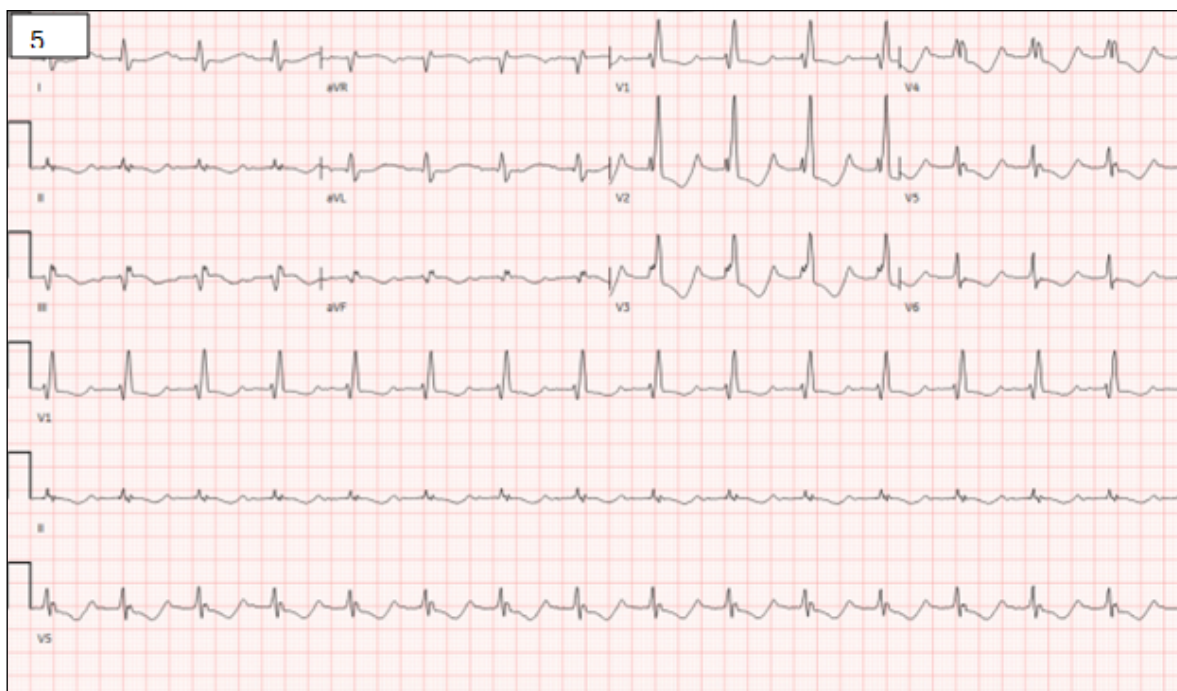


Figure 5 Electrocardiogram demonstrates sinus rhythm with first-degree atrioventricular block, right atrial enlargement, and right bundle branch block. Preexcitation is no longer observed.

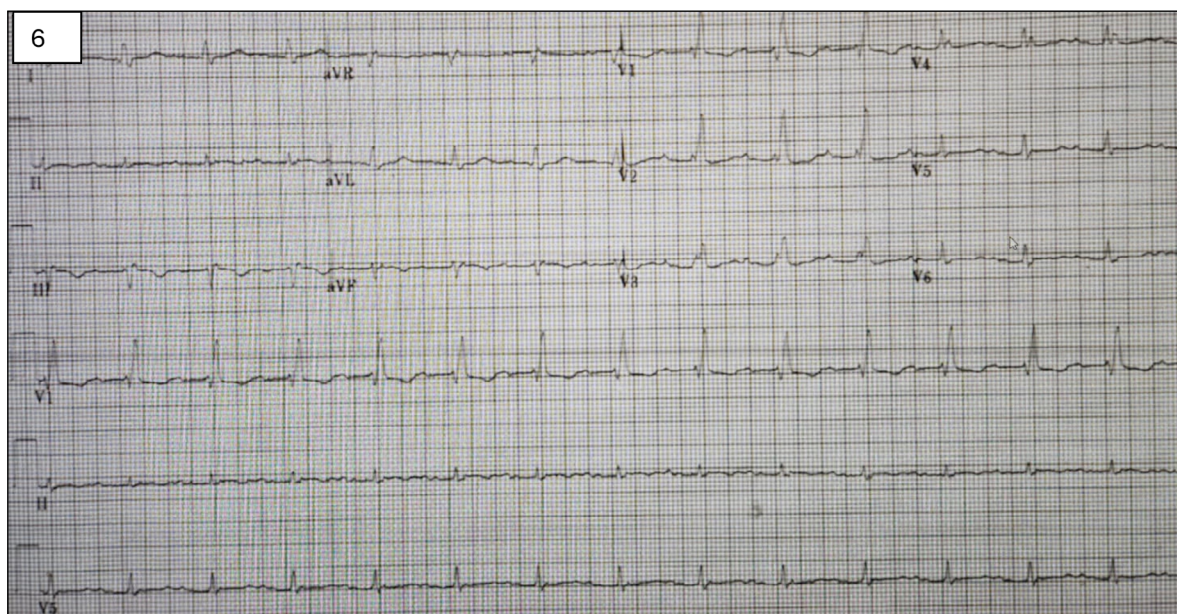


Figure 6 Electrocardiogram at 2 months showed normal sinus rhythm with no recurrence of atrial fibrillation.

DISCUSSION

AF in WPW syndrome can result from multiple factors, including rapid conduction through the AP, which shortens the refractory period and triggers AF.^{2,3} Frequent tachycardias, including atrioventricular reentrant tachycardia, can cause electrical remodeling and increase the atrial vulnerability to AF.^{2,3}

Although AP ablation can reduce AF recurrence, it does not completely prevent it, especially in patients with persistent AF inducibility or underlying atrial abnormalities. These factors contribute to the continued risk of AF even after successful AP ablation.^{2,3} WPW syndrome can manifest symptoms of tachyarrhythmia, such as palpitations, shortness of breath, dizziness, or syncope. The most common arrhythmia is atrioventricular reentrant tachycardia, although AF can also occur. ECG characteristics of AF in WPW is wide irregular QRS complexes due to electrical conduction through AP and can be polymorphic due to fusion of QRS through AP and normal conduction system.² The primary treatment for AF in WPW focuses on maintaining hemodynamic stability and using antiarrhythmics such as procainamide or ibutilide.¹

Amiodarone, as used in our case, can be effective but should be used cautiously in acute settings due to its beta-blocking and calcium channel-blocking properties, which may exacerbate AF. Atrioventricular nodal blockers, including beta blockers, calcium channel blockers, and digoxin, are contraindicated as they can enhance conduction through the accessory pathway, potentially triggering ventricular fibrillation. DCCV is considered in hemodynamically unstable AF or AF refractory to antiarrhythmic therapy.²

Catheter ablation is recommended for patients with WPW syndrome and AF, as tachyarrhythmias through the accessory pathway can lead to atrial remodeling, increasing the risk of AF. Ablation of the accessory pathway can reduce the recurrence of preexcited AF, with a success rate of 95% for RF ablation. However, patients still face the risk of future AF, particularly if they are over 50 years old. For those with unsuccessful catheter ablation or recurrent AF after accessory pathway ablation, antiarrhythmic medications may be necessary. However, these drugs can sometimes be unsuitable due to side effects, especially in older patients who are at higher risk of complications, including cerebrovascular accidents if not on anticoagulation. Studies show that in individuals with WPW syndrome and AF, the effective refractory periods of the pulmonary veins are shorter, and there is a longer conduction delay between the veins and the atrium compared to those with

WPW without AF, highlighting the role of pulmonary veins in AF development.^{5,6} PVI in that case may be a successful intervention to decrease recurrence of AF in these high-risk patients, though more studies and randomized controlled trials need to be conducted to consider this aspect.⁴ In our case, we opted to proceed with both PVI ablation and catheter ablation of the AP given the patient's elevated risk for AF in the context of advanced age and multiple comorbidities.

There was a clear rationale for performing AF ablation before AP ablation. At the beginning of the procedure, the patient presented with preexcited AF, which required immediate cardioversion to sinus rhythm to allow accurate anatomic and voltage mapping for PVI. Following cardioversion, AF ablation was prioritized for several reasons. The patient had symptomatic, persistent AF requiring definitive substrate modification regardless of the AP. Given its persistent nature and posterior wall involvement, it was felt that the AF substrate was independent of the AP. Additionally, there was concern that delaying AF ablation might lead to reinduction of preexcited AF. Although ablating the AP first could theoretically help determine whether AF was AP-mediated, clinical judgment favored addressing both arrhythmogenic substrates, with PVI first allowing for a more controlled and comprehensive approach in a single session. Ultimately, both AF and AP ablations were successfully performed.

CONCLUSION

AF is a common arrhythmia in patients with WPW, and its management is challenging, particularly in acute presentations with rapid ventricular response. While catheter ablation reduces AF recurrence and mortality, high-risk patients remain at risk. Concomitant PVI may further improve outcomes by minimizing residual risk of future AF. Additional randomized controlled trials are needed to evaluate the benefits of combining PVI with catheter ablation versus catheter ablation alone in WPW patients with AF, especially in older individuals.

KEY POINTS

- In patients with Wolff-Parkinson-White syndrome and atrial fibrillation (AF), accessory pathway (AP) ablation can reduce AF recurrence but does not eliminate the risk, especially in older patients or those with a history of AF.

- Combining pulmonary vein isolation with AP ablation may further decrease AF recurrence and improve outcomes in high-risk patients, although more studies are needed.

COMPETING INTERESTS

The authors have no competing interests to declare.

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TO CITE THIS ARTICLE:

Sabri MS, Benedict L, Souder D, Kaugars L, Patel K, Bozorgnia B, Nazir T. Lehigh Valley Procedure: Combined Pulmonary Vein Isolation and Accessory Pathway Ablation for Atrial Fibrillation in Wolff-Parkinson-White Syndrome. *Methodist DeBakey Cardiovasc J*. 2025;21(1):74-80. doi: [10.14797/mdcvj.1618](https://doi.org/10.14797/mdcvj.1618)

Submitted: 15 April 2025 **Accepted:** 09 July 2025 **Published:** 20 August 2025

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