

Novel method of superior vena cava electrical isolation with close proximity to the phrenic nerve



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Introduction

Pulmonary vein isolation (PVI) has been the cornerstone of catheter ablation for drug-refractory, symptomatic atrial fibrillation (AF) since its description more than 20 years ago.¹ Despite its success relative to other treatment options, recurrence of atrial arrhythmia is common.² Non-pulmonary vein sources are thought to be responsible for many of these recurrences, and the superior vena cava (SVC) as a source is seen in up to 40% of patients with non-pulmonary vein triggers.³ Electrically active myocardial sleeves can extend from the right atrium (RA) into the proximal portion of the SVC, and the electrical properties of this tissue have been shown to both initiate and perpetuate AF.⁴⁻⁷ The SVC can be electrically isolated from the RA by radiofrequency ablation targeting sites of earliest activation or through circumferential ablation immediately above the SVC-RA junction if activation mapping is not feasible.⁸ SVC isolation has been shown to prevent AF recurrences, either as an empiric addition to PVI in paroxysmal AF or in patients with AF thought to be arising from or perpetuated by the SVC.^{6,7} Damage to the right phrenic nerve (RPN) is the most common complication, occurring in up to 5% of cases.⁷ The RPN travels along the posterolateral aspect of the SVC and RA, and its proximity to the SVC can be assessed by pacing from within the SVC and RA to identify right diaphragmatic contraction. The pericardial layers and fat separate the RPN from the myocardium, and the length of proximity between these structures is variable. RPN injury can be prevented by avoiding ablation in the areas with diaphragmatic capture, which limits SVC isolation and successful treatment of SVC-associated AF in up to 18% of patients.⁹ Here we describe a novel approach in which the SVC was successfully isolated via a circumferential approach despite the proximity of the RPN at the SVC-RA junction without damage to the nerve.

KEYWORDS Ablation; Atrial fibrillation; Non-pulmonary vein triggers; Phrenic nerve; Superior vena cava; Superior vena cava isolation (Heart Rhythm Case Reports 2019;5:461-464)

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KEY TEACHING POINTS

- The superior vena cava (SVC) can serve as a substrate for initiation and maintenance of atrial fibrillation (AF), and isolation of the SVC can prevent AF recurrence in these patients.
- Damage to the right phrenic nerve is the most common complication, and its avoidance limits successful isolation in up to 18% of cases.
- Damage to the right phrenic nerve may be avoided by comprehensive phrenic nerve mapping and ablation parallel to the phrenic nerve until the point at which diaphragmatic capture is no longer present in the right atrium and then connecting this line to a circumferential ablation line within the SVC.

Case report

A 71-year-old man with medical history notable for hypertension and AF with failure to maintain sinus rhythm despite amiodarone and electrical cardioversion was referred to the electrophysiology clinic for tachycardia-induced cardiomyopathy and congestive heart failure with a left ventricular ejection fraction (LVEF) of 25%. He had a normal LVEF at the time of diagnosis of AF 6 months prior. Despite rate control attempts, his left ventricular function failed to improve. His electrocardiogram in clinic showed AF with a ventricular rate of 64 beats per minute, QRS duration 106 ms, abnormal repolarization, and a corrected QT interval of 437 ms. Medications included amiodarone 200 mg daily, carvedilol 25 mg twice daily, losartan 100 mg once daily, aspirin 81 mg daily, and furosemide 20 mg daily. His examination demonstrated an irregularly irregular heart rhythm but was otherwise normal. After discussion of treatment options, a strategy of catheter ablation was chosen to attempt rhythm control.

In the electrophysiology lab, the patient was intubated and general anesthesia was performed. Multiple femoral venous accesses were obtained, and a decapolar catheter was introduced into the coronary sinus. A 3-dimensional electroanatomic map

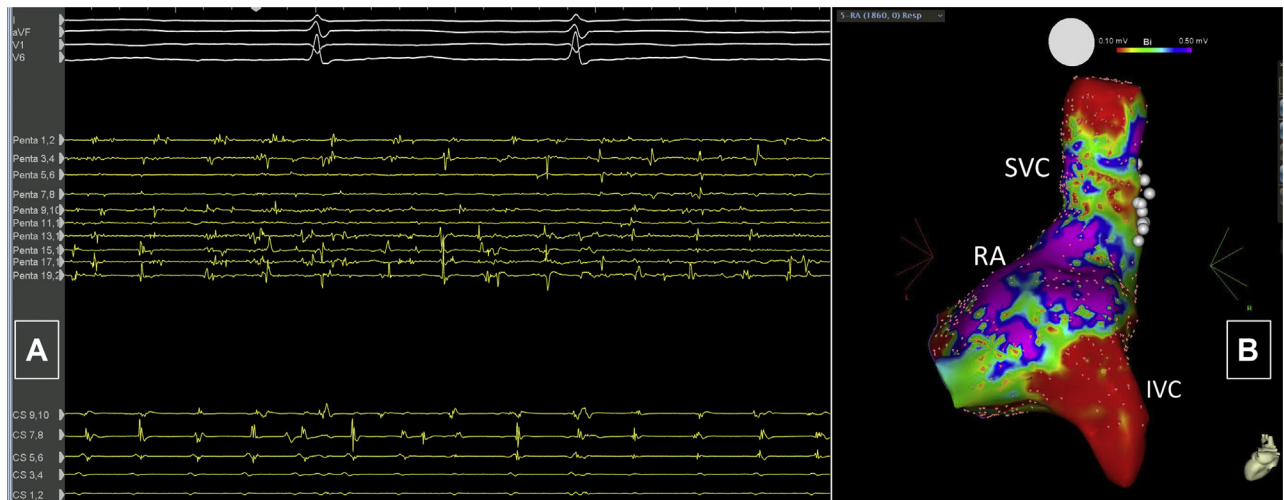


Figure 1 Electrical activity in the superior vena cava (SVC) and right atrium (RA). **A:** Electrograms labeled “Penta” indicate signals from the multipolar PentaRay mapping catheter (Biosense Webster, Diamond Bar, CA) within the SVC. Electrograms labeled “CS” indicate signals from the coronary sinus catheter. **B:** Voltage map of the RA and SVC. Electrically active tissue is seen several centimeters into the SVC. The *white dots* indicate the location of the right phrenic nerve. IVC = inferior vena cava.

was then created using a CARTO 4 mapping software (Biosense Webster, Diamond Bar, CA) and D-curve PentaRay mapping catheter (Biosense Webster) in AF owing to failed cardioversion in maintaining sinus rhythm previously. A long sleeve of electrically active tissue was noted in the SVC with complex fractionated electrical signals (*Figure 1*). Owing to the extent and complexity of electrical activity within the SVC, SVC isolation in addition to the left atrial ablation was considered for potential benefit of AF trigger and substrate elimination for persistent AF. A ThermoCool SmartTouch catheter (Biosense Webster) was used for additional mapping and ablation. No paralytics were used for the procedure. The RPN proximity was mapped using high-output pacing (20 mA, 2 ms pulse width), minimum contact force of 5 grams, and locations were tagged on the map. No phrenic nerve capture was noted on either side of or below the annotated phrenic

nerve map. Ablation was performed using a contact force of 3–20 grams and power of 25–30 W, targeting electrogram change. In addition, the Visitag module was used with the criteria of 1 mm stability, for 5 seconds, with a minimum of 3 grams contact force for 50% of the time for automated lesion annotation. A circumferential ablation was performed, approximately 1 cm above the SVC-RA junction except where diaphragmatic capture was noted. In order to avoid ablation overlying the phrenic nerve, the ablation line was directed parallel to the phrenic nerve in a caudal direction until the point at which diaphragmatic capture was no longer seen in the RA. The line was then directed parallel to the phrenic nerve on the opposing side in a cranial direction and finally connected with the circumferential ablation lesion set above the SVC-RA junction (*Figure 2*). High-output pacing was performed immediately prior to the ablation in each of the planned ablation

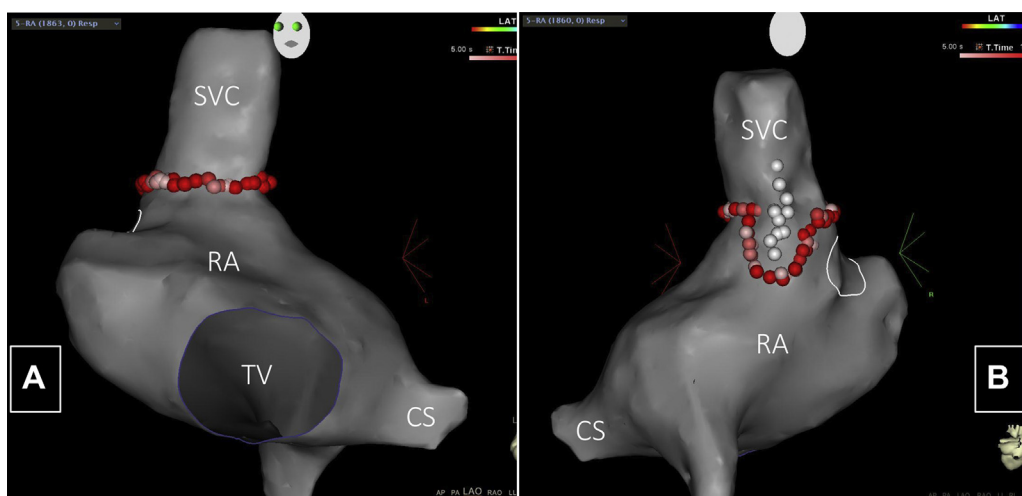


Figure 2 Anatomic map of the right atrium (RA) and superior vena cava (SVC). **A:** Left anterior oblique view. **B:** Posteroanterior view. *Pink and red dots* represent radiofrequency ablation lesions and the *white dots* in panel B represent the mapped location of the right phrenic nerve. The *white circle* represents the lateral SVC-RA junction for anatomic appreciation of the sinus node. CS = coronary sinus; TV = tricuspid valve.

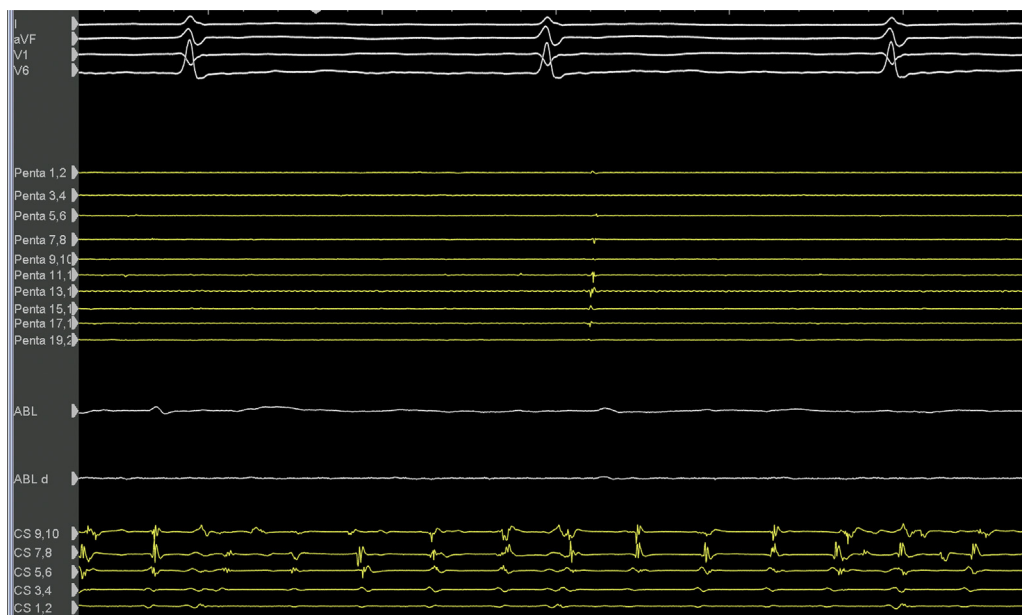


Figure 3 Entrance block with electrical firing within the superior vena cava (SVC). Electrograms labeled “Penta” indicate signals from the multipolar PentaRay mapping catheter (Biosense Webster, Diamond Bar, CA) within the SVC. Note the spontaneous electrical activation after entrance block into the SVC. Electrograms labeled “CS” indicate signals from the coronary sinus catheter.

locations adjacent to the linear phrenic map to confirm no phrenic capture. The SVC was electrically isolated with entrance block. Isolated electrical firing (Figure 3) was noted after isolation. Phrenic nerve capture was verified to be intact by pacing above the level of ablation lesion set. A transseptal puncture was then performed and left atrial ablation involving PVI and posterior wall isolation was performed. Rhythm conversion was performed with synchronized electrical cardioversion. Persistent electrical isolation was confirmed in the SVC prior to the completion of the procedure. Continued capture of the RPN was confirmed above the level of ablation. At 3-month follow-up, the patient remained free of arrhythmia off antiarrhythmic drug therapy and LVEF had normalized to 55%.

Discussion

In this report, we describe a novel approach of SVC isolation despite diaphragmatic capture at the SVC-RA junction by pursuing a comprehensive RPN mapping and a U-shaped ablation lesion set around the caudal aspect. The SVC can serve as a substrate for initiation and maintenance of AF.^{3–6} In selected patients, isolation of the SVC utilizing radiofrequency ablation can prevent AF recurrence,^{6,7} though the risk of damaging the RPN may prevent successful isolation in up to 18% of patients. Typically, when diaphragmatic capture is noted in the area of the SVC-RA junction, ablation is avoided owing to the risk of phrenic nerve damage. In many patients, SVC isolation is feasible without circumferential ablation owing to discrete muscular bundles that extend into the SVC without an encircling sleeve of electrically conductive muscle. However, a proximal circumferential SVC isolation is often required, especially if activation mapping is not feasible as in AF. In patients with close SVC/phrenic nerve proximity a technique

of activation-guided identification of sleeves in sinus rhythm and ablation of these areas within the septal and posterior RA was suggested as one of the feasible methods to successfully isolate the SVC with a small risk of phrenic nerve damage.¹⁰ Another alternative approach is by obtaining structural separation between the targeted ablation site and the RPN by epicardial access and balloon placement or saline injection.¹¹ This, however, has limits of needing epicardial access and associated risk.

Our case describes a novel method of avoiding injury to the RPN by ablating parallel to the phrenic nerve until a point within the RA where diaphragmatic capture was no longer seen. This technique also allows SVC isolation during AF without the need for activation mapping in sinus rhythm to identify sleeves of conduction to the SVC from the RA. This approach, however is limited if the RPN has continuous proximity in the SVC and the entire length of the RA. The approach is also less likely to result in SVC stenosis compared to the circumferential ablation at the same level. However, it is important to choose the level of isolation in the proximal SVC to avoid risk of stenosis, and above the anatomic location of sinus node to avoid sinus node damage.

References

1. Haissaguerre M, Jais P, Shah DC, et al. Spontaneous initiation of atrial fibrillation by ectopic beats originating in the pulmonary veins. *N Engl J Med* 1998;339:659.
2. Sultan A, Luker J, Andresen D, et al. Predictors of atrial fibrillation recurrence after catheter ablation: data from the German ablation registry. *Sci Rep* 2017; 7:16678.
3. Lin WS, Tai CT, Hsieh MH, et al. Catheter ablation of paroxysmal atrial fibrillation initiated by non-pulmonary vein ectopy. *Circulation* 2003;107:3176–3183.
4. Higuchi K, Yamauchi Y, Hirao K, et al. Superior vena cava as initiator of atrial fibrillation: factors related to its arrhythmogenicity. *Heart Rhythm* 2010; 7:1186–1191.

5. Nakamura T, Hachiya H, Yagishita A, Tanaka Y, Higuchi K, Kawabata M. The relationship between the profiles of SVC and sustainability of SVC fibrillation induced by provocative electrical stimulation. *Pacing Clin Electrophysiol* 2016; 39:352–360.
6. Corrado A, Bonso A, Madalosso M, et al. Impact of systematic isolation of superior vena cava in addition to pulmonary vein antrum isolation on the outcome of paroxysmal, persistent, and permanent atrial fibrillation ablation: results from a randomized study. *J Cardiovasc Electrophysiol* 2010;21:1–5.
7. Miyazaki S, Takigawa M, Kusa S, et al. Role of arrhythmogenic superior vena cava on atrial fibrillation. *J Cardiovasc Electrophysiol* 2014;25:380–386.
8. Arruda M, Mlcochova H, Prasad SK, et al. Electrical isolation of the superior vena cava: an adjunctive strategy to pulmonary vein antrum isolation improving the outcome of AF ablation. *J Cardiovasc Electrophysiol* 2007; 18:1261–1266.
9. Chang H, Lo L, Lin Y, et al. Long-term outcome of catheter ablation in patients with atrial fibrillation originating from the superior vena cava. *J Cardiovasc Electrophysiol* 2010;68:955–961.
10. Gianni C, Sanchez J, Mohanty S, et al. Isolation of the superior vena cava from the right atrial posterior wall: a novel ablation approach. *Europace* 2018; 20:e124–e132.
11. Ibarra-Cortez SH, Rodríguez-Mañero M, Kreidieh B, Schurmann P, Dave AS, Valderrábano M. Strategies for phrenic nerve preservation during ablation of inappropriate sinus tachycardia. *Heart Rhythm* 2016; 13:1238–1245.