Protecting the right phrenic nerve during catheter ablation: Techniques and anatomical considerations

Stephen Stark, MD, David K. Roberts, MD, Thomas Tadros, MD, James Longoria, MD, Subramaniam C. Krishnan, MD, FHRS

From the Sutter Heart & Vascular Institute, Sacramento, California.

Introduction

Phrenic nerve injury is a recognized complication of catheter ablation procedures for atrial and ventricular arrhythmias.[1,2] The posterolateral right atrium (RA) is a common site of origin for atrial tachycardias (AT), but ablation in this region can be hazardous because of the proximity of the right phrenic nerve (RPN). Proximity to the nerve may be identified by capture during pacing to avoid injury during energy application. Balloon inflation in the epicardial space and the infusion of saline and of air into the pericardial space have been described to protect the left phrenic nerve in the setting of epicardial ventricular tachycardia ablation.[3–5] Techniques to protect the RPN during endocardial atrial ablation procedures have been described but not as comprehensively.[6]

We discuss a case to highlight the difficulties of catheter ablation of "periphrenic" right AT and to describe epicardial catheter techniques for RPN protection to permit safe endocardial ablation from the posterolateral RA.

Case report

A 66-year-old woman with a 15-year history of recurrent supraventricular tachycardia that was almost incessant despite flecainide and β-blockers presented for catheter ablation. She had undergone 2 prior studies at an outside hospital. On both occasions, ablation was not performed because of the proximity of the arrhythmoghenic focus to the RPN. Her echocardiogram showed normal left ventricular systolic function with no chamber enlargement or valvular heart disease. She was offered a repeat ablation procedure with possible pericardial balloon inflation to protect the RPN, and she agreed to proceed.

The procedure was performed under general anesthesia. After isoproterenol infusion, sustained AT (cycle length 330 ms) was induced during programmed stimulation and burst pacing from the atrium. The intracardiac electrograms from a decapolar catheter placed in the coronary sinus, a high-density 20-electrode mapping catheter, and the ablation catheter as well as surface electrocardiographic leads are shown in Figure 1A. The earliest activation is seen in the distal pair of electrodes of the ablation catheter. Activation sequence mapping performed in both atria with multipolar catheter recordings and electroanatomic technology (NavX, St. Jude Medical, St. Paul, MN) revealed the earliest site of AT activation to be at the junction of the superior vena cava and the posterior RA. A fluoroscopic image (23° right anterior oblique view) of the ablation catheter positioned at this site is shown in Figure 1B. A right lateral view and a cranial view of the NavX map displaying the right and left atria are shown in Figure 1C. The area in white at the high posterior RA represents the area of origin of the AT. Similar to her previous procedures, high output pacing at this site (20 mA; pulse width of 2 ms) revealed diaphragmatic contractions, indicating proximity to the RPN. A "threshold effect" to determine the pacing output when phrenic nerve capture occurred was not tested.

A decision was made to canulate the pericardial space and deploy a balloon between the arrhythmogenic myocardium in the posterosuperior RA and the RPN that is attached to the adjacent parietal pericardium. Pericardial access was obtained with a standard 17-G epidural needle using the technique described by Sosa et al.[7] A steerable 8.5 French Agilis sheath (St. Jude Medical, St. Paul, MN) with a 18 × 40-mm balloon catheter (Meditech XXL, Boston Scientific Corporation, St. Paul, MN) was inserted and directed to the epicardium overlying the earliest endocardial AT site (Figure 2). Over a 0.035-inch extra-stiff Amplatz wire (Boston Scientific Corporation, St. Paul, MN), the balloon and sheath were advanced via the superior aortic recess or superior sinus of the pericardium to the site adjacent to the posterolateral RA and was inflated at this site. Figure 2A shows a fluoroscopic image in the right anterior view (25°) with the inflated balloon. However, pacing from the ablation catheter continued to demonstrate diaphragmatic contractions, indicating that the RPN remained adjacent to the

KEYWORDS Catheter ablation; Phrenic nerve injury; Pericardial space; Balloon inflation; Pericardial reflections
(Heart Rhythm Case Reports 2017;3:199–204)

Address reprint requests and correspondence: Dr Subramaniam C. Krishnan, Sutter Heart & Vascular Institute, 2800 L St, 6th Floor, Sacramento, CA 95816. E-mail address: Krishnan_sc@hotmail.com.

http://dx.doi.org/10.1016/j.hrcr.2016.08.012

KEY TEACHING POINTS

- Ablation of the atrial tachycardias arising from the posterolateral right atrium can be hazardous because of the proximity of the right phrenic nerve that may be injured. Proximity of the ablation catheter to the nerve may be identified by capture during pacing.
- Balloon inflation in the pericardial space has been described to protect the left phrenic nerve in the setting of epicardial ventricular tachycardia ablation. Techniques to protect the right phrenic nerve during endocardial atrial ablation procedures have not been well described.
- The maneuver of balloon inflation in the pericardial space to separate the right phrenic nerve from the arrhythmogenic atrial myocardium is more complex because of anatomical challenges unique to this location.
- The right phrenic nerve can course adjacent to a pericardial reflection, and it can be difficult to position a balloon adjacent to this reflection. We used the anterior pericardium "to push against" and thus force the posterolateral pericardium and the embedded phrenic nerve away from the ablation catheter.

ablation catheter and hence ablation was not performed. Approximately 200 cm^3 of saline was injected into the pericardial space. However, this maneuver even in combination with balloon inflation was ineffective in protecting the RPN, with diaphragmatic contractions continuing to be seen with pacing from the ablation catheter. A different maneuver was then attempted (Figure 2B). The "extra-stiff" Amplatz wire that was inserted through the central lumen of the balloon catheter was forcefully advanced further anteriorly up to the anterior pericardium. As seen in the figure, as a consequence of forward pressure, the wire tip bends backward. Consequently, the pericardial sheath is seen to straighten, indicating tension from forward pressure applied in the balloon catheter. Compared to Figure 2A, the distance between the balloon and the coronary sinus catheter is seen to be substantially greater, indicating a posterior displacement of the balloon. Pacing from the ablation catheter (white arrow) now showed no phrenic nerve capture. A 60-second radiofrequency ablation (35 W) was performed at this position with successful termination of the arrhythmia within 3 seconds of energy application (shown in Figure 2C). This was the only lesion delivered. Postablation, despite aggressive pharmacological and programmed stimulation that included infusion of >10 μg/min of isoproterenol, application of triple extrastimuli, and burst pacing, the arrhythmia remained noninducible. Approximately 200 mL of hemorrhagic fluid was removed, possibly related to the pericardial balloon. At the end of the procedure, the RPN was paced from the superior vena cava and the presence of diaphragmatic contractions was verified. The patient has been free of palpitations on no antiarrhythmic drugs after a follow-up of 12 months.

Discussion

Our message is that for "periphrenic" right AT, transposing a balloon in the pericardial space between the arrhythmogenic myocardium and the phrenic nerve will work, but there are significant problems that can occur. We feel that this is largely related to the fact that the RPN may be immediately adjacent to pericardial reflections and recesses where positioning a large balloon is difficult.[8,9] In this situation, as was the case with our patient, inflating a balloon may not completely solve the problem. Postinflation, a cylindrical balloon will simply move away from the reflection and from the RPN. The pericardial anatomy is less complex with the left phrenic nerve where the course of this nerve is not adjacent to pericardial reflections.

The arrhythmogenic myocardium in our patient was at the posterior portion of the superior vena cava and RA. Pacing from this site induced diaphragmatic contractions, indicating that the RPN was immediately adjacent to this site. Likely because of the proximity of the RPN to the pericardial reflection and possibly the postcaval recess, it was difficult to position the balloon adjacent to the nerve. We therefore had to resort to the maneuver of using the anterior pericardium "to push against" and thus force the posterolateral pericardium (with the embedded RPN) away from the ablation catheter and the arrhythmogenic myocardium. One has to be cognizant of the pericardial recesses, and the maneuver where the anterior pericardium was used to push against was crucial.

Injecting saline and creating a hydropericardium did not work. We did not inject air. We felt that this maneuver, that is, injecting air, is more likely to help separate the anterior pericardium and less likely to help separate a posterior structure such as the RPN. The big difference between the course of the right and left phrenic nerves is the fact that the RPN can course adjacent to a pericardial reflection and it can be difficult to position a balloon adjacent to this reflection. Injecting fluid and air had more of a role when deflectable pericardial sheaths were unavailable. The availability of a deflectable pericardial sheath allows balloon deployment with greater precision, which we believe obviates the need for intrapericardial instillation of fluid or air.

Other approaches, such as the use of cryoablation at the site of arrhythmia origin during phrenic nerve pacing, and turning ablation off at the first sign of phrenic nerve injury was not tested. We acknowledge that this strategy may facilitate successful elimination of the arrhythmia and has the potential to obviate the need for deployment of an intrapericardial balloon. We also did not study phrenic nerve capture throughout the earliest region of AT activation. The question of whether ablation at a proximal site within the earliest activation region be performed without risk to the phrenic nerve was not tested.

The cause of the hemopericardium is not entirely clear. We feel that it is most likely secondary to trauma to some of the blood vessels, especially veins traversing on the inner surface of the pericardium or on the epicardial surface of the heart. Manipulation of the sheath or systemic anticoagulation with heparin certainly may have played a role. There was no significant hemodynamic consequence and the bleeding stopped without needing any additional measures.

Matsumoto et al[10] showed that there was a significant variation in the course of the left phrenic nerve. It is logical that a similar variation in the course of the RPN also exists. Therefore, in future ablation procedures where the operator aims to separate the arrhythmogenic myocardium from the RPN, he or she can expect challenges based on this variation and should be prepared to come up with innovative solutions.

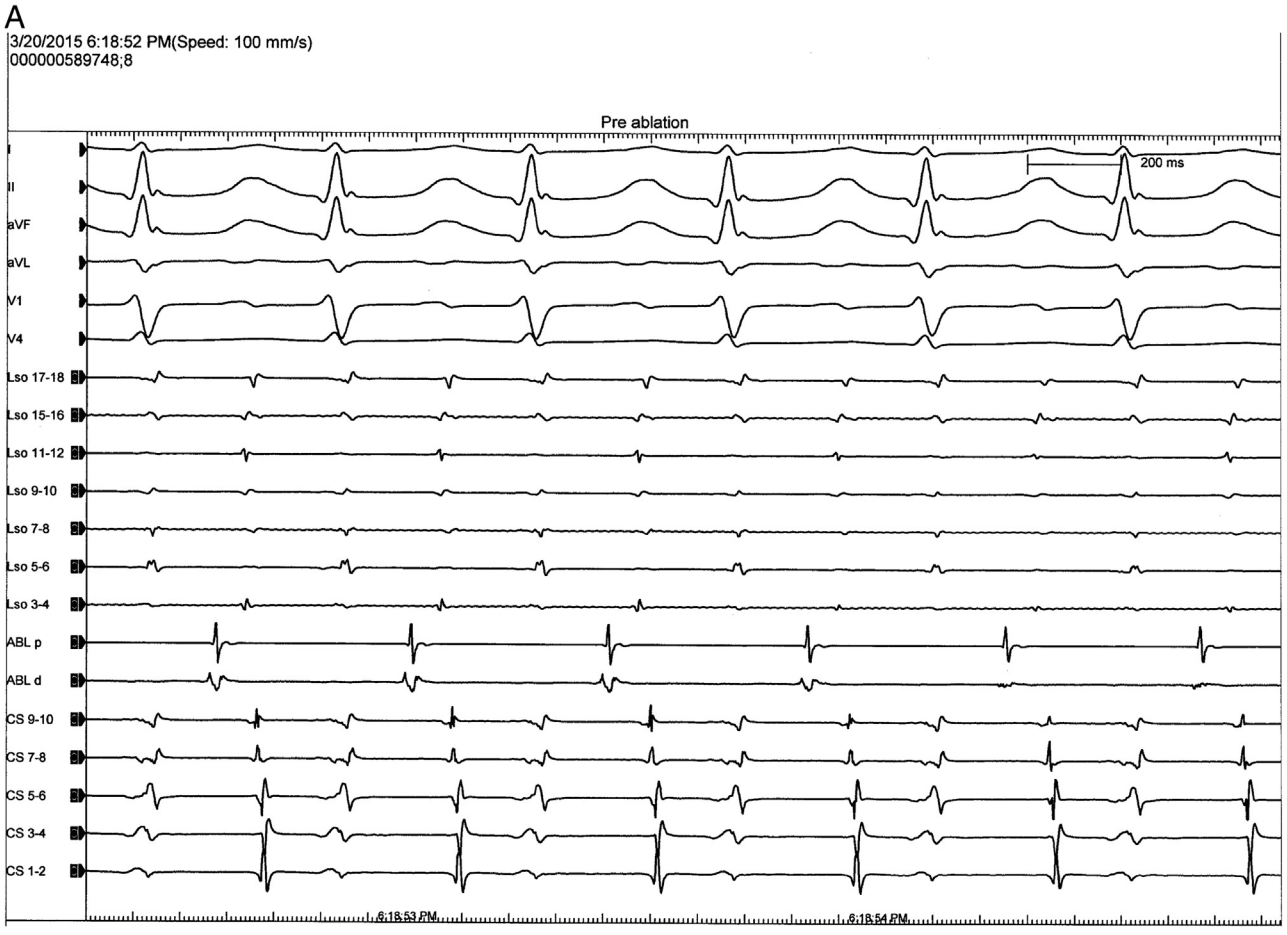
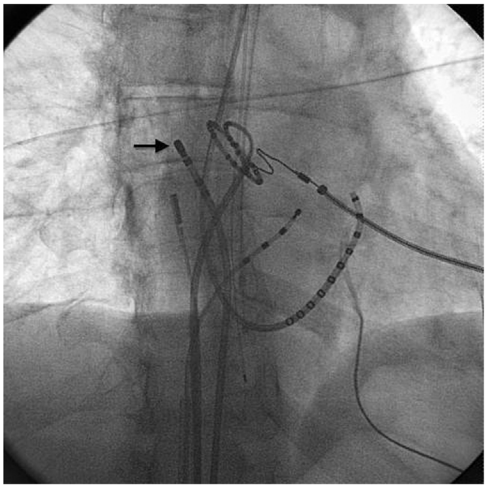


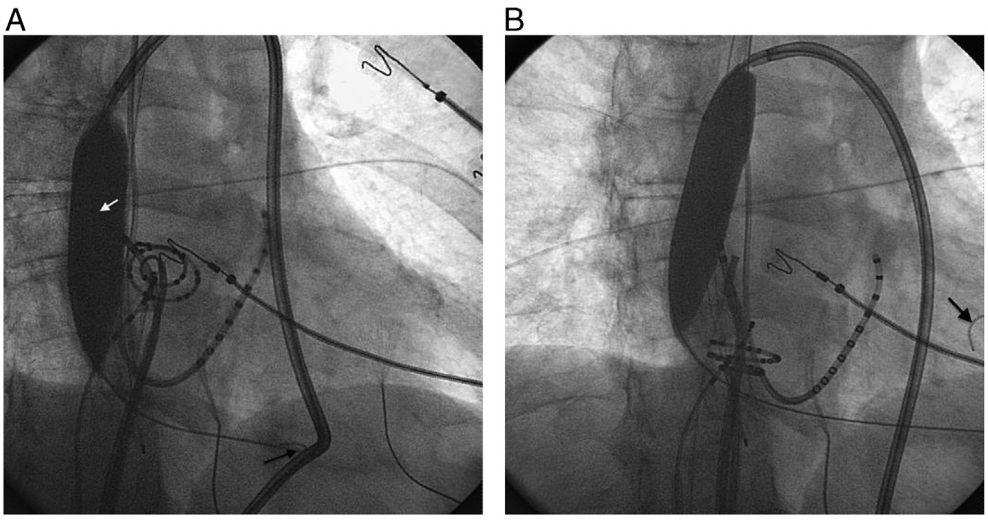
Figure 1 **A:** Intracardiac tracings and 4 surface leads recorded during atrial tachycardia. The earliest atrial activation is seen in the distal pair of electrodes of the ablation catheter (Abl d) positioned in the junction of the superior vena cava and the posterior right atrium. **B:** A 23° right anterior oblique view showing catheter positions. The ablation catheter (arrow) is positioned at the site of origin of the atrial tachycardia at the junction of the superior vena cava and the posterior right atrium. **C:** A NavX map of the right and left atria displaying geometry and activation of the atrial tachycardia. The figure on the left shows a right lateral view of the right and left atria, and the figure on the right shows a cranial view. The ablation catheter is seen to be positioned at the site of origin of the arrhythmia (colored in white) at the junction of the superior vena cava and the right atrium.

B



C

Figure 1 Continued



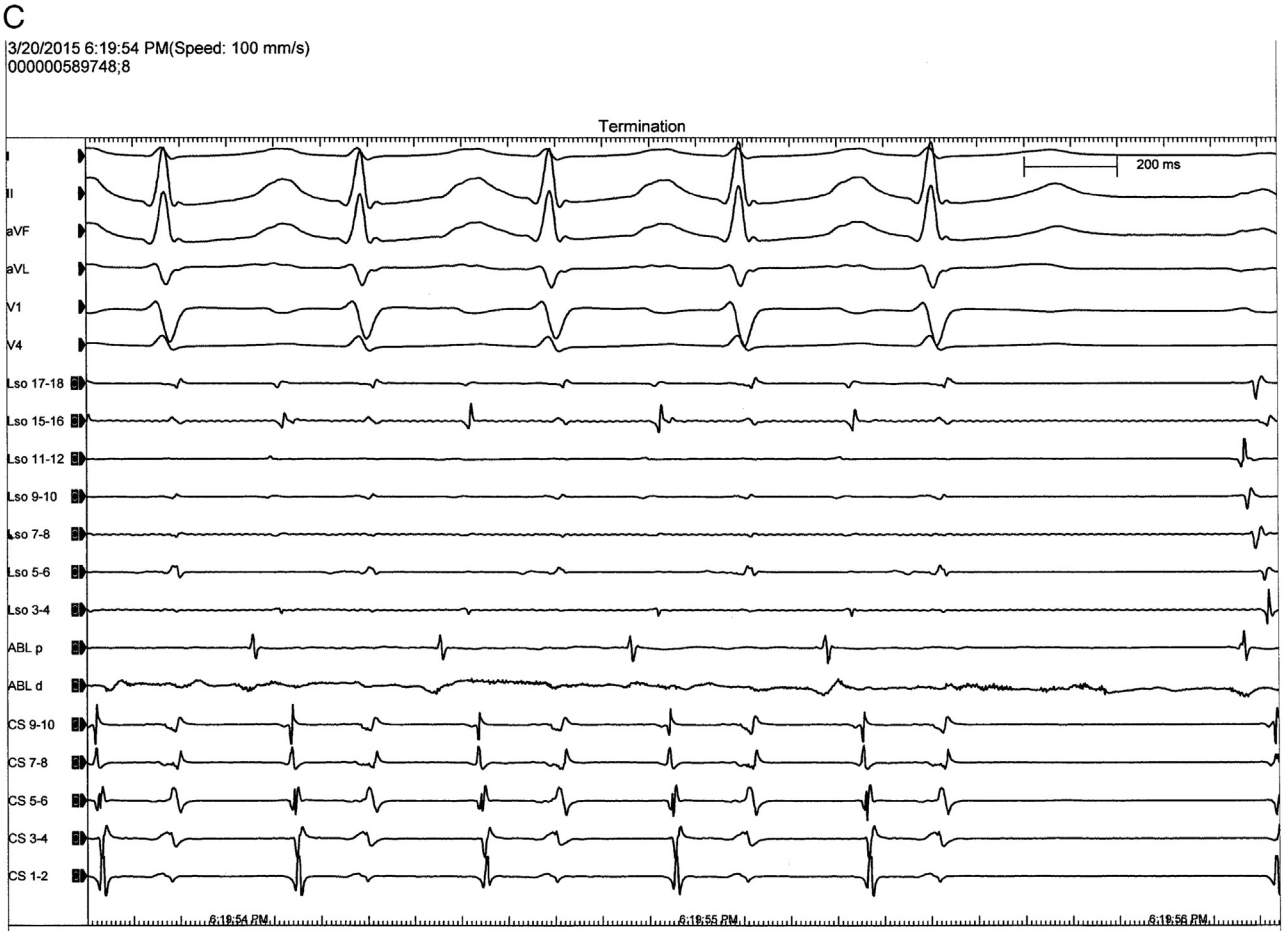


Figure 2 **A:** A 25° right anterior oblique view showing the balloon inflated and positioned in the posterolateral region adjacent to the ablation catheter. Attention is drawn to the position of the wire tip (black arrow), which is halfway between the balloon and the anterior pericardium. Pacing from the ablation catheter (white arrow) with the balloon positioned at this position showed phrenic nerve capture. Ablation was not performed at this position. Note the pericardial sheath looking slightly tortuous, indicating a lack of tension in the balloon catheter. **B:** A 27° right anterior oblique view showing the balloon inflated and positioned in the posterolateral region adjacent to the ablation catheter. Attention is drawn to the position of the wire tip (black arrow), which is pushed against the anterior limit of the pericardial space and is bent backward. Pacing from the ablation catheter (white arrow) with the balloon positioned at this position showed no phrenic nerve capture. Note the pericardial sheath straightens, indicating tension from forward pressure applied in the balloon catheter. Compared to panel A, the distance between the balloon and the coronary sinus catheter is seen to be substantially greater, indicating a posterior displacement of the balloon. Ablation was performed at this position with successful termination of the arrhythmia. **C:** Termination of atrial tachycardia seen with application of radiofrequency energy. The application of radiofrequency energy was performed after the sheath maneuver displayed in panel B was performed and pacing from this site demonstrated the absence of phrenic nerve capture. Postablation, the right phrenic nerve was intact and displayed normal function.

References

1. Bai R, Patel D, Di Biase L, et al. Phrenic nerve injury after catheter ablation: should we worry about this complication. J Cardiovasc Electrophysiol 2006;17: 944–948.
2. Sacher F, Monahan KH, Thomas SP, et al. Phrenic nerve injury after atrial fibrillation catheter ablation: characterization and outcome in a multicenter study. J Am Coll Cardiol 2006;47:2498–2503.
3. Buch E, Vaseghi M, Cesario DA, Shivkumar K. A novel method for preventing phrenic nerve injury during catheter ablation. Heart Rhythm 2007;4:95–98.
4. Matsuo S, Jaïs P, Knecht S, Lim KT, Hocini M, Derval N, Wright M, Sacher F, Haissaguerre M. Images in cardiovascular medicine: novel technique to prevent left phrenic nerve injury during epicardial catheter ablation. Circulation 2008;117:e471.
5. Di Biase L, Burkhardt JD, Pelargonio G, et al. Prevention of phrenic nerve injury during epicardial ablation: comparison of methods for separating the phrenic nerve from the epicardial surface. Heart Rhythm 2009;6:957–961.
6. Lee JC, Steven D, Roberts-Thomson KC, Raymond JM, Stevenson WG, Tedrow UB. Atrial tachycardias adjacent to the phrenic nerve: recognition, potential problems, and solutions. Heart Rhythm 2009;6:1186–1191.
7. Sosa E, Scanavacca M, d'Avila A, Pilleggi F. A new technique to perform epicardial mapping in the electrophysiology laboratory. J Cardiovasc Electrophysiol 1996;7:531–536.
8. Lachman N, Syed F, Habib A, Bisco SE, Venkatachalam KL, Asirvatham SJ. Correlative anatomy for the electrophysiologist. Part I: The pericardial space, oblique sinus, transverse sinus. J Cardiovasc Electrophysiol 2010;21:1421–1426.
9. D'Avila A, Scanavacca M, Sosa E, Ruskin J, Reddy V. Pericardial anatomy for the interventional electrophysiologist. J Cardiovasc Electrophysiol 2003;14:422–430.
10. Matsumoto Y, Krishnan S, Fowler SJ, Saremi F, Kondo T, Ahsan C, Narula J, Gurudevan S. Detection of phrenic nerves and their relation to cardiac anatomy using 64-slice multidetector computed tomography. Am J Cardiol 2007;100: 133–137.