Intentional pneumothorax avoids collateral damage: Dynamic phrenic nerve mobilization through intrathoracic insufflation of carbon dioxide

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Introduction

Phrenic nerve injury, with resultant impairment to diaphragmatic function,[1,2] is a well-recognized and feared complication of catheter ablation. Considering the proximity of the phrenic nerves to multiple cardiac structures[3] critical to arrhythmia initiation and maintenance, it is unsurprising that phrenic nerve injury has been reported after ablation of various arrhythmias.[4–6] For example, reported incidence of phrenic nerve injury exceeds 11% in trials of catheter ablation of atrial fibrillation utilizing radiographic screening.[7]

Although multiple approaches to prevent phrenic nerve injury during ablation have been described, these strategies can be difficult to implement, with unsatisfactory effectiveness and safety profiles. Here we present a case describing a novel technique utilizing an intentional pneumothorax to displace the phrenic nerve, allowing for successful and safe ablation of a focal right atrial tachycardia after other approaches had failed.

KEY TEACHING POINTS

- Phrenic nerve injury, with resultant impairment to diaphragmatic function, is a well-recognized and feared complication of catheter ablation.
- Existing strategies to prevent phrenic nerve injury during ablation through nerve displacement via percutaneous pericardial instrumentation can be difficult to implement, with unsatisfactory effectiveness and safety profiles.
- Intentional pneumothorax with intrathoracic carbon dioxide insufflation and video-assisted thoracoscopy provides direct visualization and reversible displacement of the phrenic nerve, enabling successful and safe ablation of a focal atrial tachycardia without invading the pericardium.

Case report

A 67-year-old woman was evaluated for 5 years of frequent, highly symptomatic palpitations with supraventricular tachycardia captured on ambulatory electrocardiographic monitor that correlated to symptoms. After medical therapy failed to adequately control symptoms, electrophysiology study was performed at an outside institution with ablation deferred owing to proximity of the phrenic nerve to the tachycardia focus, with concern for iatrogenic palsy. Considering symptom severity despite medical therapy, we offered the patient a repeat attempt at ablation utilizing the following strategies to avoid phrenic nerve injury: (1) cryoablation with simultaneous phrenic nerve monitoring via pacing and (2) mechanical displacement of the phrenic nerve through percutaneous subxyphoid pericardial instrumentation. Displacement approaches would include (1) an 8.5 French steerable Agilis introducer sheath (Abbott, St. Paul, MN), (2) pericardial balloon inflation (Tyshak II percutaneous transluminal 40 × 20-mm valvuloplasty catheter; Braun Interventional Systems Inc, Bethlehem, PA), and (3) pericardial saline injection.

During repeat electrophysiology study at our institution, transvenous electrode catheters were placed in the standard fashion, which included a decapolar catheter advanced to the distal coronary sinus and quadripolar catheters placed in the high right atrium, His position, and right ventricular apex. Sustained narrow complex tachycardia (cycle length: 450–520 ms), with a 1:1 A to V relationship, was reliably

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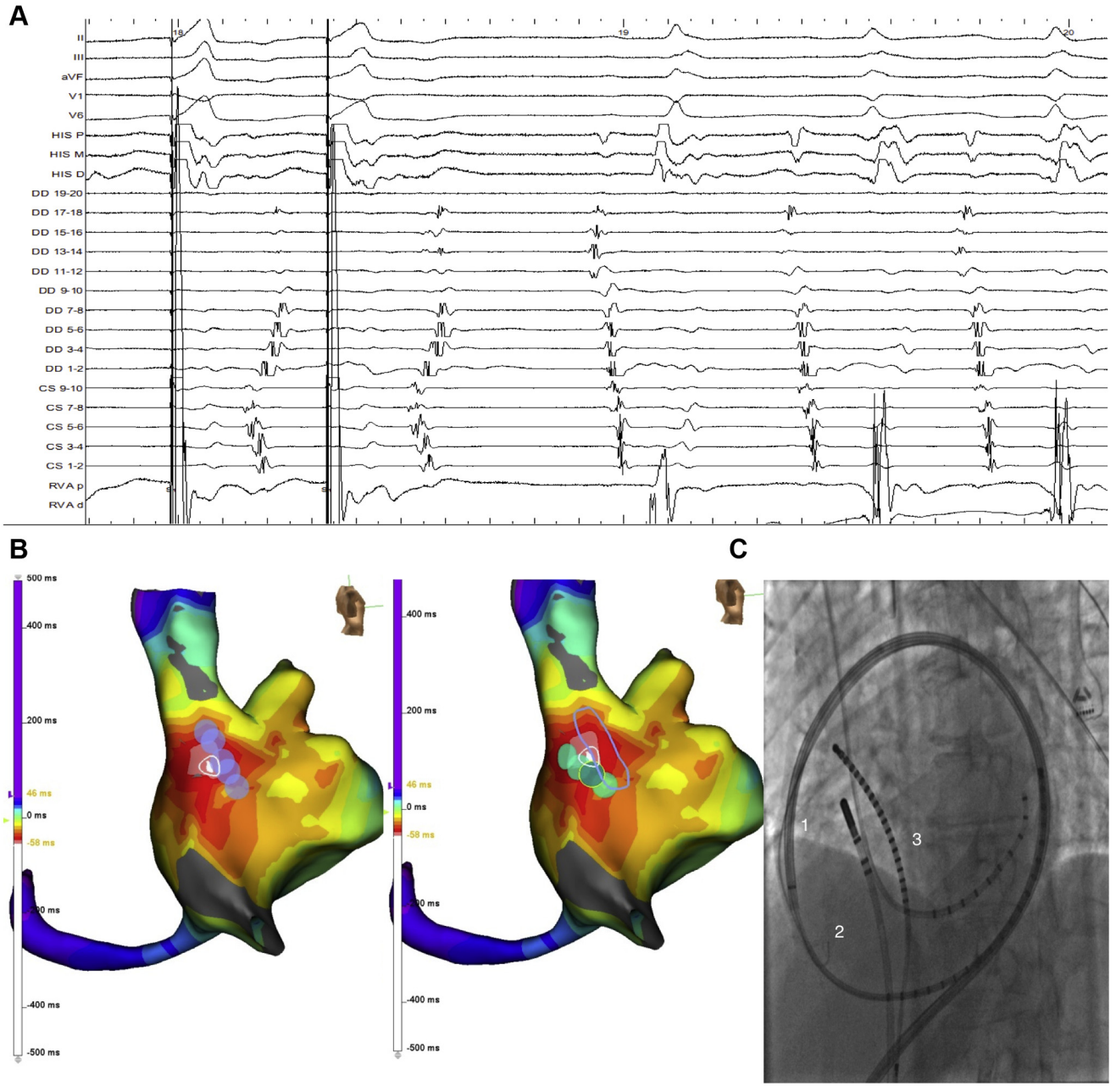

Figure 1 **A:** Ventricular-overdrive pacing during clinical tachycardia with (1) V-A-A-V response and (2) distinct atrial activation sequences and P-wave morphologies between clinical tachycardia and pacing. **B:** Activation mapping during clinical tachycardia with (1) *white outline* denoting earliest activation, (2) *purple tags* denoting locations of phrenic nerve capture, and (3) *light green tags* denoting ineffective cryoablation lesions and *dark green tag* denoting cryoablation lesion that resulted in temporary dampening of diaphragmatic excursion. **C:** Attempted phrenic nerve displacement through percutaneous subxyphoid pericardial instrumentation with (1) an 8.5 French steerable sheath and a steerable catheter, (2) placeholder J wire for a second pericardial access, and (3) endovascular 4 mm mapping/ablation catheter, duodecapolar catheter (DD) for phrenic nerve pacing, and decapolar coronary sinus catheter (CS). d = distal; HIS = His bundle catheter; m = mid; p = proximal; RVA = right ventricular apex catheter.

induced by single atrial extrastimuli from the high right atrial catheter (400:240 ms). Atrial activation sequence was high to low, with concentric activation, and a "VAAV" response was observed following ventricular overdrive pacing. An electroanatomic map of the right atrium and caval veins was constructed using EnSite Precision (Abbott) with activation mapping consistent with a focal mechanism emanating from the high posterolateral right atrium (earliest activation 23 ms before surface P-wave initiation). Pace mapping of the phrenic nerve showed immediate adjacency to the atrial tachycardia focus. An attempt at cryoablation via an 8 mm Freezor Max cryocatheter (Medtronic, Minneapolis, MN) was quickly aborted owing to dampened diaphragm contraction without effect on the clinical tachycardia. Multiple attempts at pericardial displacement of the phrenic nerve utilizing approaches described above did not result in loss of nerve capture at the target site and further ablation was deferred (Figure 1).

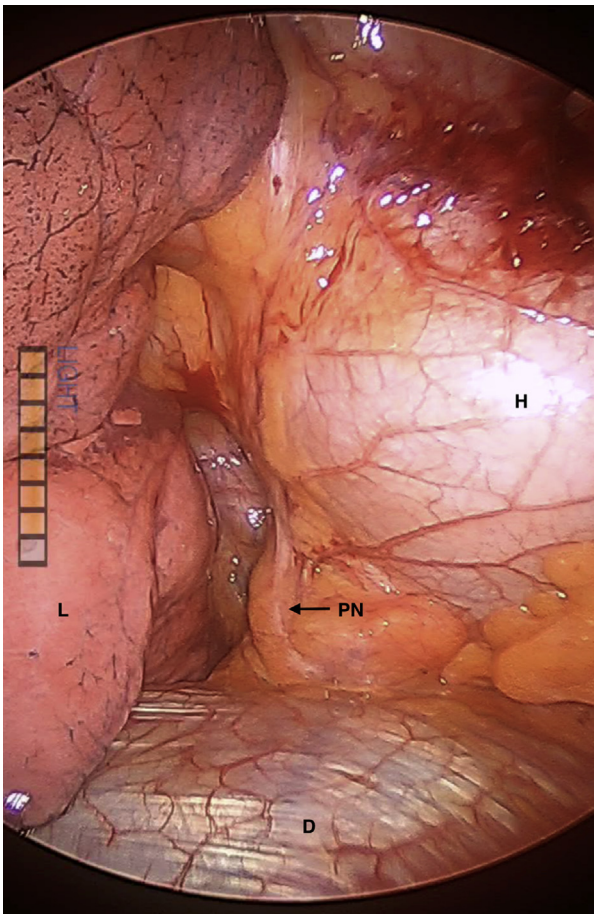

Figure 2 Thoracoscopic image of heart (H), lung (L), diaphragm (D), and posteriorly displaced phrenic nerve (PN) after intentional pneumothorax with carbon dioxide insufflation.

In pursuit of definitive treatment, we opted to bring the patient back to the lab for a hybrid approach utilizing endomyocardial catheter ablation paired with video-assisted thoracoscopic surgical mobilization and displacement of the phrenic nerve. Under general anesthesia, the left mainstem bronchus was intubated and we elected for endomyocardial electrophysiology study prior to video-assisted thoracoscopic surgical mobilization. Electrophysiology study and electroanatomic mapping (CARTO 3 System; Biosense Webster, Irvine, CA) again demonstrated an easily inducible focal atrial tachycardia emanating from the high posterolateral right atrium, with directly overlapping phrenic nerve capture despite single-lung ventilation. Therefore, the right pleural space was insufflated with carbon dioxide, utilizing a standard 3-port approach, resulting in intentional partial pneumothorax. Under direct visualization, we noted posterior displacement of the phrenic nerve from its typical course (Figure 2) and again opted for repeat endomyocardial phrenic nerve mapping.

Following intentional pneumothorax, the phrenic nerve could no longer be captured at the target site, or anywhere along its original course, with high-output pacing (20 mA at 2 ms). In clinical tachycardia, utilizing a ThermoCool SmartTouch SF ablation catheter (Biosense Webster), we ablated the target site with tachycardia termination in less than 2 seconds. A rosette of lesions was created surrounding the termination site, with 250 seconds of total ablation. After a 30-minute waiting period, no arrhythmias were inducible with and without isoproterenol infusion up to 20 mcg/min. To confirm a causative role of the iatrogenic pneumothorax in displacement of the phrenic nerve, we aspirated the pneumothorax, with intubation of the left mainstem bronchus maintained, and captured the phrenic nerve with endomyocardial pacing at the site of termination and along its original course (Figure 3).

The patient tolerated the procedure well, with an uneventful 48-hour postoperative inpatient course that included a chest tube to water seal for 24 hours. At discharge, the patient was without pneumothorax, with intact bilateral diaphragm function. The patient remains free of symptoms, off medications, with ambulatory electrocardiographic monitoring demonstrating no tachycardia recurrence.

Discussion

Considering iatrogenic phrenic nerve injury can result in symptoms that match or exceed the symptoms of arrhythmia that prompted ablation,[2] utilization of phrenic nerve mapping before ablation[8] and simultaneous monitoring for phrenic nerve injury during ablation are the standard of care. For ablation sites with especially high risk of phrenic nerve injury, several proposed methods exist to protect the nerve from injury.[4,9] However, as demonstrated in our case, development of innovative strategies to prevent phrenic nerve injury during catheter ablation are warranted.

When risk of phrenic nerve injury with ablation is present, the only approach that eliminates risk is deferred ablation, which should be thoughtfully considered. If ablation is pursued, an "ablate and wait" approach has traditionally been implemented, which involves delivery of low-power, short-duration radiofrequency therapy or cryotherapy while simultaneously assessing for weakening or loss of diaphragmatic excursion with phrenic nerve pacing. However, this approach has been associated with late recurrence despite acute procedural success.[10] Additionally, an "ablate and wait" approach does not eliminate the risk of phrenic nerve injury and has been lampooned as "closing the door after the horse has left the barn."[11]

Mechanical displacement of the phrenic nerve from the myocardium to minimize risk of injury is intuitive, with several subxyphoid pericardial approaches described, including (1) gauze packing with forceps,[12] (2) filling the pericardial space with saline and/or air,[9] and (3) a steerable sheath combined with an inflatable balloon or a steerable catheter.[4,13] However, these approaches are not uniformly successful at eliminating phrenic nerve capture, even when used in combination,[9,14] which was again demonstrated in our case. Notably, these approaches are challenging to employ, requiring frequent equipment adjustments or an outright change in approach.[13] Additionally, complication

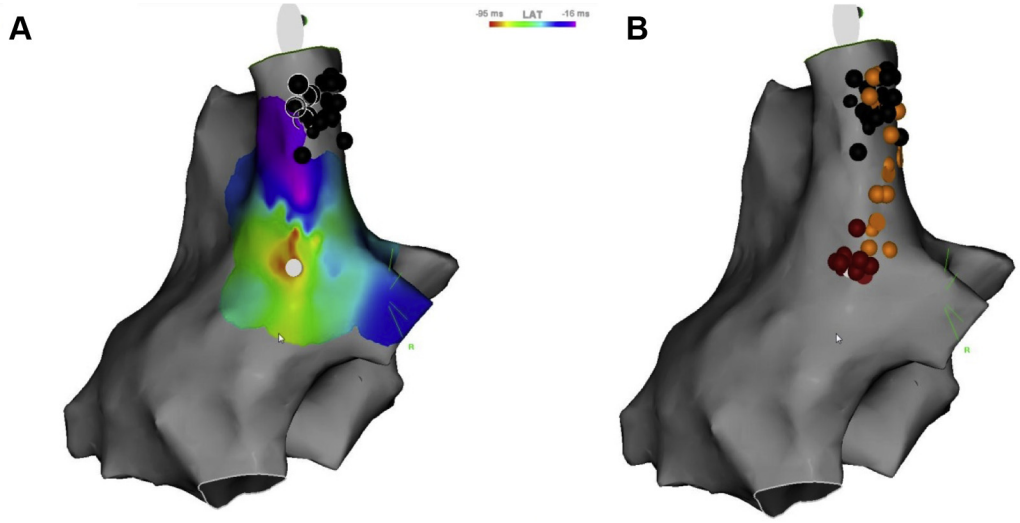

Figure 3 **A:** Earliest activation during clinical tachycardia (*white tag*) and phrenic nerve capture locations with right lung collapsed (*black tags*) after carbon dioxide insufflation. **B:** Ablation lesions with termination of tachycardia (*red tags*). *Black tags* and *orange tags* denote phrenic nerve capture locations with and without collapsed lung, respectively.

rates can exceed 20%; complications include pericardial bleeding, myocardial puncture, pericarditis, and pleuropericardial fistulas.[13] Considering the degree of crude manipulation these approaches can require, it is not inconceivable that traumatic phrenic nerve injury could result.

Our case describes a novel approach to prevent phrenic nerve injury during catheter ablation of a focal right atrial tachycardia via intentional pneumothorax created through a minimally invasive surgical approach without invading the pericardium. In our patient, the target ablation site in the high posterolateral right atrium was immediately adjacent to the phrenic nerve, with failure of a cryotherapy "ablate and wait" approach and multiple previously described approaches to displace the phrenic nerve with percutaneous pericardial instrumentation. Fundamentally, previously described phrenic nerve displacement approaches aim to increase the space between the myocardial wall and phrenic nerve. However, inward compression from thoracic structures, including the inflated lungs, maintains a constant myocardial wall–phrenic nerve relationship and resists phrenic nerve displacement. By additionally insufflating the chest cavity with carbon dioxide in our case, an intended pneumothorax resulted in reversible posterior displacement of the phrenic nerve in relation to the right atrium that could be directly visualized, allowing for ablation without risk of phrenic nerve injury at the atrial tachycardia focus. Of note, after the right pneumothorax was aspirated and while intubation of the left mainstem bronchus was maintained, the phrenic nerve was not displaced, confirming the pneumothorax as the causative intervention that resulted in displacement. Importantly, pleural carbon dioxide insufflation with and without single-lung ventilation has been shown to be safe,[15,16] although there are theoretical complications[17] and its use requires collaboration with an experienced anesthesia team. Additionally, in the event that pneumothorax does not result in sufficient phrenic nerve displacement to safely attempt ablation, this approach also allows for surgical pericardiotomy and phrenic nerve dissection and displacement. However, considering the invasive nature of this procedure, we first recommend exhaustion of more conservative percutaneous pericardial-based approaches to displace the phrenic nerve, as was done in this case.

Conclusion

Existing strategies to prevent phrenic nerve injury during ablation through nerve displacement via percutaneous pericardial instrumentation can be difficult to implement, with unsatisfactory effectiveness and safety profiles. Intentional pneumothorax with intrathoracic carbon dioxide insufflation and video-assisted thoracoscopy provides direct visualization and reversible displacement of the phrenic nerve, enabling successful and safe ablation of a focal right atrial tachycardia without invading the pericardium.

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