Epicardial Phrenic Nerve Displacement During Catheter Ablation of Atrial and Ventricular Arrhythmias

Procedural Experience and Outcomes

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Background—Arrhythmia origin in close proximity to the phrenic nerve (PN) can hinder successful catheter ablation. We describe our approach with epicardial PN displacement in such instances.

Methods and Results—PN displacement via percutaneous pericardial access was attempted in 13 patients (age 49±16 years, 9 females) with either atrial tachycardia (6 patients) or atrial fibrillation triggered from a superior vena cava focus (1 patient) adjacent to the right PN or epicardial ventricular tachycardia origin adjacent to the left PN (6 patients). An epicardially placed steerable sheath/4 mm-catheter combination (5 patients) or a vascular or an esophageal balloon (8 patients) was ultimately successful. Balloon placement was often difficult requiring manipulation via a steerable sheath. In 2 ventricular tachycardia cases, absence of PN capture was achieved only once the balloon was directly over the ablation catheter. In 3 atrial tachycardia patients, PN displacement was not possible with a balloon; however, a steerable sheath/catheter combination was ultimately successful. PN displacement allowed acute abolishment of all targeted arrhythmias. No PN injury occurred acutely or in follow up. Two patients developed acute complications (pleuro-pericardial fistula 1 and pericardial bleeding 1). Survival free of target arrhythmia was achieved in all atrial tachycardia patients; however, a nontargeted ventricular tachycardia occurred in 1 patient at a median of 13 months’ follow up.

Conclusions—Arrhythmias originating in close proximity to the PN can be targeted successfully with PN displacement with an epicardially placed steerable sheath/catheter combination, or balloon, but this strategy can be difficult to implement. Better tools for phrenic nerve protection are desirable. (Circ Arrhythm Electrophysiol. 2015;8:896-904. DOI: 10.1161/CIRCEP.115.002818.)

Key Words: atrial fibrillation ■ atrial tachycardia ■ catheter ablation ■ phrenic nerve ■ phrenic nerve injury ■ phrenic nerve protection ■ ventricular tachycardia

Collateral phrenic nerve (PN) injury can occur after endocardial catheter ablation of atrial fibrillation (AF), atrial tachycardias (AT), left-sided accessory pathways, and epicardial ablation of ventricular tachycardia (VT). Right PN injury during ablation of atrial arrhythmias originating from the crista terminalis, superior vena cava (SVC), or the right upper pulmonary vein can be explained by the anatomic proximity to the PN as it courses down the anterolateral wall of the SVC, with a distance as little as 0.3±0.5 mm and only the pericardium separating these structures. Similarly, VT originating from the basal inferolateral left ventricular epicardium is often in close proximity to the course of the left PN, with a distance <3 mm from the epicardial surface in ≤36% of patients. When present, PN injury can be varied in symptomatology (from asymptomatic to severe respiratory insufficiency) and temporality of recovery (median of 6–8 months but may take ≤28 months). Furthermore, PN injury may persist in ≤33% of patients in long-term follow-up causing significant morbidity.

Several techniques for avoiding PN injury have been reported, including pace mapping to identify sites close to the PN to avoid ablation, use of lower power settings (10 watts in one study), and PN displacement using an epicardially placed balloon, sheath/catheter combination, and pericardial injection of saline, air, or both. The experience with PN displacement techniques is limited to animal studies, isolated case reports, or small series of patients. Although these reports have been favorable in providing PN protection and arrhythmia cure, the particular technical challenges and limitations of these approaches have not been as well appreciated. In this study, we report our procedural experience as well as acute and medium-term outcomes in 13 patients with debilitating atrial and ventricular arrhythmias originating in close proximity to the PN, where PN displacement was used to assist with catheter ablation.
WHAT IS KNOWN

- The phrenic nerve can lie close proximity to site of endocardial origin of atrial arrhythmias or epicardial origin of ventricular arrhythmias.
- This may hamper success of catheter ablation and cause collateral phrenic nerve injury.
- Experience with phrenic displacement is limited to case reports, small series, or animal experiments.

WHAT THE STUDY ADDS

- Arrhythmias originating in close proximity to the phrenic nerve can be targeted successfully with phrenic nerve displacement strategies.
- These include the use of an epicardially placed steerable 4 mm-catheter combination or the use of an esophageal or gastrointestinal balloon delivered via a steerable sheath.
- This approach allows successful endocardial ablation of focal atrial tachycardia of right atrial origin and epicardial ablation of ventricular arrhythmias.
- Although acute noninducibility and freedom from target arrhythmia may be achieved in the majority of patients, significant technical and procedural challenges and risks of complications must be appreciated.

Methods

From January 2008 to December 2014, 6000 catheter ablation procedures were performed at our institution, which included catheter ablation for AF (n=2971), focal AF (n=319), supraventricular tachycardia (n=685), and VT (n=2025). From this group, 13 patients (0.22%) with atrial or ventricular arrhythmias originating in close proximity to the PN in whom epicardial PN displacement was used was included in this study. Of note, PN displacement was required in 13/208 (4.4%) procedures that required epicardial access. Procedures were performed according to protocols approved by the Brigham and Women’s Hospital Human Subject Protection Committee. The present study overlaps and expands on the population reported from our center previously. All patients gave written informed consent for their procedure.

Mapping of Atrial Arrhythmias

 Intravenous conscious sedation was performed with midazolam and fentanyl for mapping of the atrial arrhythmias. Once the decision was made to obtain pericardial access, the patient was transitioned to general anesthesia, when available for patient comfort. Paralytic agents were not administered to avoid masking PN injury. Intracardiac catheters were inserted via the femoral veins, which included a 20-pole catheter placed along the lateral right atrium with the distal poles in the coronary sinus, His-bundle catheter, right ventricular apical catheter, a multipolar mapping catheter (circular mapping or a multi-spline catheter [Pentaray, Biosense Webster, Diamond Bar, CA]), in addition to a mapping and ablation catheter. An electroanatomic mapping system (CARTO, Biosense-Webster) was used. Bipolar intracardiac electrograms were filtered between 30 and 500 Hz and recorded digitally with simultaneous 12-lead surface electrocardiograms. Site of origin of the AT was determined by examining P wave morphology and using activation mapping during AT that was present in the baseline state or induced with burst atrial pacing with or without adrenergic stimulation (eg, isoprenaline). Radiofrequency (RF) ablation (RFA) was performed using an irrigated or a nonirrigated catheter (Navistar, Thermocool, Thermocool SF, or Thermocool SmartTouch, Biosense Webster). For nonirrigated catheters, power was titrated ≤50 Watts and a temperature of 60°C. Based on the discretion of the treating electrophysiologist, cryoablation was attempted before embarking on epicardial PN displacement.

Before ablation, unipolar pacing was performed with the ablation catheter at 10 mA and 2 ms pulse width to exclude PN capture. If PN capture was present, sites adjacent to earliest activation where PN capture was absent were targeted, unless the patient had a prior failed ablation procedure using this approach. If this was not successful in abolishing the tachycardia, epicardial PN displacement was used. PN displacement was performed based on symptom severity and impairment in quality life and patient preference with consideration of perceived benefits. Post ablation, burst and programmed atrial stimulation was repeated with or without high dose adrenergic stimulation to ensure arrhythmia noninducibility.

Mapping of Ventricular Arrhythmias

Our methods for mapping and ablation of ventricular arrhythmias have been previously reported. Briefly, programmed ventricular stimulation was performed to assess morphologies of induced VTs. Sustained monomorphic VT was defined as continuous VT with an identical 12-lead electrocardiogram morphology and rate (within 20 ms) to a VT that the patient presented with before ablation. If 12-lead electrocardiograms of the presenting VT were not available before ablation, the rate cut off and intracardiac bipolar intracardiac electrograms from the implanted cardioverter defibrillator were used. Undocumented VTs were defined as inducible VTs that did not have an identical rate (>20 ms difference), 12-lead electrocardiograms morphology, or implanted cardioverter defibrillator-derived bipolar intracardiac electrogram characteristics to the VT that the patient had presented with before ablation.

Substrate mapping was performed with particular focus on the scar region facilitated by the CARTO electroanatomic mapping system (Biosense Webster). Areas of low voltage (<1.5 mV) and dense (≤0.5 mV) and electrically unexcitable scar were identified. Late potentials in the scar were tagged. Pace mapping was performed; areas of long stimulus to QRS (S-QRS) delays (>40 ms) and where pace mapping matched QRS morphology of an induced VT were tagged. If hemodynamically tolerated, VT was then reinduced and activation/entrainment mapping performed. If not tolerated, it was terminated with RFA, burst pacing, or cardioversion and substrate mapping performed. Ablation targeted presumptive channels and exits within the low-voltage area, including regions of long S-QRS (>40 ms). RFA was delivered with an irrigated catheter (ThermoCool, or ThermoCool SF; Biosense Webster) at a power of 25 to 50 Watts targeting an impedance drop of 10 to 20 ohms. Applications were repeated at target areas until they were rendered electrically unexcitable with unipolar pacing at 10 mA at 2-ms pulse width. Post ablation, programmed ventricular stimulation was repeated to confirm arrhythmia noninducibility.

Epicardial Access and Phrenic Nerve Protection Approach

Epicardial access was obtained by subxiphoid pericardial puncture as previously described. For AT ablation, only single epicardial access was obtained, unless the first epicardial access failed to provide PN displacement and the operator felt that a change of approach (eg, from anterior to posterior pericardial access or vice versa) was necessary. PN displacement strategies consisted of one or a combination of (1) saline infusion into the pericardial space; (2) epicardially placed balloon, and (3) epicardially placed sheath/nonirrigated 4 mm catheter combination. For the latter, we used a steerable nonirrigated 4 mm catheter to allow sufficient rigidity and size to enable PN catheter displacement. During endocardial ablation of atrial arrhythmias, an epicardially placed balloon or sheath/catheter combination was positioned juxtaposed to the adjacent earliest endocardial site of activation.
such that PN capture was no longer present. Operator preference dictated which tool was applied first and in subsequent intraprocedural attempts if the first attempt was unsuccessful in PN displacement. For epicardial VT ablation, single pericardial access was obtained, and the access was subsequently double wired to allow insertion of 2 sheaths over the wires; one access was for a mapping/ablation catheter delivered via a steerable sheath (Agilis, St Jude Medical, St Paul, Minnesota) and the other for use of the epicardial PN displacement, also delivered via a second steerable sheath. When epicardial ablation was deemed necessary, coronary angiography was performed before initial ablation. Unipolar pacing was performed with the ablation catheter at 10 mA, 2 ms pulse width was performed to determine whether the site of origin was adjacent to the PN; if so, ablation was first attempted at an adjacent site where PN was absent. PN displacement was performed if substrate, pace, activation, and entrainment mapping demonstrated that the critical part of the circuit was adjacent to the PN and the documented VT remained inducible, despite ablation at adjacent sites where no PN capture was evident.

**Maneuvers for PN Displacement**

When PN displacement was performed with an inflated vascular balloon (NMT Medical, Boston, MA; 18 mm×20 mm) or an esophageal balloon (Hercules 3 step 18–20 mm esophageal balloon; Cook Medical, Bloomington, IN), the balloon was delivered through a steerable sheath (Agilis, St Jude Medical). The deflated balloon was first advanced into the pericardial space and manipulated into position near the ablation catheter located at the site of intended ablation. The balloon was then inflated with the intention of displacing the PN away from the epicardium and repeat high-output pacing performed to ensure absence of PN capture. The sheath was used to provide stability and support that would allow the balloon to maintain its position, thereby creating a physical space between the epicardium and the PN or displace the PN away from the region of interest for ablation. Multiple adjustments of the balloon were made, when necessary, which included advancement/retraction, repeated inflations, and deflations to reach the desired location and to maintain its position. If multiple attempts at balloon placement were unsuccessful in PN displacement, or PN capture was still present, a nonirrigated 4 mm catheter and sheath combination were used in an attempt toward PN displacement or vice versa. When the sheath/catheter combination was used, the catheter was directed to the epicardial site of PN capture. The catheter was then advanced to create a physical space between the epicardium and phrenic nerve, such that the nerve was lifted off the epicardial surface.

RFA was attempted endocardially at the site of earliest activation (for AT) or the critical region identified by mapping epicardially (for VT) after ensuring absence of PN capture. After ablation, repeat high output pacing was performed at each site to confirm absence of PN injury by tactile sensation of diaphragmatic stimulation and quantifying fluoroscopic diaphragmatic movement. Before each ablation at a new site, repeat PN pacing was performed to ensure absence of PN capture and diaphragmatic excursion checked on fluoroscopy. Diaphragmatic excursion during respiration was assessed at the end of the procedure using fluoroscopy and compared with that obtained preprocedure.

**Follow Up**

Patients were observed in hospital and examined the following day with assessment of diaphragm excursion on examination. A chest x-ray was obtained the next day if there was concern about possible limited diaphragm motion. Patients were then followed clinically typically at 6 weeks and 3 to 6 monthly intervals thereafter; repeat imaging was obtained as per the discretion of the referring clinician. For patients with AT, periodic monitoring (varying from 24 hour to 30 day) was performed to document recurrence if symptoms were suspicious. In patients with VT, implanted cardioverter defibrillators were interrogated for recurrence of ventricular arrhythmia. Follow up also included review of records of all hospital and outpatient clinic visits and discussion with referring cardiologists and primary care physicians.

**Statistical Analysis**

The Statistical Package for the Social Sciences for Windows (IBM SPSS, release 22, Armonk, New York) was used for analysis. Continuous variables were expressed as mean±standard deviation (normally distributed) or median with ranges (if not normally distributed).

**Results**

Thirteen patients (mean age 49±16 years, 9 females) with atrial or ventricular arrhythmia origin in close proximity to the PN underwent epicardial PN displacement (Table). Patients had highly symptomatic arrhythmia (median of 24 months) and failed multiple prior antiarrhythmic drugs (median 2); all but 3 patients had failed prior catheter ablation attempts (Table). Procedural indication was AT (7 patients), SVC-triggered AF (1 patients), and epicardial VT (Table).

An epicardially placed steerable sheath/catheter combination (5 patients), a vascular balloon (3 patients), or an esophageal balloon (5 patients) was ultimately successful in PN displacement (Figure 1 and Table).

**Atrial Arrhythmias**

In all AT patients, PN capture with pacing was present at the site of earliest activation. All but 1 patient had failed one previous attempt at ablation with RF (6 patients) or both RF and cryoablation (1); one patient had 2 failed RFA attempts. In 3 patients, RFA at sites adjacent to the earliest activation without PN capture had failed. In one patient, multiple positional maneuvers had also failed to prevent PN capture, including a 30 and 45 degree wedge and positioning the patient on their left side. In 2 patients, RFA was prematurely terminated because of transient loss of PN capture during pacing in the SVC during ablation. In one patient with SVC-triggered AF, SVC isolation was prevented by PN proximity (Table).

Following epicardial access, epicardial PN displacement was attempted with a balloon only in 2/8 patients, steerable sheath/catheter combination only in 2/8 patients, and multiple modalities in 4/8 patients (Figure 1). PN displacement was ultimately successful in allowing ablation at the earliest endocardial site using a balloon in 3 patients (vascular balloon 2 and gastrointestinal balloon 1) and combination of steerable sheath/catheter in 5 patients (Figures 1 and 2).

Acutely successful ablation of the clinical AT was possible in all patients. One patient suffered a complication occurred of pericardial bleeding (240 mL) starting immediately after pericardial puncture, which was managed nonoperatively. No patient developed PN injury acutely. After a median follow up of 11 months (range 1–65 months), no patient developed symptoms or signs suggestive of diaphragmatic palsy; 4 out of 8 patients had chest imaging at a range of 1 to 17 days post procedure, which showed no evidence of diaphragmatic dysfunction. Survival free of clinical AT was achieved in all patients at a median follow-up of 11 months (range 1–65 months).

In follow-up, 2 patients developed another AT from the mid crista where PN capture was not evident, which were successfully ablated. Two patients developed presumed inappropriate sinus tachycardia; no recurrence of the index AT was noted after intensive Holter monitoring in both patients and detailed electrophysiological evaluation in 1 patient.
Particular difficulties with PN displacement are worthy of mention. This seemed to be largely related to the limited space between the right superior pulmonary vein and right atrium—SVC region that limited maneuverability in the pericardium near the right phrenic nerve. Access was attempted directly from the anterior pericardium, as well as by attempting to insert the catheter or balloon through the transverse sinus entered from the posterior aspect of the left atrium (Figure 2).27,28 First, 4/8 patients (50%) required multiple modalities (eg, balloon, steerable sheath/catheter combination) before epicardial PN displacement was ultimately successful. In one patient, multiple modalities were used, including instillation of pericardial fluid and use of a steerable sheath/catheter combination, followed by a use of a 30 × 3 mm vascular balloon, which were both unsuccessful, eventually requiring a larger (18 × 20 mm) vascular balloon for displacement (Figure 1). Illustrative case examples are shown in Figure 3 and in Data Supplement.

Second, in 3/6 patients (50%), attempted balloon placement was unsuccessful in allowing PN protection; a steerable sheath/catheter combination was ultimately successful in these patients.

Third, 3/8 patients (38%) required 2 separate pericardial punctures because of difficulties in PN displacement. In the first patient, the balloon length and tip stiffness caused displacement of the sheath/balloon apparatus anteriorly, away from the epicardial PN site, despite either switching from a posterior to anterior pericardial access; a sheath/catheter combination was ultimately successful. In the remaining 2 patients, the initial anterior access failed to provide consistent PN displacement. The first patient had failed PN displacement with the anteriorly delivered balloon; although posterior balloon delivery was ultimately successful, it would provide only a small area of consistent PN protection, and balloon inflation resulted in displacement of the right atrial anatomy necessitating remapping of the AT. In the second patient, anterior balloon placement did not provide consistent PN displacement, ultimately requiring a posteriorly delivered steerable sheath/catheter combination. Finally, in 2 patients with AT, remapping of the AT was necessary because of distortion of right atrial anatomy once the balloon was inflated.

**Ventricular Tachycardia**

Five patients with epicardial VT related to nonischemic cardiomyopathy underwent epicardial PN displacement. Representative case examples are shown in Figure 4 and in Data Supplement. Among the group of VT patients, there were a

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**Table. Baseline and Procedural Characteristics of Patients Undergoing Phrenic Nerve Displacement**

<table>
<thead>
<tr>
<th></th>
<th>Overall (n=13)</th>
<th>Atrial Arrhythmias (n=8)</th>
<th>Ventricular Arrhythmia (n=5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean±SD, years</td>
<td>40±15</td>
<td>35±13</td>
<td>49±16</td>
</tr>
<tr>
<td>Females, n</td>
<td>9</td>
<td>8</td>
<td>1</td>
</tr>
<tr>
<td>LV ejection fraction, mean±SD,%</td>
<td>52±13</td>
<td>60±5</td>
<td>40±12</td>
</tr>
<tr>
<td>Arrhythmia type</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Symptom duration, median (range)</td>
<td>24 (3–96)</td>
<td>38 (6–96)</td>
<td>13 (3–33)</td>
</tr>
<tr>
<td>Number of failed AADs, median (range)</td>
<td>2 (1–5)</td>
<td>2 (1–5)</td>
<td>3 (2–4)</td>
</tr>
<tr>
<td>Number of prior failed ablations, median (range)</td>
<td>1 (0–3)</td>
<td>1 (0–3)</td>
<td>1 (0–2)</td>
</tr>
<tr>
<td>Fluoroscopy time, minutes, median (range),</td>
<td>53 (35–70)</td>
<td>55 (35–70)</td>
<td>51 (36–54)</td>
</tr>
<tr>
<td>Anesthesia type</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Conscious sedation</td>
<td>2</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>General anesthesia from outset</td>
<td>5</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>Conscious sedation then general anesthesia for PN displacement</td>
<td>6</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>PN displacement during first procedure</td>
<td>3</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>PN displacement after prior failed ablation because of PN proximity</td>
<td>10</td>
<td>7</td>
<td>3</td>
</tr>
<tr>
<td>Radiation dose, mGy, median (range)</td>
<td>274 (40–2490)</td>
<td>126 (40–274)</td>
<td>1210 (435–2490)</td>
</tr>
<tr>
<td>Procedure duration, minutes, median (range)</td>
<td>253 (210–366)</td>
<td>225 (210–366)</td>
<td>278 (241–337)</td>
</tr>
<tr>
<td>Number of tachycardias targeted, median</td>
<td>1 (1–2)</td>
<td>1</td>
<td>1 (1–2)</td>
</tr>
<tr>
<td>Number of tachycardias successfully ablated, median</td>
<td>1 (1–2)</td>
<td>1</td>
<td>1 (1–2)</td>
</tr>
<tr>
<td>Successful PN displacement</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Balloon</td>
<td>8</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>Sheath/Catheter</td>
<td>5</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>Complications</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>PN injury (acute)</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>PN injury (follow up)</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Survival free of target arrhythmia</td>
<td>13</td>
<td>8</td>
<td>5</td>
</tr>
</tbody>
</table>

AADs indicates antiarrhythmic drugs; AF, atrial fibrillation; AT, atrial tachycardia; LV, left ventricular; mGy, milligray; PN, phrenic nerve; SD, standard deviation; SMVT, sustained monomorphic ventricular tachycardia; and SVC, superior vena cava.
total of 12 inducible VTs (median 2, range 1–4), of which 7 VTs were spontaneous and 5 VTs were undocumented. All VTs were consistent with scar-mediated re-entry. Seven of 12 inducible VTs had evidence for critical parts of the re-entry circuit in close proximity to the left PN established by the presence of late potentials with S-QRS delays >40 ms and ≥11/12 pace map match and entrainment mapping. Critical sites were located in the basal lateral (4 patients) and mid/apical lateral left ventricular epicardium (1 patient) directly underneath the left PN. In all patients, these sites had evidence of PN capture. Three patients had previous RFA attempts at adjacent epicardial sites where PN capture was not evident; however, VT had recurred in all.

All patients underwent successful PN displacement using the vascular (1 patient) or the gastrointestinal balloon (5 patients). In 2 patients, absence of PN capture was difficult to achieve, requiring frequent repositioning (Figure 4), and was achievable only once the balloon was directly over the ablation catheter.

Successful ablation of the targeted (and spontaneous) VTs was possible in all patients. Acute noninducibility of any VT was achieved in 4/5 patients; 1 patient had 2 undocumented VTs inducible post ablation. No patients developed PN injury either acutely. Complications occurred in 1 patient who develop a pleuro-pericardial fistula and moderate peri-carditis; this resolved during treatment with colchicine. After

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**Figure 1.** A, Flow diagram showing epicardial phrenic nerve (PN) displacement in patients with atrial arrhythmias. Repeat intraprocedural attempts involving the balloon required switch from an anterior to a posterior pericardial access or vice versa. Note, in patients with ventricular arrhythmias, only the epicardial balloon was used, which was successful in all cases (see text for details). B, Method of PN displacement and their success according to the type of arrhythmia.
a median follow-up of 13 months (range 1–65 months), no patient develop symptoms or signs suggestive of diaphragmatic palsy; 4 out of 5 patients had chest imaging at a range of 1 to 39 days post procedure, which showed no evidence of diaphragmatic dysfunction. Survival free of any VT was achieved in all but 1 patient at a median follow-up of 13 months (range 1–65 months). One patient who had VT recurrence appeared to have recurrence of a different VT than that which was targeted during the PN displacement based on implanted cardioverter defibrillator interrogation.

Discussion
This study describes the procedural experience of 13 patients with atrial or ventricular arrhythmias originating within close
proximity to the PN in whom epicardial PN displacement strategies were used in preventing PN injury. PN displacement was achieved with an epicardially placed vascular or gastrointestinal balloon (62% of patients) or an epicardially placed sheath/4 mm catheter combination (38%). This allowed successful endocardial ablation-targeted ATs and SVC isolation that were limited by close proximity to the right PN and successful epicardial ablation of targeted VTs in close proximity to the left PN. Importantly, no PN injury occurred acutely and in follow-up. Acute noninducibility and freedom from the target arrhythmia in follow-up was achieved in all patients.

Although epicardial PN displacement was ultimately successful in all patients, there were considerable technical and procedural challenges worthy of consideration. First, significant maneuverability of the apparatus was necessary to establish and maintain consistent PN displacement (especially with the balloon). In 2/5 (40%) epicardial VT ablations, absence of PN capture during pacing from the ablation catheter could only be achieved when the balloon was directly over the ablation catheter. Second, in patients with atrial arrhythmias, frequent crossover between modalities was necessary (balloon then sheath/catheter or vice versa), with 50% of attempted balloon placements being unsuccessful in providing PN displacement subsequently requiring a sheath/catheter combination for success. Further, in patients with atrial arrhythmias, 38% of patients required repeat pericardial access to change the approach (eg, anterior to posterior approach or vice versa) to allow adequate PN displacement. Third, remapping was sometimes necessary because of anatomic right atrial distortion created by balloon inflation in patients with atrial arrhythmias. The technical challenges appeared to be particularly pronounced in patients with AT requiring right PN displacement. Comparably, no crossover or repeated pericardial access was necessary in epicardial VT ablation. The data suggests that sheath/catheter combination is a reasonable technique to attempt for right PN displacement during AT ablation, whereas the steerable sheath/balloon is reasonable for left PN displacement during VT ablation. Finally, complications were not infrequent, occurring in 2/13 patients (15%) related to pericardial access; furthermore, fluoroscopy times and radiation exposure were significant. These findings highlight that better tools for phrenic nerve protection are highly desirable.

Prior Studies

Pacing to avoid ablation at sites of capture is the most commonly used approach of PN protection; however, this strategy may not be successful if arrhythmia is of focal endocardial origin directly over the course of the PN (as in crista AT) or a critical component of a re-entrant epicardial VT circuit is directly underneath the course of the PN. Animal studies, isolated case reports, and small series of patients undergoing PN displacement and catheter ablation after pericardial air insufflation,11,17 saline infusion, 17,20 saline plus air instillation, or displacement with an epicardially placed sheath/catheter or a balloon have been reported.2,16,19 Di Biase et al when comparing air, saline, or both versus balloon placement in 8 patients found that the combination of air and saline prevented PN capture in 88% of patients, saline infusion never prevented PN capture, and the balloon was only successful in preventing PN capture in 37% of cases.17 No complications occurred in this cohort. As air is a poor conductor of electricity, concerns about inability to defibrillate the heart have been highlighted with pericardial air insufflation.18,20 Saline infusion causes...
tamponade physiology, and the operators must be vigilant for hemodynamic compromise. Although these reports and series have shown ultimately successful PN protection, the technical and procedural challenges and potential complications have not been appreciated. In this study, we highlight the limitations of current tools requiring frequent crossover, complexities in maneuverability, and frequent need for apparatus repositioning, long procedural times and radiation exposure, technical challenges with mapping, and the attendant risks of pericardial access (and sometimes, repeated access). All these factors must be considered in the risk-benefit analysis of this procedure. In addition, given the rarity of requirement for PN displacement (representing only 0.22% of all catheter ablation procedures at our center), PN displacement should be handled in centers highly experienced with epicardial interventional procedures.

**Limitations**
As this was not a prospective study, no specific order of PN displacement strategies was followed, and the results are likely influenced by accumulation of procedural experience over time. We assessed proximity to the PN by pacing. The optimal means of pacing is not clear, and some advocate higher output pacing than was used in this study. We also do not know whether PN capture at a site would always indicate that ablation would produce PN block. This may particularly be true along the thick crista terminals. It is possible that PN capture happens when the catheter is lying along side the crista terminals with the PN adjacent to the thinner atrial wall. However, we had not been successful in finding a site in the region where PN capture did not occur in these patients. The critical amount of energy needed to produce tissue conduction block while salvaging PN function is unknown. Experimental studies suggest that the PN is exquisitely sensitive to RF energy, with permanent PN injury occurring with nerve temperatures as low as 45°C, indeed in one clinical study, PN injury occurred in 1.1% of patients with ablation at 10 Watts at sites of PN capture during SVC isolation.

We did not perform routine chest imaging to check for diagrammatic dysfunction in all patients; imaging was available in 8/13 patients performed on the discretion of the referring physician. All patients had evidence of normal diaphragmatic excursion at the end of the procedure as assessed by fluoroscopy, and none had clinical signs or symptoms of dysfunction in follow-up. However, it is highly unlikely that delayed PN injury would have manifested.

The sample size of this study was small, but given the rarity of the need for PN displacement (0.22% of all ablation procedures), this series represents one of the largest series from a high-volume center. Multicenter registries may yield further insights into the utility and limitations of different PN protection strategies.

**Conclusions**
Atrial and ventricular arhythmias that originate in close proximity to the PN can be targeted successfully with PN displacement tools, such as an epicardially placed steerable sheath/nonirrigated 4 mm catheter combination or a vascular or esophageal balloon; however, considerable technical and procedural challenges must be appreciated. PN displacement seems particularly effective with use of an epicardially placed balloon placed through a steerable sheath. This study underscores the importance of better tools that allow safe and consistent PN protection.

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**References**


Epicardial Phrenic Nerve Displacement During Catheter Ablation of Atrial and Ventricular Arrhythmias: Procedural Experience and Outcomes

Saurabh Kumar, Chirag R. Barbhaiya, Samuel H. Baldinger, Bruce A. Koplan, Melanie Maytin, Laurence M. Epstein, Roy M. John, Gregory F. Michaud, Usha B. Tedrow and William G. Stevenson

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Supplemental Material

Supplemental Figure Legends

Supplemental Figure 1: 60 female with persistent atrial fibrillation triggered from a SVC focus in whom SVC isolation was hindered by PN capture in its lateral aspect. An epicardially placed sheath/catheter combination (Cath + SS epi) allowed PN displacement to allow successful SVC isolation. Shown are right Abbreviations: Abl-ablation catheter, Cath- catheter, CS- coronary sinus, endo- endocardial, epi-epicardial, LAO-left anterior oblique, RAO-right anterior oblique, SS- steerable sheath, SVC- superior vena cava.

Supplemental Figure 2: 35 year old male with non-ischemic cardiomyopathy who had epicardial VT (A). Substrate mapping showed a preponderance of late potentials (B) within the lateral epicardial LV low voltage scar (C) in close vicinity to the left PN (black dots), prohibiting ablation during the index procedure. Ablation at sites adjacent to late potentials without PN capture did not abolish VT, which recurred in follow up. Repeat attempt at epicardial ablation after PN displacement was successful (D) with abolishment of late potentials and non-inducibility of the targeted VTs originating form close proximity to the PN. Abbreviations: LV- left ventricle.
Supplemental Figure 1

Circular mapping catheter in SVC (endo)

Abl (endo)

Cath + SS (epi)

CS

RAO

Circular mapping catheter in SVC (endo)

Abl (endo)

Cath + SS (epi)

CS

LAO