A 44-year-old man with nonischemic cardiomyopathy was referred for radiofrequency catheter ablation of recurrent sustained monomorphic ventricular tachycardia refractory to sotalol and mexiletine. The recurrent ventricular tachycardia led to multiple appropriate implantable cardioverter–defibrillator interventions. A preprocedural cardiac magnetic resonance showed subepicardial delayed gadolinium enhancement in the basal-lateral left ventricle (Figure [A]). Programmed ventricular stimulation up to 4 extrastimuli from multiple right and left ventricular sites failed to induce any sustained ventricular tachycardia. High-density left ventricular endocardial voltage map showed normal bipolar voltages, with a focal area of unipolar abnormalities (cut-off value 8.3 mV) in the basal-lateral left ventricle, consistent with the area of late gadolinium enhancement at cardiac magnetic resonance. Percutaneous subxiphoid pericardial access was obtained, and epicardial voltage map confirmed a small area of late and fragmented low-voltage electrograms at the basal-lateral left ventricle, opposite to the area of unipolar abnormalities (Figure [B]). Repeat programmed ventricular stimulation from the epicardium also failed to induce any ventricular tachycardia, and decision was made to perform substrate-based ablation targeting the epicardial late potentials. Intraprocedural coronary angiogram revealed no proximity to the coronary arteries. High-output pacing (20 mA, 2-ms pulse width), however, demonstrated intermittent left phrenic nerve (LPN) capture. A deflatable decapolar catheter was advanced to the left subclavian vein and maneuvered to point posteriorly and inferiorly (Figure [C]). High-output pacing (50 mA, 2-ms pulse width) from the distal bipolar of the decapolar catheter resulted in LPN capture (Movie in the Data Supplement); the LPN capture threshold was 30 mA with 2-ms pulse width. Radiofrequency ablation was delivered at a site adjacent to the one demonstrating LPN capture while monitoring for LPN injury during pacing from the left subclavian vein. No weakening or loss of left hemidiaphragm contraction for the entire length of the ablation lesion was confirmed.

Phrenic nerve injury is a well-reported complication of epicardial catheter ablation, which may potentially lead to invalidating symptoms.1–3 LPN injury can also occur after ablation in the region of the left atrial appendage, or during ablation of left lateral accessory pathways.3 The risks of permanent injury of the phrenic nerve may also be higher when using radiofrequency compared with cryoenergy. The most commonly used preventative measure is to pace at high output at the site selected for ablation to evaluate for LPN capture.2 In case of capture, ablation is either aborted or performed after separating the LPN from the epicardial surface. Approaches to separate the LPN from the epicardial surface include balloon inflation and instillation of saline with or without air,3 even with these approaches, however, LPN injury can still occur.1 At variance with the right phrenic nerve, where direct monitoring of injury during ablation is possible by observing the right hemidiaphragm contractility while pacing from the postero-lateral aspect of the superior vena cava, thus far no method has been described to monitor for LPN injury during ablation. We report a novel method to monitor for LPN injury during radiofrequency application, which involves pacing from the inferior and posterior aspect of the left subclavian vein. In this region, the LPN typically enters the thoracic cavity, although, in rare instances, it may cross the subclavian vein anteriorly or directly penetrate the vessel.4 Therefore, occasionally, it may be necessary to manipulate the pacing catheter more anteriorly to ensure phrenic nerve capture. The main limitations of the described technique are (1) that the pacing threshold for the phrenic nerve in this case is higher than the maximal pacing output of many laboratory stimulators and (2) that this technique has not yet been evaluated for sensitivity or specificity or clinical utility. On the basis of this report, an adequately designed prospective study is important to evaluate in how many patients LPN capture from the left subclavian vein is possible and the average threshold for capture (evaluating different current outputs and pulse widths); in particular, because pacing at 50 mA is not available in many laboratories, it would be important to assess in how many cases capture can be achieved at wider pulse widths at 10 and 20 mA. In addition, the clinical usage of this maneuver deserves further evaluation; for instance it is conceivable that a phrenic nerve branch could be injured without appreciating a subjective change in diaphragmatic excursion and that irreversible injury could have occurred by the time loss of diaphragmatic contractility is appreciated.
Conclusions
The LPN can be captured with high output pacing from the left subclavian vein. This may represent a novel method to monitor for LPN injury during catheter ablation.

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None.

References

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