Characterization of the phrenic nerve course within the epicardial substrate of patients with nonischemic cardiomyopathy and ventricular tachycardia

Roger Fan, MD,* Oscar Cano, MD,*† Siew Yen Ho, PhD, FRC Path, FESC,‡ Rupa Bala, MD,* David J. Callans, MD, FHRS,* Sanjay Dixit, MD, FHRS,* Fermin Garcia, MD,* Edward P. Gerstenfeld, MD,* Mathew Hutchinson, MD,* David Lin, MD,* Michael Riley, MD, PhD,* Francis E. Marchlinski, MD, FHRS*

*From the *Electrophysiology Section, Cardiovascular Division, Department of Medicine, University of Pennsylvania School of Medicine, Philadelphia, Pennsylvania, †Universitat Autònoma de Barcelona, Barcelona, Spain, and ‡National Heart and Lung Institute, Imperial College and Royal Brompton and Harefield NHS Trust, London, United Kingdom.*

BACKGROUND Patients with nonischemic cardiomyopathy and ventricular tachycardia (VT) often have low-voltage areas in the lateral left ventricular (LV) epicardium that serve as the VT substrate. The course of the left phrenic nerve in this region may pose a challenge to successful and safe ablation.

OBJECTIVE The purpose of this study was to delineate the left phrenic nerve course in patients with nonischemic cardiomyopathy and suspected epicardial VT and to characterize its relationship with the VT substrate.

METHODS In 10 patients with nonischemic cardiomyopathy undergoing epicardial VT mapping and ablation, the course of the phrenic nerve was defined by pacing. The extent of epicardial LV low-voltage areas (<1.0 mV) was characterized by electroanatomic voltage mapping.

RESULTS Eight of 10 patients had low-voltage areas involving the lateral epicardial LV, and 7 of these 8 patients had sites of phrenic capture within these areas. Ablation was limited due to location of the phrenic nerve in two patients. In one of these patients, a balloon catheter was successfully used to mechanically protect the phrenic nerve during ablation. In the other five patients, adjacent ablation sites were targeted at which no phrenic capture with high-output pacing was demonstrated prior to ablation. In all patients undergoing ablation, the targeted VT became noninducible, and no patient demonstrated phrenic nerve injury.

CONCLUSION In most patients with nonischemic cardiomyopathy undergoing epicardial VT ablation, the phrenic nerve courses through a lateral LV low-voltage area in proximity to potential sites for ablation. Strategies to identify and protect the phrenic nerve are important.

KEYWORDS Nonischemic cardiomyopathy; Ventricular tachycardia; Voltage mapping; Epicardial; Catheter ablation; Phrenic nerve

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Introduction

In most patients with nonischemic cardiomyopathy and ventricular tachycardia (VT), electroanatomic areas of low voltage are found at the base of the left ventricle (LV) adjacent to the mitral valve annulus. The extent of these low-voltage areas often can be greater on the epicardium than on the endocardium.[1–3] The majority of VTs appear to originate from these areas of low voltage.[1,2] Because of the limited effectiveness of endocardial radiofrequency ablation for VT in nonischemic cardiomyopathy, percutaneous epicardial ablation is often required. This technique was pioneered by Sosa et al[4] for treatment of VT in patients with Chagas disease. One of the limitations of epicardial ablation for VT in nonischemic cardiomyopathy is the potential proximity of the left phrenic nerve to the VT substrate, which may limit the safety and efficacy of radiofrequency ablation. Injury to the phrenic nerve and consequent diaphragmatic paralysis is a recognized complication of catheter ablation. Right phrenic nerve injury following catheter ablation of the right superior pulmonary vein ostium, superior vena cava, and lateral right atrium has been reported.[5,6] Left phrenic nerve injury following ablation in the region of the left atrial appendage, a left posterolateral accessory pathway, and during epicardial ablation for VT in nonischemic cardiomyopathy has been reported.[5,7,8]

The purpose of this study was to delineate the course of the left phrenic nerve using pacing maneuvers and to characterize its relationship to the epicardial VT substrate in nonischemic cardiomyopathy. We also sought to determine

Address reprint requests and correspondence: Dr. Francis E. Marchlinski, 9 Founders Pavilion, Hospital of the University of Pennsylvania, 3400 Spruce Street, Philadelphia, Pennsylvania 19104. E-mail address: francis.marchlinski@uphs.upenn.edu. (Received June 22, 2008; accepted September 29, 2008.)

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how frequently the proximity of the phrenic nerve to the VT substrate limited radiofrequency ablation.

Methods

Twenty-three consecutive patients with nonischemic cardiomyopathy underwent epicardial ablation for monomorphic VT. Of these patients, 10 underwent detailed mapping of the phrenic nerve course during the epicardial procedure and were included in the study. All patients were referred to the Hospital of the University of Pennsylvania for electrophysiologic evaluation and catheter ablation. The risk of mapping and ablation were discussed in detail, and all patients gave written informed consent. All procedures were performed following the institutional guidelines of the University of Pennsylvania Health System. In all patients, a decision was made to perform an epicardial ablation because of an unsuccessful endocardial LV ablation before or at the time of the epicardial procedure. The diagnosis of nonischemic cardiomyopathy was established by LV ejection fraction $\leq$0.50 and the lack of significant (>75%) coronary artery disease, prior myocardial infarction, or primary valvular abnormalities. Eight patients had an implantable cardioverter-defibrillator (ICD). All patients presented with spontaneous monomorphic VT that was documented by either ECG or stored electrogram data from the ICD.

Programmed stimulation to attempt VT induction was performed from both right ventricular (RV) and LV sites. The stimulation protocol included delivery of up to triple extrastimuli from multiple ventricular sites at multiple drive cycle lengths.

Pericardial access

Pericardial needle access was obtained via a subxiphoid approach using a Tuohy needle as previously described.[4] This was performed with the patients under general anesthesia. If endocardial LV mapping or ablation was performed, heparin was stopped and fully reversed with protamine prior to pericardial needle access.

Epicardial bipolar voltage mapping

All patients underwent magnetic electroanatomic voltage mapping of RV and LV epicardium. Bipolar RV and LV epicardial electrograms during sinus rhythm (n = 6 patients) or paced rhythm (n = 4 patients) were recorded from either a NaviStar catheter with a 4-mm distal tip electrode or a NaviStar ThermoCool irrigated-tip catheter with a 3.5-mm distal tip electrode (Biosense Webster, Inc., Diamond Bar, CA, USA), both with a 2-mm ring. Bipolar signals were filtered at 30 to 400 Hz and displayed at speed of 200 mm/s on the CARTO system (Biosense Webster). The peak-to-peak signal amplitude was measured automatically. A three-dimensional anatomic shell of the RV and LV epicardium was constructed, and electrogram signals were coupled and displayed as color gradients on a bipolar voltage map. The reference value for defining abnormal electrograms in the LV epicardium has been established based on voltage maps in eight patients with normal LVs.[9] Normal epicardial electrograms were defined as >1.0 mV, which corresponds to 95% of the signals from normal epicardial LV recorded at a distance of at least 1 cm from the defined large vessel coronary vasculature. Dense low-voltage areas were arbitrarily defined as <0.5 mV for display purposes, and the border zone was defined as a transition between dense low-voltage and normal tissue (0.5–1.0 mV). Of importance, low electrogram amplitudes have been described around the atrioventricular groove as well as surrounding the coronary arteries as a result of the normal distribution of fat tissue in the epicardium.[9] However, in these areas, normal electrogram morphology and timing were generally demonstrated. In contrast, the presence of confluent areas of abnormal low voltage always required the presence of low voltage as well as evidence of >80 ms wide, split and/or late electrograms. The abnormal low-voltage area was measured using the area measurement software available on the CARTO mapping system.

Identification of epicardial VT ablation site

If hemodynamically tolerated monomorphic VT was present, activation mapping identified early sites of activation, and entrainment mapping was performed to identify components of the VT circuit as appropriate targets for VT ablation using standard criteria.[10,11] For unmappable VTs, ablation targeted sites of late potentials and pace maps matching the clinical VT.[12]

Phrenic nerve mapping

From the larger cohort of 23 patients with nonischemic cardiomyopathy undergoing epicardial ablation, 10 patients who underwent detailed phrenic nerve mapping were included in this study. The other 13 patients were excluded due to physician preference not to perform the detailed phrenic nerve pace mapping required. A phrenic nerve point was determined by eliciting diaphragmatic stimulation with bipolar pacing at 10 mA at a pulse width of 2 ms from the distal bipole of the ablation catheter. Detailed phrenic nerve mapping was performed by pacing over the anterior and lateral LV in the process of sinus rhythm point acquisition in 1 to 2cm steps. By mapping multiple points of phrenic nerve capture over the anterior and lateral epicardial LV, the course of the phrenic nerve was delineated.

Ablation

Radiofrequency ablation with either a 4-mm NaviStar catheter or a 3.5-mm NaviStar ThermoCool catheter was performed only after coronary angiography demonstrated that the coronary vessels were not in proximity to the ablation site and that there was no phrenic nerve capture at an output of 20 mA with a pulse width of 2 ms. For non-irrigated ablation, power was limited to 50 W for 60 seconds with a maximum temperature of 50°C to 80°C targeting an impedance drop of 10 to 15 Ω. Irrigated ablation used an output of up to 40 W and maximum temperature of 42°C to 45°C. Intrapericardial fluid accumulation was monitored closely and removed by manual withdrawal or by continuous suction. In all patients, the ablation was considered successful if the targeted VT was rendered noninducible with programmed stimulation up to triple extrastimuli at the end of the case.