

Percutaneous Endocardial and Epicardial Ablation of Hypotensive Ventricular Tachycardia with Percutaneous Left Ventricular Assist in the Electrophysiology Laboratory

PAUL A. FRIEDMAN, M.D., THOMAS M. MUNGER, M.D., NORMAN TORRES, M.D.,
and CHARANJIT RIHAL, M.D.

From the Division of Cardiovascular Medicine and the Department of Anesthesiology, Mayo Clinic, Rochester, Minnesota, USA

Percutaneous Endocardial and Epicardial Ablation of Hypotensive VT. Ventricular tachycardia (VT) in the setting of structural heart disease is challenging to treat with percutaneous catheter ablation due to the presence of complex substrate, multiple morphologies, hemodynamic instability, and epicardial circuits. When substrate-based approaches fail, however, it may be impossible to map and ablate hemodynamically unstable arrhythmias. We describe a novel approach to endocardial and epicardial mapping and ablation of hypotensive VT using a percutaneous left ventricular assist device in the electrophysiology laboratory, permitting near-surgical access to cardiac structures. (*J Cardiovasc Electrophysiol*, Vol. 18, pp. 106-109, January 2007)

catheter ablation, ventricular tachycardia, electrophysiology

Introduction

Ventricular tachycardia (VT) in the setting of structural heart disease is challenging to treat with percutaneous catheter ablation due to the presence of complex substrate, multiple morphologies, hemodynamic instability, and epicardial circuits.^{1,2} Ablation of unstable rhythms can be performed with substrate mapping strategies that do not require arrhythmia induction, most notably in patients with coronary artery disease.^{1,2} When these approaches fail, however, it may be impossible to map and ablate hemodynamically unstable arrhythmias.

We describe a novel approach to endocardial and epicardial mapping and ablation of hypotensive VT using a percutaneous left ventricular assist device in the electrophysiology laboratory.

Clinical Case

A 55-year-old man was referred for VT ablation. He had initially presented in 1997 with presyncope and documented nonsustained VT. Echocardiography had demonstrated an ejection fraction of 45%. At electrophysiologic study VT was induced. A dual chamber ICD was implanted. In 2003, following ICD discharge, a coronary angiogram demonstrated normal coronary arteries. Amiodarone was instituted. The patient experienced one or two episodes of dizziness followed by shock due to VT annually. In 2005, due to more frequent shocks, amiodarone was increased to 400 mg daily and subsequently used in combination with propafenone 150 mg three times daily and later mexilitine 200 mg three times daily. Due to lack of rhythm control, an electrophysiology study was performed. Hypotensive VT was induced

that was poorly responsive to antitachycardia pacing (ATP), ultimately requiring shock. Sinus rhythm mapping including pace mapping was performed, and ablation at an anterolateral basal site with a pace-map matching VT did not eliminate arrhythmia. The patient received nine shocks in 2 months, prompting referral to our center for reablation or transplantation.

Ablation Procedure

Preprocedure echocardiography excluded ICD lead thrombus and showed an ejection fraction of 37%. The patient was brought to the electrophysiology laboratory in the postabsorptive state and general anesthesia electively induced. A 7 F sheath was placed via the right internal jugular vein and a Swan Ganz catheter (Edwards Life Sciences, Irvine, CA, USA) positioned. Via the right femoral vein, a 10 F sheath (for intracardiac echocardiography [ICE]) and two 8 F sheaths (for a multipolar CS catheter and an ablation catheter) were placed; a 5 F sheath was placed in the left femoral artery. Programmed stimulation resulted in hypotensive VT with SBP 30 mmHg resistant to ATP, necessitating shock (Fig. 1). Subsequently, a Tuohy needle was advanced via the epigastrium to the pericardial space guided by puffs of contrast, as described by Sosa et al.^{3,4} Upon entry into the pericardial space, a guide wire was advanced, over which an 8 F sheath was positioned to permit epicardial mapping and ablation.

Left ventricular assist

A transeptal puncture was performed under ICE guidance and a 21 F cannula for return of oxygenated blood was positioned in the left atrium (Fig. 2, left panel). Care was taken to position all intake holes across the interatrial septum into the left atrium. A guide wire was positioned in the left common femoral artery and an 8 F Perclose system (Abbot Laboratories, Abbott Park, IL, USA) was used to place sutures around the left femoral percutaneous arteriotomy site. The sutures were not tied, but were positioned for later use ("preclose" technique). A 17 F arterial return cannula was then inserted over a guidewire. The intake and outflow cannulae were

Address for correspondence: Paul A. Friedman, M.D., Division of Cardiovascular Medicine, Mayo Clinic, Rochester, MN 55905. Fax: 507-255-2550; E-mail: pfriedman@mayo.edu

Manuscript received 17 May 2006; Revised manuscript received 5 August 2006; Accepted for publication 8 August 2006.

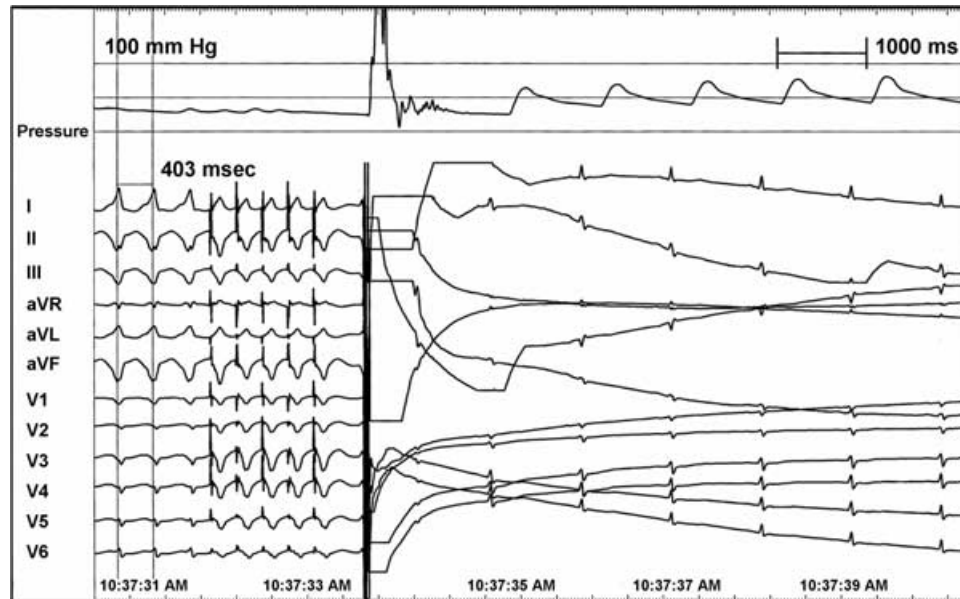


Figure 1. Arterial pressure and 12-surface ECG leads during ventricular tachycardia (cycle length 403 msec). The systolic pressure is 30 mmHg until a shock is delivered (large artifact). The blood pressure gradually rises during normal rhythm following the shock. The last of several rounds of failed burst pacing is seen just before the shock.

connected to the Tandem Heart (CardiacAssist, Inc., Pittsburgh, PA, USA) percutaneous ventricular assist device (PVAD). Flow rates of over 4 L/minute were achieved.^{5,6}

Mapping and ablation

Programmed ventricular stimulation induced the same VT that had been hypotensive. With PVAD support, the VT was hemodynamically tolerated with a minimally pulsatile blood pressure of 95 mm Hg (Fig. 3) and cardiac output that ranged from 4.3 to 7.9 L/minute. Serial measurements of cardiac output, hemoglobin and arterial blood gases were made during bypass support. The patient remained in VT for 1 hour and 45 minutes with LV assist support, during which elec-

troanatomical voltage and activation maps of the arrhythmia were made and endocardial ablation at the inferior base of the LV performed (Fig. 4). After 1 hour and 45 minutes of VT, the systolic blood pressure began to drift downward. Burst pacing failed to terminate the VT, and a shock was delivered. VT remained easily inducible. An angiogram was performed to delineate the coronary arteries, and ablation then delivered epicardially at sites opposite the endocardial linear lesion (Fig. 2, right panel), which also happened to be associated with scar identified on the CARTO map and by intracardiac echocardiography. This rendered the clinical VT noninducible. More aggressive stimulation resulted in additional VTs. One was refractory to ATP and external shock, requiring ICD shock for termination. Due to the difficulty in VT termination, additional inductions were not performed. Additional ablation was guided by pacing mapping and the previously created voltage map.

Postoperative course

Following ablation, the PLVAD was rapidly weaned and discontinued. Cannulae were removed in the electrophysiology laboratory and the previously placed left femoral arteriotomy sutures tied achieving hemostasis without difficulty. The patient was extubated immediately postoperatively. The hemoglobin dropped to 8.4 g/dL in the first 24 hours and two units packed red cells transfused. Physical examination and computed tomography of the neck and abdomen excluded active bleeding, and additional transfusion was not required. Nonsteroidals were administered for postoperative pain. This resulted in a creatinine rise from 1.4 to 2.5 mg/dL, which returned to baseline with medication discontinuation. A moderate pericardial effusion was noted that was observed and remained stable. A left femoral bruit was noted and ultrasound confirmed an arteriovenous fistula, which was observed. The patient was dismissed on amiodarone 200 mg daily. One week postablation the patient exercised 6 minutes on a Naughton protocol with no arrhythmia. Six weeks after ablation the

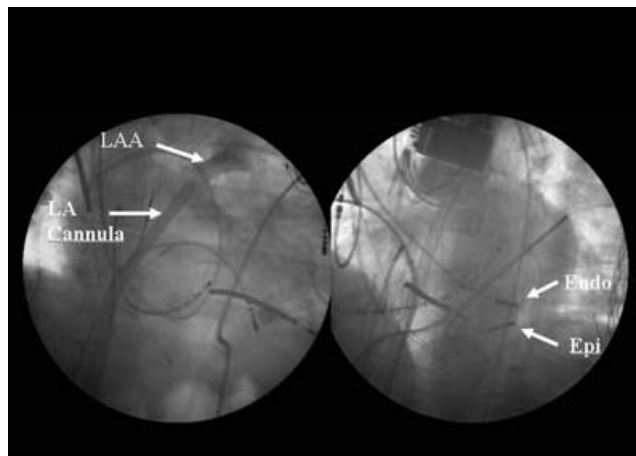


Figure 2. Placement of the PVAD system (left) and endocardial and epicardial mapping (right). In the left panel contrast is delivered via the left atrial (LA) cannula, filling the left atrial appendage (LAA), confirming its position. The right panel depicts the inferobasal position of the endocardial (endo) and the epicardial (epi) percutaneous catheters.

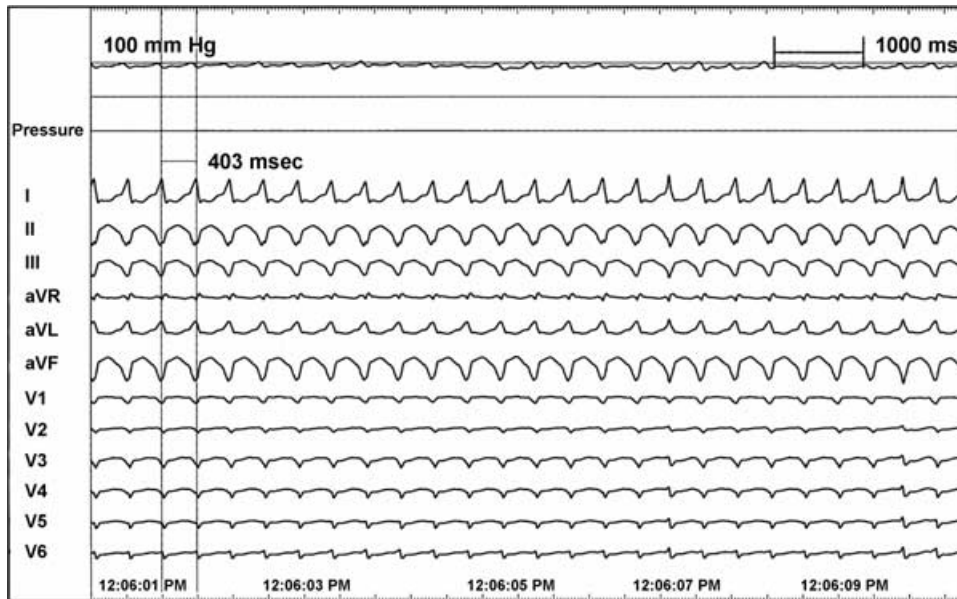


Figure 3. Induction of hypotensive VT during PVAD support. Note that the arterial pressure is 95 mm Hg, but with minimal pulsatility due to external pump support. The pulsatility present likely reflects right ventricular function. The ventricular tachycardia is identical to the rhythm shown in Figure 1.

patient returned to his physically demanding job (lifting heavy crates) without restriction. Two months postablation ICD interrogation showed no arrhythmia, and the patient reported feeling exceptionally well. The patient continues work and feels well 7 months postablation.

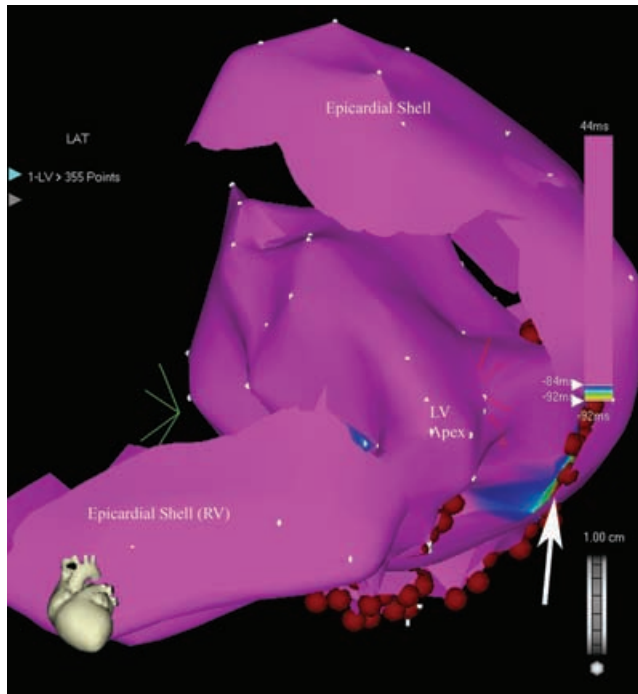


Figure 4. Endocardial and epicardial electroanatomical map of ventricular tachycardia. In this left anterior oblique (LAO) view, the epicardial shell is cut away to reveal the left ventricle. The white arrow indicates site of early activation during VT, with propagation from lateral to septal (note blue color directed toward the right ventricle). Linear ablation lesions (rust spheres) were delivered endocardially and epicardially, and are anchored by the mitral valve (not seen in this view).

Discussion

This case demonstrates in-lab percutaneous endocardial and epicardial ablation of hemodynamically unstable VT by means of percutaneous left ventricular assist to support cardiac output and systemic blood pressure. LV support facilitated successful mapping and ablation of hemodynamically unstable VT in a patient in whom previous substrate-based mapping during sinus rhythm had failed. A hypotensive VT that had required immediate cardioversion was subsequently induced and mapped for nearly 2 hours with percutaneous left ventricular assist support.

Limited conclusions can be drawn from a single procedure. However, this case provides proof of concept of PVAD hemodynamic support and percutaneous endocardial and epicardial structures to permit delivery of therapy that in past required cardiovascular surgery. Hemodynamically supported access to multiple cardiac structures may enhance therapeutic options.

The Tandem Heart PVAD has been previously used in the setting of cardiogenic shock and high-risk percutaneous coronary interventions.^{5,6} In patients with shock and prolonged use (over 72 hours), PVAD support resulted in superior indices of perfusion than intraaortic balloon pump (IABP) therapy, but with no mortality benefit and increased risk of limb ischemia and need for transfusion.^{5,6} Unlike the IABP, the PVAD is not gated to the cardiac rhythm, permitting its use during VT, as in the present case. However, the PVAD does depend on the right ventricle to pump blood through the pulmonary vasculature for oxygenation. Right ventricular fatigue during VT may have contributed to the dropping blood pressure after prolonged VT. Additionally, ventricular fibrillation would not be tolerated despite PVAD support due to loss of RV function.

This case demonstrates both the potential advantages and pitfalls of PVAD support during VT ablation. The ability to map hypotensive VT permits greater definition of the

clinically pertinent arrhythmogenic substrate, possibly improving ablation outcomes. Providing PVAD support requires experienced operators in interventional cardiology, cardiac anesthesiology, cardiac electrophysiology, and it requires extensively trained technicians. Our patient did not have coronary artery disease. However, the experience with PVAD support in coronary disease patients suggests this population should not be excluded.^{5,6} Our patient required a transfusion without obvious bleeding, likely due to blood loss during insertion and removal of the device. Additionally, an arteriovenous fistula developed at the femoral arterial access site, highlighting the potential difficulty associated with large cannulae. In our patient, this did not lead to clinical sequelae. Percutaneous epicardial access was helpful in our case; this approach may be limited in patients with previous coronary bypass. It is possible that combined endocardial and epicardial substrate mapping during sinus rhythm alone would have been effective; however, the PVAD enabled arrhythmia mapping that we found useful.

In summary, we demonstrate the use of left ventricular percutaneous assist to render hypotensive VT tolerable, permitting mapping. Additional experience with this technique

will define its role in the armamentarium of tools used in the electrophysiology laboratory.

References

1. Marchlinski FE, Callans DJ, Gottlieb CD, Zado E: Linear ablation lesions for control of unmappable ventricular tachycardia in patients with ischemic and nonischemic cardiomyopathy. *Circulation* 2000;101:1288-1296.
2. Brunckhorst CB, Delacretaz E, Soejima K, Maisel WH, Friedman PL, Stevenson WG: Identification of the ventricular tachycardia isthmus after infarction by pace mapping. *Circulation* 2004;110:652-659.
3. 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.
4. Sosa E, Scanavacca M: Epicardial mapping and ablation techniques to control ventricular tachycardia. *J Cardiovasc Electrophysiol* 2005;16:449-452.
5. Thiele H, Sick P, Boudriot E, Diederich KW, Hambrecht R, Niebauer J, Schuler G: Randomized comparison of intra-aortic balloon support with a percutaneous left ventricular assist device in patients with revascularized acute myocardial infarction complicated by cardiogenic shock. *Eur Heart J* 2005;26:1276-1283.
6. Thiele H, Lauer B, Hambrecht R, Boudriot E, Cohen HA, Schuler G: Reversal of cardiogenic shock by percutaneous left atrial-to-femoral arterial bypass assistance. *Circulation* 2001;104:2917-2922.