A 69-year-old man with a history of ischemic cardiomyopathy who had multiple implantable cardioverter-defibrillator shocks for sustained monomorphic ventricular tachycardia (VT) despite antiarrhythmic therapy underwent VT ablation. Because of the patient’s severe peripheral vascular disease, left ventricular (LV) access was obtained via transseptal access using a Brockenbrough needle (BRK, St Jude Medical, Minnetonka, MN) over a medium curve, 8.5F Agilis sheath (St Jude Medical) under fluoroscopy and intracardiac ultrasound (ICE) guidance (AcuNav, Siemens, Mountainview, CA). Systemic heparin was given with the activated clotting time ranging from 250 to 340 seconds. A 7.5F bidirectional, deflectable, 3.5-mm-tip, external-irrigated ablation catheter (EZ STEER ThermoCool, Biosense Webster, Diamond Bar, CA) was advanced into the LV, and an electroanatomic map was created using the CARTO 3 mapping system (Biosense Webster). A large endocardial inferobasal scar was visible on the voltage map. Programmed electric stimulation and burst pacing were performed from several right ventricular and LV sites with 3 drive train cycle lengths extending the lesion set into the lateral aspect of the scar (6 RF applications; mean RF duration of 106 seconds per lesion; total RF duration of 611 seconds). After 10 minutes of focal RFA for the clinical VT, further RF was delivered, extending the lesion set into the lateral aspect of the scar (6 RF applications; mean RF duration of 106 seconds per lesion; total RF duration of 640 seconds). No steam pops or sudden impedance drops were observed during ablation (Figure, B). Fluoroscopy did not reveal a significant change of the heart border. After the final RF lesions, programmed electric stimulation was performed, showing that VT was no longer inducible. Twelve minutes after the final RF delivery, a sudden drop in systemic blood pressure from 140/90 to 80/60 mm Hg was noted. ICE images revealed a 1.5-cm pericardial effusion (Supplemental Video 1). Intravenous heparin was discontinued, and fluids and protamine were given intravenously. An emergent pericardiocentesis was performed with drainage of 650 mL of nonclotting blood. Initially, systemic blood pressure quickly recovered, but during the next 2 hours, continuous bleeding to the epicardial space with hemodynamic compromise and additional removal of 550 mL was noted prompting further fluid resuscitation, blood products, and positive inotropes. Further ICE images demonstrated a large epicardial thrombus (Supplemental Video 2). The patient was taken to the operating room for an urgent sternotomy. A large hematoma was evacuated from the pericardium. A round area of blanched myocardium (2 cm in diameter) was localized to the lateral aspect of the inferior wall in the midsection between the mitral valve and the LV apex. This area of epicardial necrosis and surrounding edema correlated with the recorded endocardial ablation site for the clinical VT on the 3D mapping system. The perforation site with active bleeding was localized at the medial aspect of the necrotic area (Figure, A, blue arrow). The perforation was closed with monofilament sutures. Postoperative pericardial drainage subsided. After 24 hours, the patient was successfully extubated and mediastinal chest tubes were removed on postoperative day 3.
LV perforation has been reported during endocardial RFA for VT. To our knowledge, this is the first time that an LV perforation site is mostly unclear. Calkins et al described as a potential tool to prevent the occurrence of steam pops, which can predispose to cardiac perforation, particularly when higher-energy settings (power greater than 30 to 60 seconds in a specific location). However, no dramatic impedance changes were seen during the ablation in this case. The maximal power (50 W) was on the upper end of frequently used power settings, and we did not test for noncapture after delivering the initial RFA lesions, which could have potentially limited the number of lesions given. However, energy delivery and monitoring parameters were within those reported in large clinical trials, but still resulted in a LV transmural lesion and perforation. To our knowledge, this is the first time that an LV perforation has been reported during endocardial RFA for VT. This raises the question if a LV perforation may account for a significant percentage of the cardiac tamponade complications observed in clinical trials. As the number of patients with structural heart disease undergoing VT ablations increases, electrophysiologists must become aware of this potential risk. Future evaluation of the optimal ablation parameters should be considered.

**Disclosures**

None.

**References**


**Key Words:** ablation | cardiac tamponade | mapping | necrosis | ventricular tachycardia
Left Ventricular Perforation During Cooled-Tip Radiofrequency Ablation for Ischemic Ventricular Tachycardia
Alejandro Jimenez, Richard Kuk, Ghada Ahmad, Jing Tian, Jose Garcia, Anastasios Saliaris, Stephen Shorofksy and Timm Dickfeld

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SUPPLEMENTAL MATERIAL

Video 1. Intracardiac ultrasound showing large pericardial fluid posterior to the right ventricle.

Video 2. Intracardiac ultrasound showing large pericardial hematoma after evacuation of 650 ml of pericardial blood.