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 seen on the voltage map. Programmed electric stimulation and burst pacing were performed from several basal scar was seen 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 and up to triple extrastimuli with a minimal coupling interval of 200 ms. During programmed electric stimulation, VT was induced with a right bundle-branch block morphology, right superior axis, and a tachycardia cycle length of 537 ms, matching the leadless electrogram morphology of the clinical VT. Pace mapping at the lateral edge of the inferobasal scar showed a 12/12 pm correlation with the clinical VT (Figure, D). Intermittent ICE imaging during the voltage/p ace mapping showed no pericardial effusion. Radiofrequency ablation (RFA) was performed using a powered controlled setting (Stockert 70 RF Generator, Biosense Webster, Diamond Bar, CA) and a COOLFLOW irrigation system with a saline infusion rate of 30 mL/min, maximum power of 50 W (starting with 30 W and a gradual increase), temperature limit not to exceed 50°C, and an impedance delta no greater than 10 Ω (7 RF applications; mean RF duration of 87 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 intraoperatively. 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 described as a potential tool to prevent the occurrence of cardiac tamponade with endocardial RFA lesion (yellow circle) seen on the inferior LV wall of the clinical mapping system. A 12/12 pace map matching the clinical VT was achieved at this ablation site (blue location point within yellow circle), as seen in D.

Discussion
Catheter ablation is a well-established option to decrease VT recurrence and implantable cardioverter-defibrillator shocks in patients with ischemic/nonischemic heart disease. Acute procedural complications include a mortality rate of up to 3% and a risk of major complications up to 10%. Cardiac tamponade has been reported in up to 2.7% of cases, but the perforation sites are mostly unclear. Calkins et al reported a left atrial appendage perforation with transseptal puncture, whereas Stevenson et al described a case of tamponade after the patient abruptly sat up without specified perforation site. To the best of our knowledge, perforations of the LV have never been previously described during endocardial VT ablation and are thought to be very unlikely because of the LV myocardial thickness. Indeed, transmural lesions in the LV are not typically achieved as evidenced by the difficulty to eliminate epicardial circuits with endocardial RF alone. Additionally, the properties of fibrous tissue appear to decrease the risk of perforation further when ablating in an area of myocardial scar. Because of the greater disparity between tissue and electrode tip temperatures during RFA with irrigated catheter systems, impedance monitoring has been described as a potential tool to prevent the occurrence of steam pops, which can predispose to cardiac perforation particularly when higher-energy settings (power >40 W) are applied for 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

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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.