Cryoballoon ablation for refractory ventricular tachycardia

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Introduction

Ventricular arrhythmias (VAs) may arise from the endocardium, including mobile endocavitary structures (papillary muscles and the right ventricular moderator band), midmyocardium, or epicardium. Radiofrequency (RF) ablation of VAs is limited by lesion depth, catheter contact, and stability, particularly with mobile endocavitary structures. Focal cryoablation of VAs has been performed via an endocardial approach[1] or used intraoperatively at the time of cardiac surgery.[2] There are currently 2 reports of cryoballoon ablation of VA arising from endocavitary structures in the right ventricle (RV).[3,4] We present a series of 7 cases (Table 1) where cryoballoon ablation (Medtronic Arctic Front™; Medtronic, Minneapolis, MN) was used as an ablation strategy for therapy-refractory VAs arising from the right and/or left ventricle (LV).

KEY TEACHING POINTS

- Ventricular arrhythmias may arise from mobile endocavitary structures (papillary muscles and the right ventricular moderator band), midmyocardium, or epicardium. Radiofrequency ablation of ventricular arrhythmias is limited by lesion depth, catheter contact, and stability—particularly with arrhythmias originating from endocavitary structures.
- Cryoballoon ablation provides adhesive stability on endocavitary structures and possibly allows for the formation of deeper, more homogeneous lesions.
- Newer balloon design technologies could improve contact with the myocardial surface and incorporate high-density mapping on the balloon surface, which may improve effectiveness in ablating ventricular arrhythmias from endocavitary structures.

Case report

Case 1

A 60-year-old male patient with ischemic cardiomyopathy (ejection fraction [EF] 30%–35%), apical infarct, presented with ventricular tachycardia (VT) and multiple implantable cardioverter-defibrillator (ICD) shocks. Two previous ablations failed. An epicardial substrate was suspected. He was referred for reablation. An endocardial voltage map demonstrated an apical scar (Figure 1). An apical VT was induced but extensive endocardial RF ablation failed to terminate the VT. Epicardial mapping was not an option owing to adhesions. The cryoballoon was used to create transmural apical lesions. The cryoballoon was placed in an apical LV location (Figure 1) and firm forward pressure was held to promote tissue contact. Four overlapping freezes were performed covering the apical scar. VT was not inducible at the conclusion of the procedure and has not recurred during a follow-up of 18 months.

Case 2

A 55-year-old male patient with a structurally normal heart was referred for fatigue and frequent premature ventricular contractions (PVCs) with a 34% PVC burden. Activation mapping of the PVCs showed a diffuse area of early activation in the RV moderator band. Despite extensive RF ablation, PVC suppression did not occur. Cryoballoon ablation was performed in the apical septal RV, positioning the balloon under intracardiac echocardiographic guidance. Cryoballoon ablation was chosen over focal cryoablation given the diffuse area of early activation, with the hypothesis that the cryoballoon could provide a larger surface area of contact, creating a deeper lesion. This resulted in a change in PVC morphology, indicating a more lateral exit site. The residual

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Table 1 Clinical characteristics and outcomes of cryoballoon ablation in 7 patients

Patient # Age Sex	Structural disease	Arrhythmia substrate	EF	Prior failed therapies	Number of cryoballoon freezes	Total freeze duration (s)	Minimum temperature achieved (Celsius)	Result	Success?
Patient 1 60 Male	ICMP	LV apical ischemic scar	30%–35%	Endocardial RF ablation ×2, amiodarone, metoprolol	4	1410	-38	No VA recurrence	Yes
Patient 2 55 Male	None	RV papillary muscle, moderator band, fascicular system	50%–55%	Carvedilol	5	1172	-33	PVC recurrence	No
Patient 3 49 Male	NICMP	LV basal lateral epicardial scar	35%–40%	Endocardial RF ablation ×2, amiodarone, metoprolol	1	219	-27	No VA recurrence	Yes
Patient 4 57 Female	Valvular NICMP	LV papillary muscle PVCs	50%–55%	Endocardial RF ablation, could not tolerate medical therapy	2	418	-32	Improved PVC burden with reduced palpitations	Yes
Patient 5 33 Female	None	LV papillary muscle PVCs	60%–65%	Endocardial RF ablation ×8, flecainide, propafenone, metoprolol, amiodarone, sotalol, and propranolol	5	812	-31	PVC recurrence	No
Patient 6 46 Male	None	Idiopathic VF due to RV moderator band PVCs	60%–65%	Endocardial RF ablation, amiodarone, mexiletine, quinidine	5	1570	-37	No VA recurrence	Yes
Patient 7 29 Female	NICMP	LV fascicular PVCs	20%–25%	Endocardial RF ablation, amiodarone, mexiletine	4	1260	-31	PVC recurrence	Yes

EF = ejection fraction; ICMP = ischemic cardiomyopathy; LV = left ventricle; NICMP = nonischemic cardiomyopathy; PVC = premature ventricular complex; RF = radiofrequency; RV = right ventricle; VA = ventricular arrhythmia; VF = ventricular fibrillation.

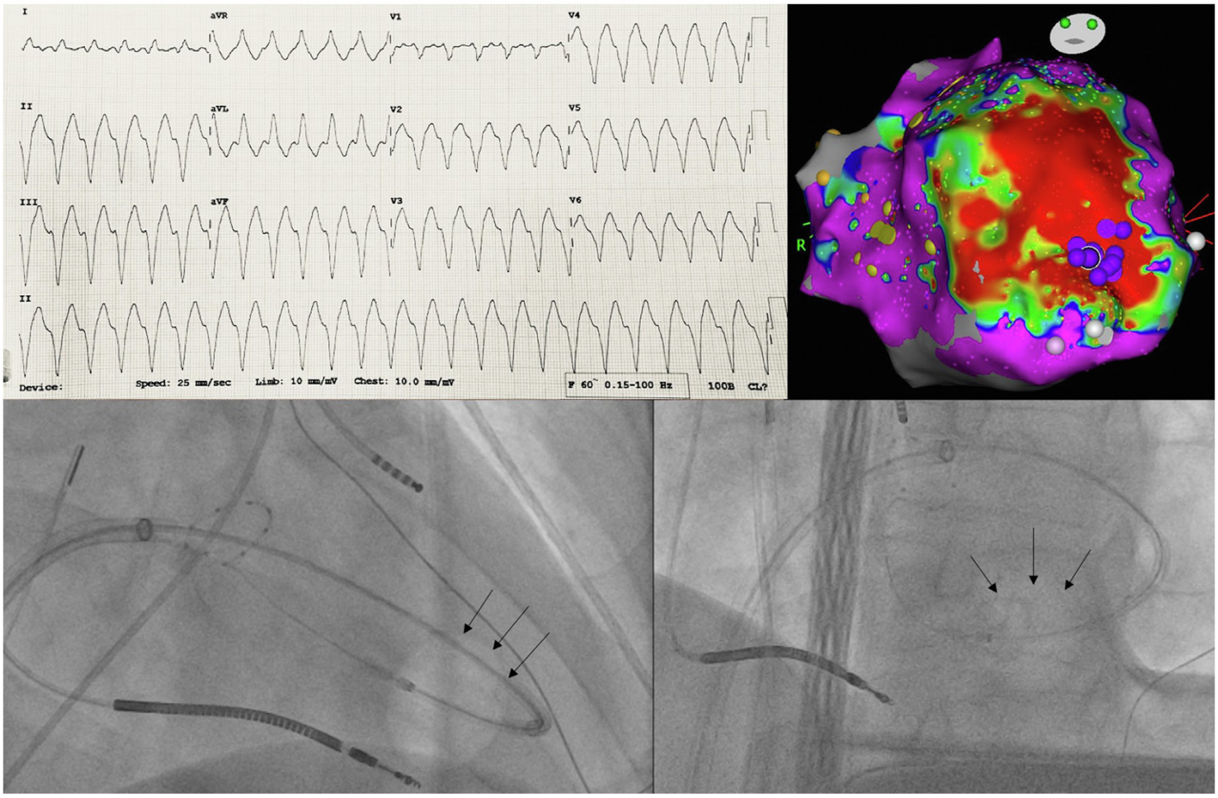

Figure 1 Top left: Twelve-lead electrocardiogram (case 1) showing ventricular tachycardia originating from the apical left ventricle (LV). Top right: Electroanatomic voltage map showing dense apical LV scar and purple ablation tags at the site of failed endocardial radiofrequency ablation. Bottom: Fluoroscopic views showing cryoballoon placement in the LV apex (bottom left, right anterior oblique; bottom right, left anterior oblique).

PVCs were ablated with RF energy, resulting in a low burden of multifocal PVCs at the conclusion of the procedure. Repeat event monitoring showed a 32% PVC burden.

Case 3

A 49-year-old male patient with nonischemic cardiomyopathy (EF 35%–40%) and VT with multiple ICD shocks was referred for ablation. Endocardial voltage mapping showed only a small territory of reduced voltage in the basal inferior and lateral LV walls. Activation mapping and electrocardiographic morphology with a pseudo-delta wave suggested an epicardial focus from this location. PVCs of similar morphology also spontaneously occurred. Extensive endocardial ablation did not terminate the VT or suppress PVCs and attempts at epicardial access were unsuccessful owing to adhesions. The cryoballoon was placed at the basal lateral LV guided by fluoroscopy in right anterior oblique and left anterior oblique views, with delivery of a single freeze. The PVCs were suppressed after a single application and sustained VT did not recur during a follow-up of 17 months.

Case 4

A 57-year-old woman with a history of valvular heart disease (EF 50%–55%, bioprosthetic mitral valve replacement)

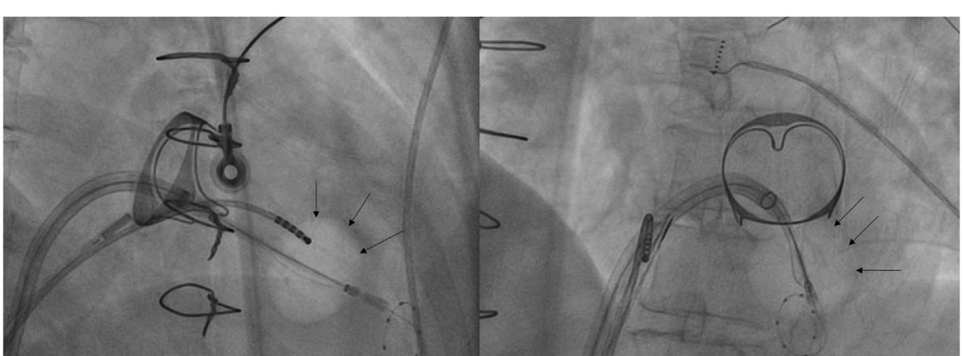

Figure 2 Cryoballoon placement through the bioprosthetic mitral valve under fluoroscopic guidance showing the right anterior oblique view on the left and left anterior oblique view on the right.

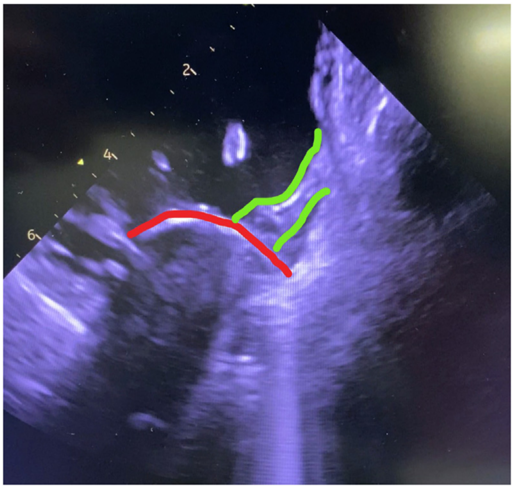

Figure 3 Intracardiac echocardiography demonstrating contact between the cryoballoon (*red*) and moderator band (*green*).

presented with LV anterolateral papillary muscle PVCs (burden 16%) after failed ablation. Activation mapping showed earliest activation in the anterolateral papillary muscle and RF energy delivery in this territory failed to suppress the PVCs. The cryoballoon was advanced into the LV over a guidewire and positioned at the location of the anterolateral papillary muscle (Figure 2). Two cryoballoon freezes were delivered at this location with successful PVC suppression. At follow-up, the patient reported markedly reduced palpitations and the follow-up PVC burden was 2%.

Case 5

A 33-year-old female patient with a structurally normal heart presented with highly symptomatic PVCs refractory to 8 prior RF ablation attempts (burden 8.5%). Endocardial activation mapping showed the area of earliest activation to be the anterolateral papillary muscle. RF energy delivery at this site did not suppress the PVCs. The cryoballoon was positioned at the failed ablation site on the anterolateral papillary muscle, and 5 freezes were delivered, with marked reduction in PVC frequency. A repeat event monitor was obtained after the procedure, which showed a reduced PVC burden of 4.4%.

Case 6

A 46-year-old male patient with idiopathic VF (EF 60%–65%) was transferred for VT storm after failed RF ablation of RV moderator band PVCs. He had received >40 ICD shocks in the prior 3 days. PVC morphology was consistent with moderator band origin. Under intracardiac echocardiographic guidance (Figure 3), the cryoballoon was positioned on the RV moderator band. Five freezes were delivered, which eliminated the triggering PVC. During a follow-up of 6 months, VT has not recurred.

Case 7

A 29-year-old female patient with a history of nonischemic cardiomyopathy (EF 20%–25%) and prior catheter ablation presented with drug-refractory VAs. Repeat ablation was performed with mechanical support. A voltage map showed only a small territory of midseptal scar. Fascicular PVCs originating in this area incessantly triggered VF. RF energy delivery did not suppress the PVCs. The cryoballoon was placed against the mid septum in this area and 4 freezes were delivered. The patient remained free of sustained VAs at a follow-up of 4 weeks.

Discussion

We are reporting a series of 7 patients who underwent cryoballoon ablation of therapy-refractory VAs. Considering that conventional RF ablation had failed and that we had no other options, cryoballoon ablation was used as a bail-out strategy, off label without approval from the Institutional Review Board or the device manufacturer, with the intention to achieve an acceptable clinical result for the patient. Cryoballoon ablation was used in a spectrum of arrhythmia substrates: endocavitary structures in 4 patients (2 RV moderator band, 2 LV papillary muscle), left fascicular in 1 patient, and deep LV myocardial in 2 patients. Cryoballoon ablation was successful in 5 of 7 patients. In all cases, the cryoballoon was chosen as the ablation modality, given prior RF ablation failure and either an intracavitary VA focus for which the cryoballoon could provide enhanced stability or a deep intramural focus with the hypothesis that the cryoballoon could potentially achieve a deeper lesion. The cryoballoon was positioned using guidance from fluoroscopy, intracardiac echocardiography imaging, and visualization of the circular mapping catheter on mapping software. As the optimal freeze protocol in the ventricle is not known, we empirically placed 1–2 freezes in locations at which we achieved an acute response, and up to 5 freezes at locations where an acute response was not observed. Our report expands the limited observational data published about the use of cryoballoon technology for VAs, and includes patients with LV arrhythmia substrates. Advantages of cryoballoon ablation include adhesive stability on RV and LV endocavitary structures and the ability to deliver a deeper, more homogeneous myocardial lesion. Major complications did not occur, but further study is required to confirm the safety of use of the cryoballoon in the ventricle. Technical challenges during cryoballoon positioning limit its application as an ablation tool for VA. Care must be taken not to entangle the circular mapping catheter in the subvalvular apparatus. The use of a guidewire may help to overcome this limitation. The current cryoballoon technology cannot be viewed as a go-to tool for ablation of VAs. Newer balloon technologies modified to improve contact with the myocardial surface that may incorporate high-density mapping on the balloon surface may improve applicability of this technology during ablation of VAs and will require further study.

Conclusion

We report 7 patients who underwent cryoballoon ablation of VAs refractory to conventional endocardial RF ablation. Cryoballoon ablation was successful in 5 of 7 patients. Advantages of cryoballoon ablation may include adhesive stability on endocavitary structures and the creation of deep homogeneous myocardial lesions. Newer balloon technologies designed to improve myocardial contact that may incorporate high-density mapping on the balloon surface may expand utility of this technology for the ablation of VAs.

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