Ablation of atrial arrhythmias in patients with cardiogenic shock on mechanical circulatory support

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Introduction

Use of mechanical circulatory support (MCS) in patients with cardiogenic shock (CS) and for circulatory protection during ventricular tachycardia (VT) ablation is increasing. However, evidence for supraventricular tachycardia (SVT) ablation is limited to a single case report.[1] SVT ablation is performed in symptomatic but stable patients that experience recurrence despite medical therapy. However, there are currently no recommendations for catheter ablation when noninvasive therapies have been exhausted, and definitive SVT treatment is required to regain hemodynamic stability. We present 5 cases of SVT ablation in critically ill patients on MCS.

Case report

Patients ranged from 28 to 77 years of age. They included a 29-year-old man with atrial tachycardia (AT) with intra-aortic balloon pump (IABP), a 55-year-old woman with atrial fibrillation (AF) with rapid ventricular rate on extracorporeal membrane oxygenation (ECMO), a 31-year-old man supported with left ventricular assist device (LVAD)/CentriMag right ventricular assist device (RVAD) with atrioventricular nodal reentrant tachycardia (AVNRT), and a 46-year-old man with AVNRT supported by an Impella. The fifth patient was a 77-year-old man with a destination therapy (DT) Heart-Mate 3 LVAD who had cavotricuspid isthmus (CTI)-dependent IART (Supplemental Table 1).

In 4 cases, patients were transferred with decompensated systolic heart failure for consideration of advanced therapies including durable MCS and/or orthotopic heart transplant (OHT). The fifth patient had a DT LVAD and he was admitted with a small bowel obstruction (SBO) that triggered IART. Full clinical details for each patient are provided in Supplemental Table 1.

The 29-year-old man was admitted with nonischemic cardiomyopathy (NICM) and incessant SVT. He had a left ventricular EF of 10% and moderate right ventricle (RV) dysfunction. During SVT, he sustained heart rates in excess of 200 beats per minute (bpm) despite medications. SVT was refractory to electrical and chemical (amiodarone) cardioversion. He remained persistently hypotensive and attempts to titrate vasoactive agents (dobutamine and nitroprusside) resulted in a greater burden of SVT and hypotension. An IABP was placed, but hypotension during SVT prevented reliable diastolic augmentation. The patient was taken for ablation and AT was localized to the right atrial appendage (RAA) and targeted for ablation (Figure 1). He was cannulated for ECMO and transesophageal echocardiography (TEE) was performed during the procedure in the event that additional hemodynamic support was required. During the first procedure, general anesthesia likely suppressed the high adrenergic tone that resulted from CS. Although AT was eventually induced, activation mapping was limited, since the AT could not be sustained. The patient remained normotensive during mapping (Supplemental Table 2). Following the procedure, spontaneous AT salvos arising from the RAA were noted, but TEE demonstrated adequate IABP augmentation (Figure 1). The patient's AT burden decreased and his clinical status improved and included an invasive hemodynamic profile with lower filling pressures and higher cardiac output, improved ejection fraction (EF), extubation, inotrope wean, and removal of the IABP. However, he continued to have symptomatic AT salvos and empiric repeat ablation was performed at the base of the RAA toward the tricuspid annulus (Figure 2). He has been arrhythmia free at 1-year follow-up, his EF has normalized, and all antiarrhythmics have been discontinued.

The 55-year-old woman had an NICM, EF 20%, and rapid AF. She suffered a near cardiac arrest before being cannulated for ECMO and subsequently transitioned to LVAD/CentriMag RVAD. When attempts were made to wean her RVAD support, rapidly conducting AF, refractory to amiodarone, was thought to be responsible for acutely worsened

KEY TEACHING POINTS

- It is important to appreciate that supraventricular tachycardias (SVTs) can precipitate cardiogenic shock and right heart failure, and thus preclude the ability to wean from vasoactive and mechanical support.

- It should be recognized that SVTs in the setting of cardiogenic shock can be resistant to chemical and electrical cardioversion.

- Catheter ablation for SVT can be performed safely with adjunctive mechanical support and should be considered early in a patient's hospital course when cardioversion is not durable.

right-sided filling pressures and cardiac output. Direct current cardioversion (DCCV) was successfully performed, but the rhythm almost immediately reverted to AF. The patient underwent atrioventricular node (AVN) ablation, her single-chamber implantable cardioverter-defibrillator was reprogrammed to VVI 100 bpm, and her RVAD was explanted the following day. Additionally, her invasive hemodynamic profile and inotrope/vasopressor requirement all improved. She underwent OHT 33 days later.

The 31-year-old man with NICM, EF <20%, and AVNRT experienced hypotension despite inotropic therapy, so an IABP was inserted. However, hypotension during SVT paroxysms compelled an upgrade in hemodynamic support. Furthermore, SVT persistence precluded decannulation. While his ECMO outflow cannula was clamped, self-terminating VT was induced, which terminated AVNRT. However, AVNRT quickly resumed. Radiofrequency ablation (RFA) was attempted for the AVNRT on ECMO, but a steam pop occurred so, alternatively, cryoablation of the slow pathway was performed successfully (Figure 3). The next day his milrinone was weaned, antiarrhythmics were stopped, and invasive monitoring was discontinued. Two days after ablation, he was decannulated and transitioned to a temporary LVAD. Following extubation and recovery of renal function, he underwent LVAD HeartMate 2 implantation.

The 46-year-old man with NICM, EF 15%, and AVNRT refractory to adenosine was transferred to our institution with
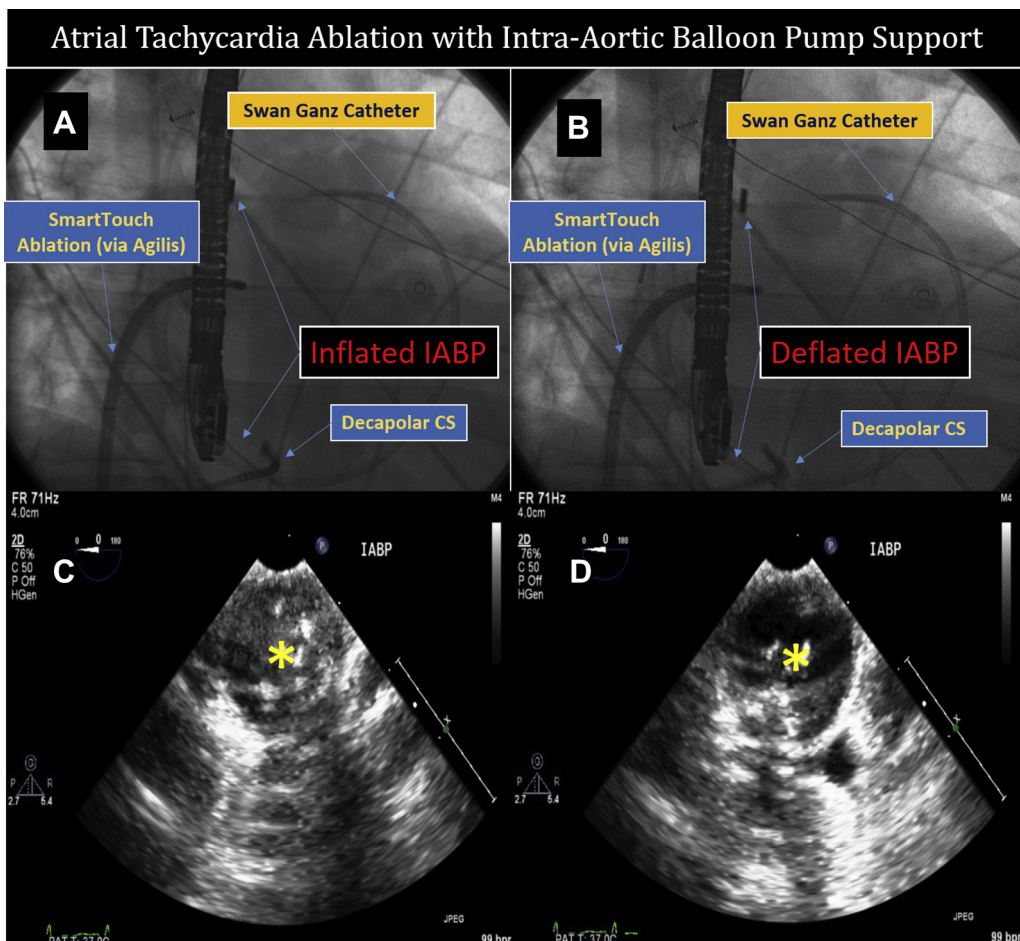

Figure 1 Fluoroscopy of intra-aortic balloon pump (IABP) support during radiofrequency ablation of an atrial tachycardia with the balloon **A:** inflated and **B:** deflated. Transesophageal echocardiography of the aorta in short-axis view that demonstrates the balloon pump (*asterisk*) **C:** inflated during diastole and **D:** deflated during systole. CS = cardiogenic shock.
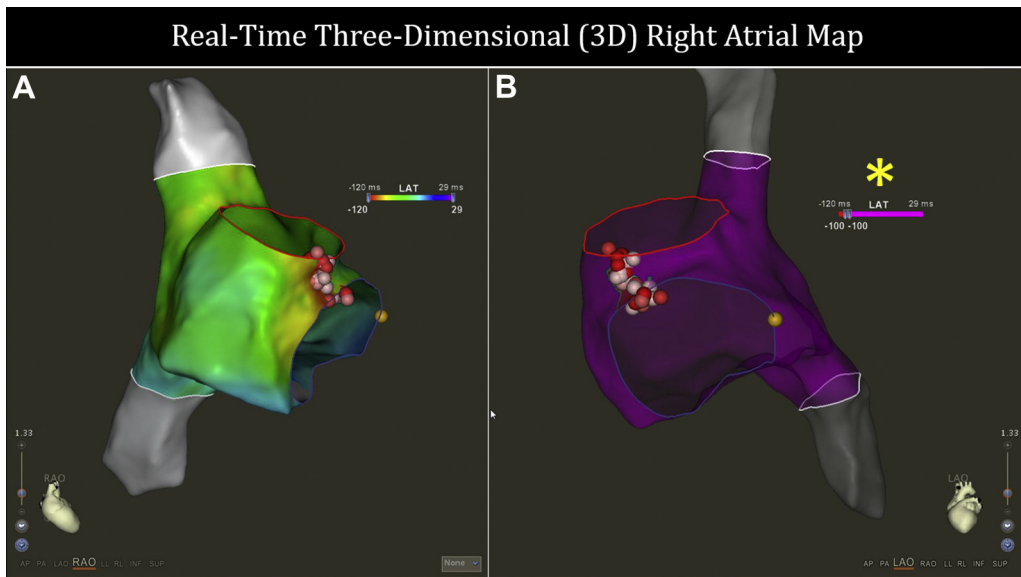
Figure 2   Activation map and ablation lesions in **A:** right anterior oblique (RAO) and **B:** left anterior oblique (LAO) with the activation window (*asterisk*) narrowed to focus on area of earliest activation.

an Impella 2.5. Despite MCS, he was hypotensive with systolic blood pressure (SBP) < 80 mm Hg during tachycardia. He converted successfully with adenosine but AVNRT was incessant and precipitated hypotension. He underwent successful slow pathway modification, which resulted in immediate blood pressure stabilization, decreased filling pressures, higher cardiac output, EF improvement, extubation, inotrope and vasopressor wean, and removal of the Impella less than 24 hours later.

The 77-year-old man with ischemic cardiomyopathy status post DT HeartMate 2 LVAD and IART who had an SBO was not hypotensive. However, there was concern that rapid rates and spontaneous long episodes of atrioventricular block during IART could impair the ability to maintain hemodynamic stability if his SBO progressed to a surgical emergency. He underwent a successful CTI ablation (AV conduction and HV interval were normal in sinus rhythm). Notably, following ablation, his RV function
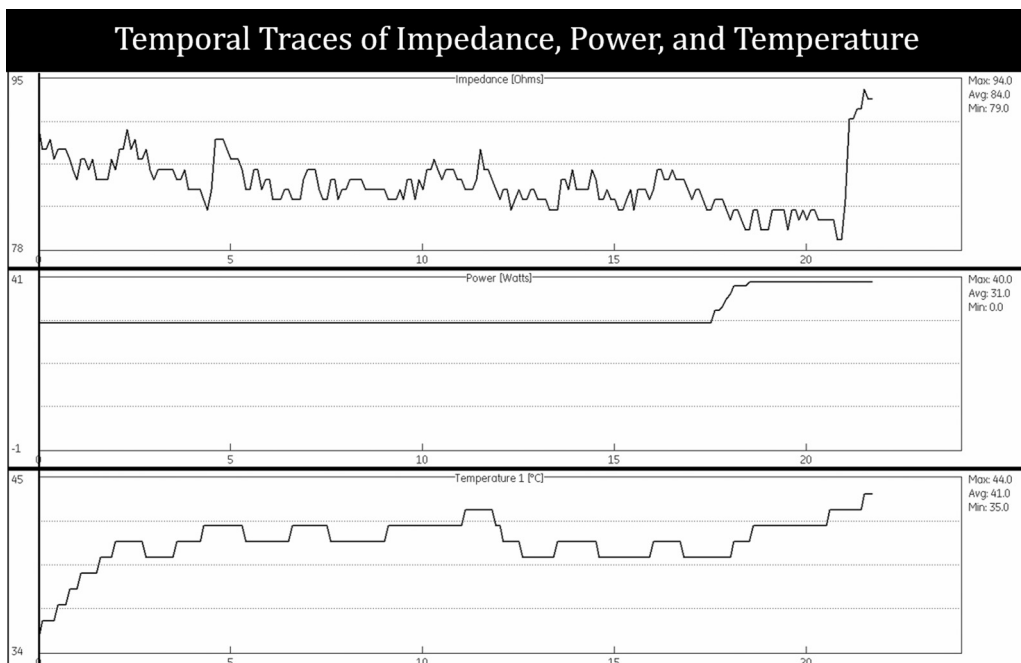

Figure 3   Sharp impedance rise at the end of ablation and increasing temperature signifying a steam pop.

improved on transthoracic echocardiography and his SBO resolved with conservative measures.

Discussion

Guidance for management of hemodynamically unstable patients with SVT that is refractory to medical therapy and cardioversion is absent from the ACC/AHA guidelines.[2] When considering management for refractory SVT in CS patients who require MCS (particularly if RV failure or pulmonary hypertension is present), ablation should be considered. Our case series describes 5 patients with CS and various SVTs. In each case, SVT contributed to hemodynamic embarrassment (hypotension refractory to vasoactive agents and/or progressive multiorgan failure). With the exception of the 77-year-old man with an SBO that triggered IART, the patients were persistently hypotensive (SBP < 90 mm Hg) and 2 threatened cardiac arrest (SBP < 80 mm Hg) during tachyarrhythmia. Additionally, all 4 suffered multiorgan failure. These 4 patients were all managed with exhaustive medical therapy before ablation. Once medical therapy was exhausted, patients were referred for catheter ablation.

The hemodynamic impact from SVT and intra-atrial tachyarrhythmias is well established. Long-standing persistent SVT is a well-known cause of NICM and heart failure.[3] In acute CS, particularly in acute RV failure or severe RV dysfunction, atrial tachyarrhythmias hasten physiologic instability.[4] Pulmonary hypertension also heightens hemodynamic vulnerability when SVTs arise. In a retrospective review, Tongers and colleagues[5] found that in patients with pulmonary hypertension, SVT was almost invariably associated with marked clinical deterioration and RV failure (84% of SVT episodes).

Treatment of atrial tachyarrhythmias has been shown to stabilize hemodynamics. Ventricular function and cardiac output has been shown to improve with restoration of sinus rhythm from AF. A similar improvement has been found for patients with AT and atrial flutter (AFL).[6,7]

While more conservative treatments are attractive in patients with profound CS, they come with a broad array of complications. Structural heart disease disqualifies the use of many antiarrhythmic drugs (AADs) and multiorgan failure increases the risk for side effects to an unacceptable degree. In our series, 3 patients were treated with amiodarone without success, while the others had contraindications to AADs.

Furthermore, SVT ablations are highly effective. There is concern that efficacy might be compromised in CS patients. For example, sedation likely suppressed the adrenergic tone sustaining the AT in the 29-year-old man. As a result, limited activation mapping during the first procedure and sinus voltage mapping during the patient's second procedure were the primary guides for ablation. In the other 4 cases, standard maneuvers were possible without hypotension. Regarding ablation technique, low flow from ECMO likely limited the ability to cool the RF catheter in the patient with AVNRT and a steam pop resulted. However, the slow pathway was successfully modified with cryoablation. Although alternative approaches were utilized in 2 patients, ablation was successful in all 5 (Supplemental Tables 2 and 3).

AVRNT and atrioventricular reentrant tachycardia are successfully ablated greater than 95% of the time, whereas AT and AFL termination have slightly lower rates of durable success, 80%–100%.[8,9] There is a similar rate of success for AVN ablations. A meta-analysis of 21 studies involving 1181 patients has found AVN ablation to be effective, with a statistically significant improvement in left ventricular EF.[10]

In a heterogeneous population of catastrophically ill patients, the ability to safely and effectively perform the procedure is a concern. All patients required anticoagulation with MCS. None of the patients had bleeding complications and all sheaths were removed at the end of the case. Stability during programmed electrical stimulation was a concern, given each patient's critical condition, but all patients tolerated pacing maneuvers without hypotension. None required DCCV during ablation (Supplemental Tables 2 and 3).

The efficacy of SVT ablation is matched by its safety. A meta-analysis by Spector and colleagues[11] found that among SVT studies, all-cause mortality was 0.1%, and adverse events were reported in 2.9% of patients. However, case #3 challenges the safety of RFA for patients supported by ECMO. The unpredictable intracardiac loading conditions with ECMO may impair RF catheter ablation and lead to a disparity between electrode temperature and tissue temperature. As a result, tissue temperatures may far exceed catheter tip temperatures and steam explosions or "pops" can occur (Figure 3). Some might hesitate to utilize an irrigated catheter in this region; and so alternatively, cryoablation can be performed.

Different benefits and limitations accompany each type of MCS and despite data that describe the efficacy and safety of SVT ablation, evidence for MCS-dependent CS patients is absent (Supplemental Table 4). Therefore, outcomes from using MCS during ischemic and nonischemic VT ablation serve as a benchmark. In the largest retrospective review to date, 194 patients (109 percutaneous and 85 nonpercutaneous LVAD) underwent scar-mediated VT ablation. Following propensity matching, no differences were seen between groups for acute procedural outcomes and the primary end point (recurrent VT, heart transplantation, or death).[12]

In each case, there was no procedurally related complication and all 5 patients gained immediate hemodynamic stability following ablation. Each patient was rapidly weaned from MCS and successfully discharged from our facility. In addition, 4 of the patients had no arrhythmia recurrence. The patient described in case #1 (RAA AT) experienced a significant reduction in his arrhythmia burden and he was discharged arrhythmia free after a second ablation during the same hospitalization. A durable body of evidence needs to accumulate before there is precedent to make recommendations regarding the timing and appropriate use of MCS with SVT mapping and ablation, but the prolonged time of hemodynamic stability during VT pace mapping and ablation

provides justification for SVT ablation in profound CS patients.

Conclusion

This case series illustrates that SVT ablation can be safely performed in profound CS patients reliant on IABP and continuous flow devices. It also provides guidance to clinicians for management of SVT when the arrhythmia provokes instability despite exhaustive medical therapy, including cardioversion and MCS. Moreover, the potentially devastating consequences that SVT can have for patients with RV failure and pulmonary hypertension should compel the consideration of ablation once CS is recognized. When CS with multiorgan failure is present despite MCS, although by no means definitive, this case series provides some precedent for therapeutic options beyond ineffective medications and cardioversion.

Appendix
Supplementary data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2018.11.008.

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