Direct Transthoracic Access to the Left Ventricle for Catheter Ablation of Ventricular Tachycardia

Short title: Hsieh; Direct transthoracic LV access for VT ablation

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Abstract

Background: Percutaneous approaches for radiofrequency ablation of ventricular tachycardia (VT) in the left ventricle are typically trans-arterial retro-aortic, antegrade transmitral via an interatrial septal puncture, or epicardial. However, all three approaches may be contraindicated in certain cases. We describe 2 cases of VT ablation where aortic and mitral valve replacements did not permit utilization of any of these techniques.

Methods and Results: Direct access to the left ventricular cavity was achieved with a percutaneous puncture through the intercostal space overlying the apex in the first case, and through a left minithoracotomy in the second. A sheath was then inserted via the Seldinger technique, allowing catheter access for mapping and ablation of the ventricular tachycardia. After successful ablation, the sheaths were withdrawn and hemostasis achieved. A large left hemothorax occurred from the left ventricular apical puncture in the first case. Direct closure with a purse string suture in the second case achieved hemostasis.

Conclusion: Direct percutaneous LV puncture is a viable option for mapping and ablation of left ventricular VT. A mini-thoracotomy allows better hemostatic control. This technique has a role where other percutaneous approaches are contraindicated.

Key words: Catheter ablation, tachyarrhythmias, electrophysiology
Introduction

Percutaneous electrophysiological interventions in the left ventricle are usually performed through a trans-arterial retro-aortic approach or an antegrade transvenous approach via an interatrial septal puncture and then across the mitral valve. More recently, percutaneous pericardial access techniques have also been utilized. However, there are occasions where all three methods are not feasible. We report our experience with 2 such cases where both aortic and mitral valve prostheses and obliteration of the pericardial space from previous cardiac surgery contra-indicated all 3 approaches.

We used direct transthoracic left ventricular apical punctures for left ventricular access and ablation. This technique was used in the past for diagnostic cardiac catheterization, but was largely abandoned when simpler and safer techniques were developed. (1)

Case 1

This 65-year-old man had both aortic and mitral St Jude tilting disc replacements for rheumatic heart disease. His background cardiac history included a previous non-ST-elevation myocardial infarction, dilated cardiomyopathy (left ventricular diastolic dimension 79mm, ejection fraction 14%), dual chamber AICD implanted for clinical VT, and paroxysmal atrial fibrillation. Other medical history included pulmonary hypertension (74mmHg), obstructive sleep apnea, gastro-esophageal reflux, chronic airways disease and a distant stroke without current sequelae.

The patient presented with incessant VT with up to 30 conscious ICD discharges in a single day causing significant psychological distress. VT was refractory to anti-arrhythmics
including amiodarone and lidocaine, both of which appeared pro-arrhythmic, and to pace termination from his ICD. A trial of mexiletine decreased, but did not prevent recurrences. There were at least 3 clinical VTs. (Figure 1)

The patient agreed to an ablation procedure via direct left ventricular puncture. Under general anesthesia, the apex was palpated. A 7cm Cook needle was attached to pressure monitoring and was advanced through the overlying intercostal space towards the cavity of the LV until the pressure waveform indicated entry. A short J-tip guide wire was introduced and a standard 8Fr side arm vascular access sheath was then advanced into the LV cavity. (Figure 2) Decapolar catheters were inserted into the coronary sinus and the right ventricular apex from femoral venous access sites. Full anticoagulation with heparin was started aiming for an ACT > 300 seconds.
Biventricular voltage maps were created using the CARTO system and a D curve irrigated tip Navistar Thermocool catheter (Johnson and Johnson, Pennsylvania USA). A low voltage area consistent with scar was present in the inferobasal RV but there were generally well-preserved electrograms in the LV. Substrate and channel mapping were therefore not useful. Fractionated double potential electrograms were present in the anterobasal LV adjacent to the prosthetic valves. Sustained VT was readily induced with the first delivered single ventricular extrastimulus. VT had a LBBB, right-axis deviation morphology, cycle length 490ms, and VA dissociation and was well tolerated. Mapping showed that the earliest activation, occurring 60ms prior to the onset of the QRS, was in the anterobasal LV in close proximity to the prosthetic valves. (Figure 3) Radiofrequency application here terminated VT in 4 seconds (maximum power 50W, maximum temperature 48 degrees Celsius). (Figure 4)

Further ablations were applied to the area. After the ablations were complete, a fast VT (CL 310ms) was inducible with 3 extrastimuli, but was not hemodynamically stable and required external cardioversion. Since the troublesome slow clinical VT appeared successfully ablated, and the LV end-diastolic pressure had risen from 30mmHg at the beginning of the procedure to 45mmHg, the procedure was terminated. The LV sheath was removed (total duration in situ 2 hours) with a surgeon on standby but good hemostasis appeared to have been achieved. The patient was observed closely, and a repeat transthoracic echocardiogram soon after the completion of the procedure did not reveal any pericardial fluid. In view of the poor LV function, double mechanical valves and paroxysmal AF, the heparin was not reversed.

Ninety minutes post-procedure, the patient deteriorated and became hypotensive. An urgent left thoracotomy was organised, but further compromise occurred during induction of
anesthesia requiring CPR for 20 minutes. Surgical exploration revealed bleeding from the left ventricular apex to the left pleural cavity with 2.5L of blood present in the left pleural space. The pericardial space was confirmed to be obliterated by adhesions without any significant collection or clot. The LV puncture site was oversewn and a chest drain was inserted.

The patient made a remarkably quick recovery and was extubated the next day with no new hemodynamic or cerebrovascular sequelae. There were only rare short runs of VT in hospital. There were no episodes of sustained slow VT. Anti-tachycardia pacing terminated a single episode of faster VT on late follow up at 4 months.

Case 2

A 66 year old man presented with recurrent VT storms. His past medical history included both aortic and mitral Starr Edwards valves implanted in 1966 for rheumatic heart disease. The left circumflex artery was likely injured during the surgery, which led to chronic congestive cardiac failure related to poor left ventricular systolic function. He also developed chronic atrial fibrillation, atrio-ventricular heart block and ventricular tachycardia.

An implantable cardioverter-defibrillator was implanted, and subsequently upgraded to a biventricular device. The left ventricular lead was implanted in a branch of the middle cardiac vein. Unfortunately, pacing from this site was pro-arrhythmic and he developed recurrent incessant VT with a morphology identical to pacing from the lead site. Multiple attempts to deactivate and reactivate the lead were not successful in suppressing the VT. A new LV pacing lead was implanted in a different LV vein, which led to an improvement in his cardiac failure and reduction in episodes of VT.

Unfortunately, this improvement was short-lived and he redeveloped frequent recurrences of VT and worsening congestive cardiac failure. There were at least 3 morphologies of VT, the
most frequent of which had an RS pattern in V1 with left-axis deviation and a cycle length 400-640ms, suggesting a septal or paraseptal origin. (Figure 5) The patient elected to proceed to an ablation procedure.

A decapolar catheter was placed in the coronary sinus and another in the right ventricular apex from femoral venous approaches. A coronary angiogram was performed to demonstrate the coronary artery anatomy.

A short left sub-mammary incision was made, and a rib retractor used to improve exposure. The pericardial space was obliterated by post-surgical adhesions. Prolene purse strings were placed in the LV apex, and a short 8Fr sheath was inserted between them into the left ventricle via the Seldinger technique. Full anticoagulation with heparin was started aiming for an ACT > 300 seconds.

A contact substrate map in native paced rhythm was performed in both ventricles using the CARTO system with a D-curve CARTO Navistar Thermocool catheter. There were low amplitude and fractionated signals with marked delayed potentials in the infero-apical LV wall and apex. This area was close to the tip of the original LV biventricular pacing lead. (Figure 6)

VT was easily inducible with 2 extrastimuli, and had a cycle length of 430ms. It was poorly tolerated hemodynamically. Its LBBB left-axis deviation morphology was consistent with the first clinical VT morphology. Thereafter, the patient developed spontaneous VT cycle length 560ms with LBBB left-axis deviation morphology that was well tolerated. Earliest activation in VT was in the inferoapical LV at the same site as the areas with delayed potentials. (Figure 7)
Radiofrequency energy was applied to this area and terminated VT (maximum power 50W, maximum temperature 50 degrees Celsius). This site and adjacent areas were ablated. Finally, the catheter was placed on the corresponding epicardial surface through the thoracotomy, but no local signals could be recorded.

After ablation of the clinical VT, a different faster VT (cycle length 310ms) was inducible with 4 extrastimuli. There was no recordable blood pressure with this VT and immediate cardioversion was required. This VT had a RBBB, right-axis deviation morphology and intracardiac electrograms indicated origin in the basal septum. Since the clinically troublesome VT had been ablated, and there was background poor LV function with multiple morphologies of VT, more ablation aiming to ablate all inducible VTs was not attempted.

Subsequent to the completion of the ablation, the catheter was removed, and placed epicardially through the mini-thoracotomy. Access to all regions was difficult due to pericardial adhesions, but it was possible to manipulate the catheter to a portion of the epicardial LV surface. The local electrograms obtained were of poor quality.

The sheath was removed (total duration 1.5hrs in situ), and hemostasis was achieved by tying the pursestrings. An intercostal catheter was placed in the left pleural space via a separate stab incision and removed the next day without significant drainage. Intravenous unfractionated heparin was restarted 4 hours post-procedure.

The patient was discharged well 3 days later. He was free of VT over the next 3 months. Amiodarone was ceased. VT then recurred and amiodarone was restarted with no further recurrences for 2 months.
Discussion

Percutaneous direct LV puncture was used in the 1960-80s for measuring gradients across stenosed aortic valves or valve prostheses. (1, 2) The rate of major complications was 3-8%, including ventricular arrhythmias, pneumothorax, hypotension, vasovagal symptoms, and accidental puncture of the lung, bronchus, right ventricle, coronary arteries, or intercostal vessels. (3, 4) Walters et al described the Massachusetts General Hospital experience with 38 cases where the overall complication rate was 8%, with 1 hemopericardium, 1 hemothorax requiring thoracotomy and decortication, and 1 episode of VF during the procedure. (5).

Importantly, there have been no documented cases of tamponade in patients with previous cardiac surgery. (2, 4-9)

A recent case report included 2 patients requiring direct measurement of their left ventricular pressure with aortic and mitral valve replacements. They reported no significant complications, but stressed the importance of only using a 21-gauge needle for the puncture, and a 4-Fr catheter for ventriculography. (8)

However, in therapeutic cases, larger sheath sizes are often required. In another case series, (7) 8 patients with a varying array of prosthetic valve combinations underwent direct percutaneous left ventricular puncture for a variety of both diagnostic and therapeutic reasons. Their sheath sizes ranged from 4-Fr to 9-Fr. They found this technique especially helpful in approaching postero-septally located, perivalvular mitral prosthetic leaks. They reported only a single adverse event, with a left-sided hemothorax developing several hours after the completion of the procedure in a patient with aortic and mitral prosthetic valves. They attributed the hemorrhage to trauma to an intercostal vessel, and stressed the importance of positioning the needle immediately superior to the rib. In a case series of 22
patients with a previous Fontan repair, direct transthoracic puncture was used to access the pulmonary venous atrium. Two patients (9%) developed a right hemothorax requiring thoracocentesis, probably related to hemorrhage from the right internal mammary artery. (9) Lim et al. (7) described the theoretical risk of direct hemorrhage from the LV apex, as in our first described case, as low. During LV systole, when the LV pressure is high, the myocardium is actively contracting, and therefore should close the hole. During LV diastole, the LV pressure is low, thereby minimizing the risk of bleeding. This remains true as long as left ventricular function is normal. Our patients had severe LV systolic dysfunction and markedly elevated diastolic pressure. By the same logic as above, LV dysfunction would promote bleeding. During systole, an akinetic apical region would not contract and could allow the free passage of blood, and during diastole the higher diastolic LV pressure would promote bleeding.

The other important consideration is the screening method for bleeding after the procedure. Braunwald et al. in a series of 200 patients described only a single case of tamponade in a patient without previous cardiac surgery.(10) We used transthoracic echocardiography as the major imaging investigation in this regard. However, in patients with obliterated pericardial spaces from previous cardiac surgery, it is probable that the bleeding will be directed towards the left pleural space as in our case. We advocate close clinical observation and serial transthoracic echocardiography to image both the pericardial and the left pleural spaces to look for this complication.

There are 2 different techniques described for locating the puncture site. The first is direct palpation of the apex of the heart. The second is use of transthoracic echocardiography to confirm the location of the apex.(6) It has been documented previously that the palpation
method may be inaccurate, and is usually more medial than the true apex, especially in patients with RV enlargement. A needle inserted here has a higher risk of RV trauma.(11) A third option is a mini-thoracotomy to expose the left ventricular apex. This allows for better hemostatic control with purse string closure of the left ventricle. This approach may be useful in patients at high risk of embolic events requiring early anticoagulation.

There has only been 1 reported case of RF ablation performed through direct LV puncture. This was done via a thoracotomy in a 10-month-old child with VT arising from posteroseptal region of the mitral valve annulus that had failed ablations from retrograde and transeptal approaches. (12) Our case series here is the first to describe the procedure in adults with severe LV dysfunction and major comorbidities.

There were some issues in these cases worthy of comment. Firstly, electrophysiology catheters are typically 115cm in length, allowing access from femoral sites. However, their length makes control of the catheters difficult when only the first 5-10cm of the catheter is within the patient. We found it helpful to have the catheter stretch out along the axial length of the patient, and to have the operator stand on the right side of the patient. A short catheter would be helpful but is not commercially available.

The second issue is access to the LV apical endocardial surface. This requires looping back of the catheter tip from the LV cavity and partially withdrawing the access sheath to achieve good contact. Vigilance is required to prevent inadvertent removal of the sheath from the LV cavity. The catheter should be looped back with a u-bend initially, then the access sheath and catheter withdrawn as a single unit until the tip of the ablation catheter contacts the endocardium. Variable flexion and extension of the ablation catheter then allows reasonable
access to the LV apex and adjacent regions using this technique (Figure 6). Provided the ablation catheter remains in the LV cavity, the sheath can usually be re-advanced into the cavity if it falls out.

Another tactic is to leave the tip of the sheath in the mid-cavity but advance the ablation catheter distally until it curves backwards and then keep advancing until it reaches the apex. With this approach, care must be taken to ensure the ablation catheter does not become entrapped in the prosthetic valves (Figure 8). Neither technique adversely affected the quality of the local electrogram.

The use of a mini-thoracotomy did not cause significant movement of the heart relative to the reference catheter and the CARTO registration remained accurate.

Another issue is concurrent epicardial access for mapping and ablation. Mini-thoracotomy allows peri-apical pericardial adhesions to be surgically dissected and permits epicardial mapping and ablation of the exposed region. However, more extensive dissection of adhesions is not favourable because of poor exposure of the more remote ventricular surfaces and the potential risk of lacerating coronary vessels or grafts. Achieving haemostasis in that circumstance is likely to be difficult and might require conversion to a full sternotomy and institution of cardiac bypass.

**Conclusion**

We describe 2 cases where direct percutaneous LV puncture, contact mapping and catheter ablation were used effectively to treat incessant scar-related ventricular tachycardia. Hemorrhage into the left pleural space occurred in the first case and was prevented in the second by using a mini-thoracotomy and purse-string ties to control bleeding from the left
ventricular puncture site. It is important to observe the patient closely after the procedure looking for bleeding into the left pleural space. This technique can have a useful role in catheter ablation when traditional access sites are precluded.

Conflict of Interest Disclosures: None

References:


Figure Legends:

**Figure 1:** ECG’s of VT: (ECG 1) RBBB morphology, right axis deviation, and a cycle length of 440ms. (ECG 2) RBBB morphology, normal axis, and cycle length 460ms. (ECG 3) LBBB pattern, right axis deviation, and cycle length 360ms.

**Figure 2:** RAO (left) and LAO (right) view of the direct percutaneous access sheath (arrow). Contrast injected into sheath.

**Figure 3:** RAO Cranial (left) and LAO Cranial (right) views of activation. Earliest activation (red) in close proximity to prosthetic valves.

**Figure 4:** Recording of RF ablation at anterobasal LV terminating the VT within 4.3s of application of the ablation.

**Figure 5:** 12-lead ECG of clinical VT.

**Figure 6:** RAO (left) and LAO (right) views of ablation catheter through the apical sheath. The tip is in an area of fractionation and delayed potentials and close to the old LV lead (arrow). Note the ablation catheter is withdrawn and flexed to reach the apex.

**Figure 7:** RAO (left) and LAO (right) views of activation in the left ventricle during VT. Earliest activation in the inferoapical left ventricle (red).

**Figure 8:** RAO (left) and LAO (right) views of ablation catheter, which has been advanced to create a large loop within the LV to reach peri-apical regions.
Aortic root

Apex

Earliest activation
Direct Transthoracic Access to the Left Ventricle for Catheter Ablation of Ventricular Tachycardia

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