

Epicardial access facilitated by carbon dioxide insufflation for redo ventricular tachycardia ablation in a patient with arrhythmogenic right ventricular dysplasia and dense adhesions



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Introduction

Ventricular tachycardia (VT) ablation for arrhythmogenic right ventricular dysplasia (ARVD) is a well-established therapy for arrhythmia control in circumstances of failure or intolerance to medical therapy.^{1,2} The fibrofatty infiltration inherent in the pathology of ARVD generally more extensively involves the epicardium compared with the endocardium of the right ventricle (RV), and occasionally the left ventricle. Not surprisingly, VT ablation approaches limited to the endocardium may confer more limited arrhythmia control.^{1,3,4} However, epicardial ablation has often been relegated to expert centers owing to the technical challenge associated with access and relatively high complication rates. The technical difficulty with obtaining epicardial access is heightened in patients wherein dense adhesions may be present, such as patients with a history of prior epicardial ablation, pericarditis, or cardiac surgery. Recently, a multicenter Epi-CO₂ registry published the feasibility of coronary vein exit and carbon dioxide (CO₂) insufflation to facilitate safe epicardial access and allow diagnosis of pericardial adhesions prior to attempted access.⁵ Here we report a case of a redo epicardial VT ablation in a patient with ARVD and dense adhesions, facilitated by CO₂ insufflation.

Case report

A 64-year-old man was referred to our institution for a redo VT ablation. He originally presented with syncope and VT in 1997. Two separate electrophysiology studies disclosed

KEY TEACHING POINTS

- Percutaneous epicardial access for electrophysiological procedures may be challenging in circumstances where adhesions may be present, such as prior epicardial ablation, pericarditis, or cardiac surgery.
- Several approaches for epicardial access may minimize the risk of puncture-related complications, such as favoring an anterior approach, the use of a micropuncture needle or “needle-in-needle,” or the use of a pressure sensor needle, but do not eliminate the chance of inadvertent right ventricular puncture.
- Insufflation of CO₂ into the pericardial space via coronary vein exit is feasible and may improve the safety as well as feasibility of access into the pericardial space in challenging cases.
- Coronary vein exit with a very narrow-profile microcatheter (1.9F) may lower the bleeding risk associated with intentional coronary vein exit for use with CO₂ insufflation–facilitated epicardial access.

KEYWORDS ARVD; Epicardial; Epicardial access; Ventricular tachycardia; VT ablation
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a VT circuit deemed unamenable to ablation and, as a result, a single-chamber implantable cardioverter-defibrillator (ICD) was implanted. Ultimately, he was recognized as having ARVD. Owing to recurrent VT despite use of 240 mg sotalolol twice daily and mexiletine, with resultant medication-related side effects (fatigue, erectile dysfunction), a combined endocardial and epicardial VT ablation was performed at an outside institution in 2004. Anti-inflammatory agents were administered for postprocedural

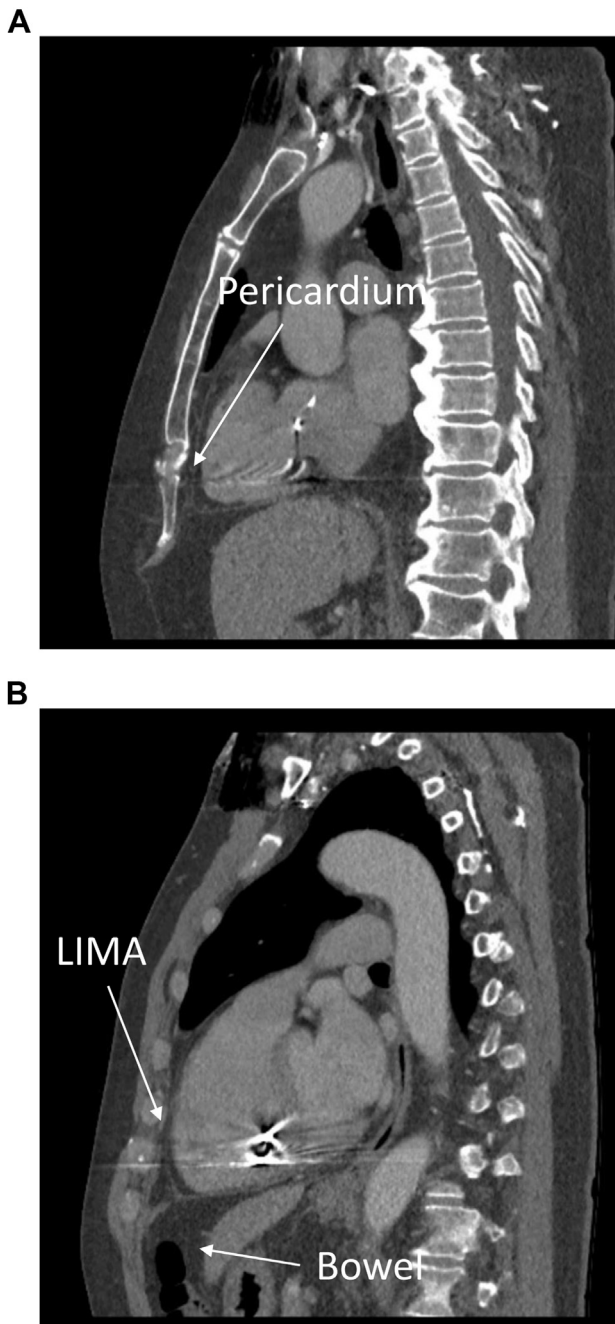


Figure 1 A: Sagittal midline computed tomography image demonstrating window between pericardial space and sternum. B: Slightly more lateral image demonstrating left internal mammary artery (LIMA) and bowel loops in close proximity.

pericarditis at that time. After doing well for many years, he presented with recurrent VT to an emergency room in Florida 8 months prior to his referral to our institution. He was placed on amiodarone but owing to medication intolerance, his cardiologist referred him for redo catheter-based ablation. The patient reported no heart failure–related symptoms and was not on maintenance diuretics.

A preprocedural workup featured an echocardiogram that demonstrated a severely enlarged and dysfunctional RV with preserved left ventricular systolic function. He also

underwent a contrast-enhanced chest computed tomography scan for procedural planning, demonstrating a normal course of the internal mammary arteries and a relatively narrow space between the sternum and pericardial space at the midline (Figure 1A). Slightly more leftward, the space was equally narrow and impeded by prominent bowel loops as well as the presence of the left internal mammary artery, altogether threatening safe access to the pericardial space (Figure 1B). Therefore, a decision was made to insufflate the pericardial space with CO₂ to increase the safety window for epicardial access.

The patient's amiodarone was held for 5 days prior to the planned procedure. He was brought to the electrophysiology laboratory in the fasting state and general anesthesia was induced. ICD therapies were suspended. Bilateral femoral venous and right femoral arterial access were obtained. The procedure was performed with CARTO 3 V7 guidance (Biosense Webster, Irvine, CA). An intracardiac ultrasound catheter (8F, SOUNDSTAR; Biosense Webster) was inserted into the right atrium and RV and utilized to create sound-based 3-dimensional shells of the relevant anatomy and monitor the pericardial space. The coronary sinus (CS) was cannulated with an SL2 sheath (8.5F, 63 cm; Abbott, Abbott Park, IL) over a deflectable decapolar catheter (7F, CS Bi-Directional D-F curve; Biosense Webster). The catheter was exchanged for a MagicTorque guidewire (0.35", 260 cm, Boston Scientific, Marlborough, MA), which was advanced into the distal anterior interventricular vein (AIV). A JR 4.0 diagnostic catheter (100 cm, DxTerity JR 4.0; Medtronic, Minneapolis, MN) was advanced over the wire into the mid AIV and used to perform venography, highlighting the relevant vasculature and multiple suitable small vessels for intentional puncture (Figure 2A). The MagicTorque wire was exchanged for an 0.14" angioplasty wire (PT Graphix, Straight tip, 300cm; Boston Scientific) over which a microcatheter (1.9F, 135 cm, Caravel; Asahi, Seto, Japan) was advanced as distally as possible into a small nonseptal branch. A selective venogram led to myocardial staining (Figure 2B). The wire was exchanged for a high-tip-weight wire (MIRACLEbros, 180 cm, tip load 12.0 gf; Asahi; chosen for high tip weight and penetration force), which was advanced to perforate the branch. Further advancement of the wire was limited and it would not cross far beyond the midline, presumably owing to adhesions over the RV. The microcatheter was advanced cautiously, approximately 3–4 cm past the point of puncture. A subsequent contrast injection through the catheter demonstrated pericardial layering (Figure 2B). Then, 100 cc of CO₂, previously equilibrated to 1 atm, was slowly insufflated, separating the pericardial layers to a sufficient degree to permit puncture in the left lateral fluoroscopic view (Figure 2C). No hemodynamic compromise was noted following the insufflation. Interestingly, the majority of CO₂ was restricted to the inferior border of the pericardial space in a loculated fashion, highlighting the anatomical extent of adhesions from the patient's prior epicardial access (Figure 2C). A "needle-in-needle" approach with a micropuncture needle (21G, MAK-NV; Merit

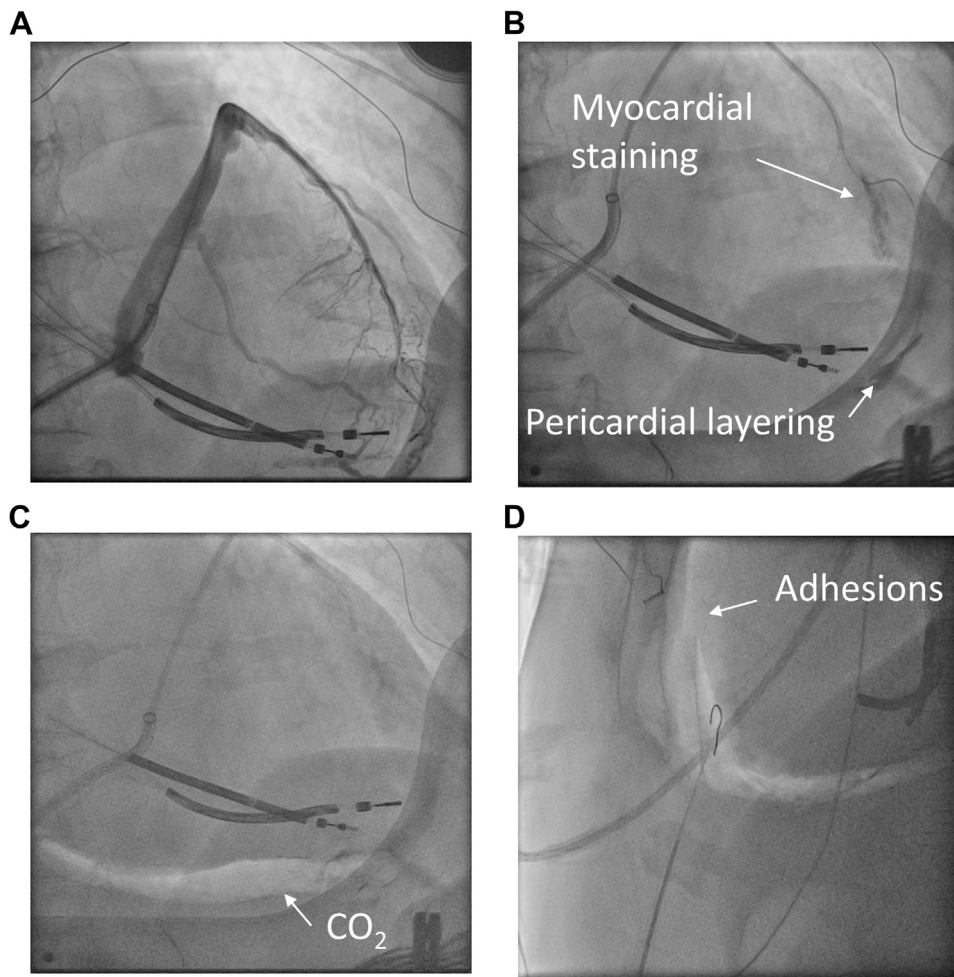


Figure 2 A: Venogram through diagnostic JR4 catheter of the anterior interventricular venous branches. Magic wire extended into the distal vessel. B: Venous exit with contrast layering in the pericardial space. Myocardial staining is noted from prior contrast injection from the distal venous branch prior to puncture. C: CO₂ insufflation demonstrating compartmentalization inferiorly owing to adhesions. D: Pericardial puncture in area without adhesions.

Medical, South Jordan, UT), preloaded with an 0.18" wire to avoid CO₂ escape, was utilized to puncture the space with a low anterior approach where the pericardial layers were successfully separated (Figure 2D). The wire would not advance far superiorly or encircle the pericardial silhouette, but the presence of CO₂ co-segregating with the wire confirmed that the plane of entry was indeed pericardial (Supplemental Movie). A deflectable sheath (Agilis Epi, 40 cm, 8.5F; Abbott) was subsequently advanced into the pericardial space and an open-irrigation force-sensing radiofrequency ablation catheter (ThermoCool ST; Biosense Webster) was advanced and used to gently lyse the adhesions. During the procedure, all initial and any accumulating pericardial fluid was drained through the side port of the sheath. No bleeding was demonstrated.

Three-dimensional electroanatomical voltage maps were then created of the epicardial and endocardial RV with the use of the ablation catheter or decapolar mapping catheter (Figure 3A, 3B; DecaNav; Biosense Webster). A hexapolar catheter was inserted with distal poles in the RV apex. The initial decapolar catheter used to obtain CS access was inserted back in the CS for atrial recording. Programmed

stimulation from the RV apex was performed in an attempt to induce the clinical VT (VT 1, Figure 3C), but only nonsustained runs were seen. With aggressive triple extrastimuli, another VT (VT 2, Figure 3D) was noted with cycle length (CL) of 240 ms, QRS duration 155 ms. It was not hemodynamically tolerated and rapidly cardioverted. Later with pace mapping of the endocardium, a third VT (VT 3, Figure 3E) was induced with CL of 390 ms and QRS duration 221 ms. VT 3 had a slurred upstroke and maximal deflection index of 0.74, suggesting an epicardial origin. It was similar to the clinical VT (VT 1) but with a longer CL and earlier precordial transition. It was also not hemodynamically tolerated and therefore pace-terminated. VT 3 was pace mapped with a long stim-to-QRS delay from the middle of the lateral epicardium in an area of dense scar. Close pace maps to VT 1 were also noted epicardially along the outflow tract and more superior aspect of the lateral base. Prior to ablation, coronary angiography was performed and an acute marginal branch was noted to cross the RV epicardium. No ablation was performed within 1 cm of the vessel. No phrenic capture was noted in any area of interest either. Ablation was performed over the aforementioned regions of pace match and all discernable

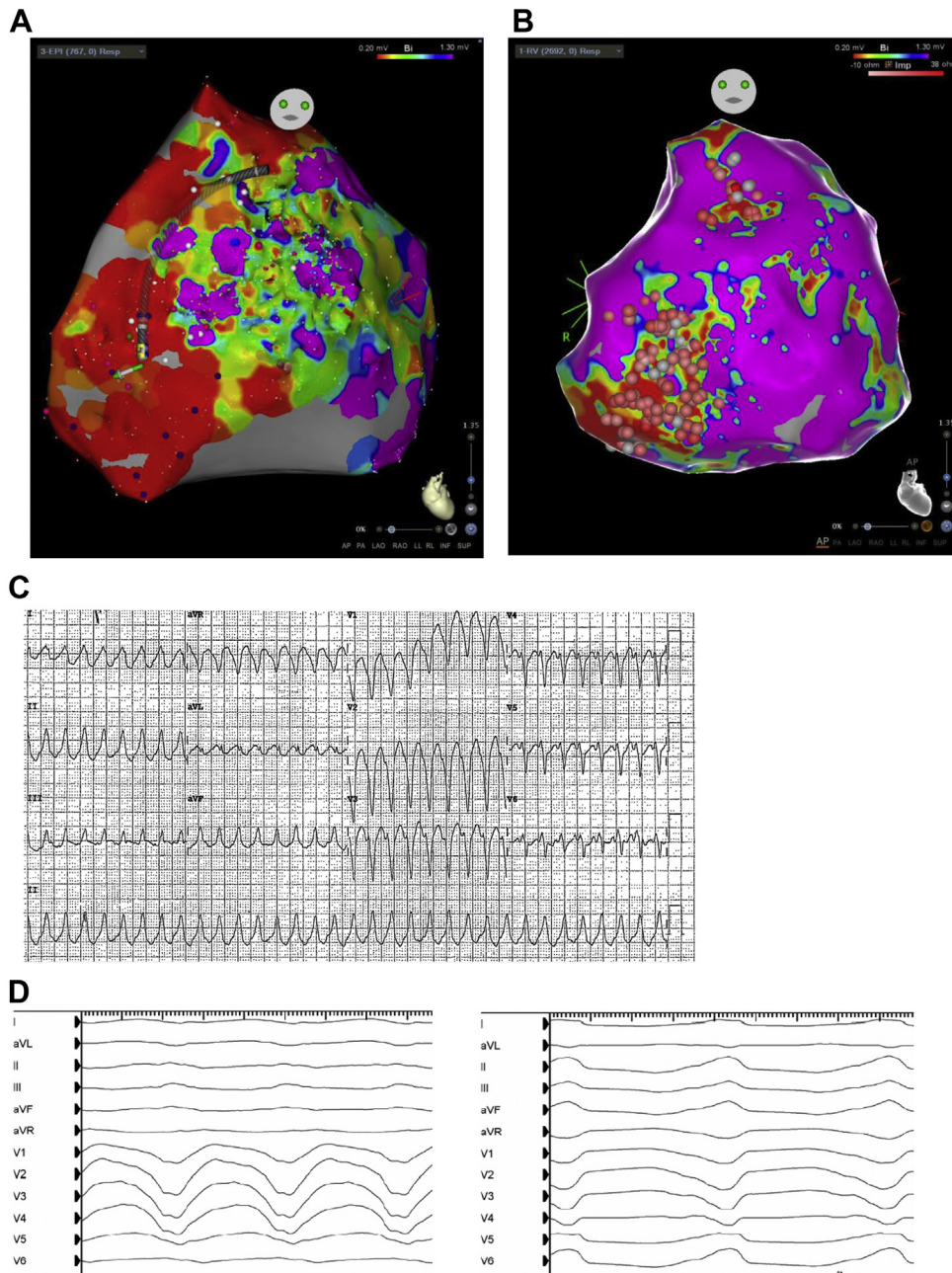


Figure 3 A: Epicardial voltage map. Catheter projection over site of 12/12 pace map to ventricular tachycardia (VT) 3. B: Endocardial voltage map with endocardial lesions tagged. C: The patient's clinical VT 1. D: VT 2. E: VT 3.

fractionated and late potentials were targeted. Endocardial ablation was also performed sandwiching the outflow tract sites and an area of patchy scar at the inferior lateral base. Lack of pace capture was documented along all areas targeted for ablation with high-output unipolar pacing (up to 20 mA, 2 ms). With triple extrastimuli from the RV apex, no sustained arrhythmias remained inducible.

Throughout the case, the pericardial space was continuously aspirated. As no intrapericardial bleeding was noted, the access was removed after administration of 120 mg of intrapericardial methylprednisolone. ICD detections were resumed. All access sheaths were removed with manual pressure for

hemostasis and the patient was observed overnight. An echocardiogram performed the following morning demonstrated absence of any pericardial effusion. The patient complained of mild chest pain, which was treated with colchicine, and he was discharged later that morning in stable condition. No recurrences have been documented to date.

Discussion

Since the advent of epicardial percutaneous access by Sosa and colleagues,⁶ several alterations have been developed to minimize the risk associated with epicardial puncture,

including favoring an anterior approach, use of a micropuncture needle or “needle-in-needle,” or use of a pressure sensor needle.^{7–11} However, RV puncture remains a common complication, and while usually bleeding is self-limited, it occasionally requires surgical correction, particularly for a “through-and-through” double RV puncture, which may go unnoticed until the end of a procedure.¹² Insufflation of CO₂, as shown by Juliá and colleagues,⁵ has an excellent safety profile and virtually eliminates the risk of RV or coronary puncture.^{5,13,14} In their series, no complications were noted from the pericardial access itself. In the case described above, their technique provided more separation for puncture from the sternum and mammary vessels in a patient with an enlarged RV, and also provided visualization of pericardial adhesions prior to attempts at access, so that puncture could be guided into areas without adhesions. In addition, despite difficulty with wire advancement, the presence of CO₂ improved capacity to confirm the appropriate tissue plane and avoid procedural abandonment for what otherwise would have been uncertain. The AIV was chosen as a target in this case owing to the ease of engaging it with the initial 0.35” wire. However, in considering future use of this technique for upcoming cases, the authors have considered favoring a CS branch other than the AIV, in order to avoid any theoretical risk related to wire puncture in proximity to the left anterior descending artery or inadvertently engaging septal vessels. Nevertheless, targeting the AIV proved safe in this case as well as those reported by Juliá and colleagues.⁵

It may strike one as counterintuitive to intentionally puncture a hole in a vessel, just to avoid the potential of creating a hole in the myocardium, particularly as micropuncture-based puncture of the RV rarely leads to significant bleeding. In our case, however, we chose a microcatheter with an exceptionally narrow profile, only 1.4F at the tip and 1.9F at the distal shaft, thereby being even narrower and conferring roughly 3-fold greater resistance than the outer diameter of a 21G micropuncture needle (0.63 mm vs 0.82 mm) or the 2.5–2.6F microcatheters (0.83 mm to 0.87 mm) used by Juliá and colleagues.⁵ That that puncture hole harbors substantially greater resistance and occurs within the lower pressure of the distal coronary venous system likely explains the absence of bleeding in our case example.

Another concern may relate to the hemodynamic influence of pericardial CO₂. We intentionally insufflated slowly and only to a degree where sufficient pericardial separation was appreciated. It is unclear if tamponade may have occurred if greater CO₂ were inflated. However, in the registry data from Juliá and colleagues,⁵ no such cases were noted, either from excess CO₂ or from any untoward bleeding from the coronary venous exit. Several features make CO₂ an attractive insufflation agent for this purpose. Unlike oxygen or air, it does not support combustion. Furthermore, its high solubility lends itself to being rapidly absorbed and excreted through the lungs, minimizing the duration of any effects on hemodynamics or defibrillation thresholds. Finally, its buoyant nature imparts a tendency to accumulate anteriorly in a supine patient. Despite these

advantages, similar results might be obtained from injecting a suitable volume of contrast or saline, the former actually facilitating a posterior approach by accumulating inferiorly.¹⁵ Overall, we feel that this approach may improve the safety of epicardial puncture for electrophysiological procedures.

Conclusion

In summary, we present a case of redo epicardial access facilitated with CO₂ insufflation. We believe that such an approach may improve the safety of percutaneous epicardial access in a general sense, but in this case was also instrumental in highlighting the presence and localization of adhesions. Further study is needed to compare epicardial access techniques in a prospective manner to determine the relative risks and drawbacks of specific techniques.

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Appendix Supplementary data

Supplementary data associated with this article can be found in the online version at <https://doi.org/10.1016/j.hrcr.2020.12.008>.

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