

Uncovering “bipolar blindness” with high-density orthogonal mapping at the scar-related critical isthmus in repaired congenitally corrected transposition of the great arteries



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Introduction

Scar-mediated atrial and ventricular arrhythmias are leading indications for hospitalization and risk factors for sudden cardiac death in the postoperative congenital heart population.¹ Successful transcatheter arrhythmia ablation would, in turn, decrease hospitalizations, improve patient quality of life, and drive down healthcare costs. Arrhythmia mapping and ablation in these patients, however, is plagued by low-voltage substrates, multiple circuits, and tachycardia recurrence.^{2,3} Herein, we discuss a patient with congenitally corrected transposition of the great arteries who underwent a double-switch procedure and subsequently developed atrial arrhythmias that were refractory to antiarrhythmic therapy and antitachycardia pacing. Transcatheter ablation using high-density mapping delineated his complex arrhythmia circuit, permitting successful ablation.

Case report

The patient is a 28-year-old man with congenitally corrected transposition of the great arteries, large inlet ventricular septal defect (VSD), and straddling tricuspid valve through the VSD, as well as valvular and subvalvular pulmonary stenosis. At age 15 he underwent an arterial switch with a Mustard procedure (“double switch”), removal of accessory papillary muscle of tricuspid valve, VSD closure, and anterior commissuroplasty of the aortic valve. Preoperative cardiac magnetic resonance imaging revealed delayed enhancement at the mid-ventricular lateral left

ventricle consistent with a transmural myocardial infarction of unclear etiology. At age 18 he underwent aortic valve repair using a valve-sparing aortic root replacement with a 24 mm Hemashield tube graft and suture closures of residual VSDs. At age 23 he underwent aortic valve replacement for severe symptomatic aortic regurgitation with a 23 mm bovine pericardial valve along with surgical unroofing of the posterior coronary artery. Postoperatively he had complete heart block and a transvenous atrial-antitachycardia dual-chamber permanent pacemaker was placed.

The patient remained relatively asymptomatic until age 25 when he presented with symptomatic intra-atrial reentry tachycardia that was refractory to medical therapy (metoprolol, sotalol) and antitachycardia pacing, requiring several cardioversions. He was subsequently taken to the electrophysiology lab, where he underwent an ablation procedure that included 3-dimensional electroanatomic mapping (EnSite Precision, Abbott, St Paul, MN). Electrode catheters were placed in the left atrial appendage, inferior baffle, and subpulmonary right ventricle. A high-resolution mapping catheter (HD Grid; Abbott) was used to perform a substrate/scar map of the systemic venous atrium. Subsequently, at a paced cycle length of 180–200 ms, an intra-atrial reentry tachycardia (cycle length 320 ms) was serially induced. The HD Grid was used to create an activation map demonstrating that only 60% of the tachycardia cycle length was contained within the systemic venous atrium. Propagation and sparkle maps demonstrated an area of breakout at the superior vena cava posteriorly. Using an irrigated ablation catheter (FlexAbility; Abbott) a radiofrequency lesion was placed at this site with no effect on tachycardia. To delineate the complete tachycardia circuit, a transbaffle puncture was performed under fluoroscopic guidance, during which the initial tachycardia was terminated and could not be reinduced. During attempted reinduction, a second tachycardia (cycle length 290 ms) was induced. Entrainment pacing demonstrated that the cavotricuspid

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KEY TEACHING POINTS

- Congenitally corrected transposition of the great arteries is characterized by discordant atrioventricular and ventriculoarterial connections with an incidence of 1 in 33,000 live births, accounting for <0.1% of congenital heart defects. Most patients (94%) have an associated congenital heart defect, most commonly tricuspid valve abnormalities (91%), ventricular septal defect (79%), subpulmonary obstruction (44%), and dextrocardia (25%–30%).
- Congenital (preoperative) arrhythmia substrates include (1) abnormal AV node with dual AV nodes and an abnormal His bundle with a 2% annual risk of complete heart block, (2) accessory pathway-mediated tachycardia (most commonly due to a left posteroseptal accessory pathway), and (3) ventricular arrhythmias (chronic systemic right ventricular dysfunction).
- Postoperative arrhythmias are related to the postsurgical substrate. Most commonly, ventricular septal defect or tricuspid valve repair frequently result in complete heart block. Less commonly, a double-switch procedure (atrial switch and arterial switch) can result in low-voltage, scar-related arrhythmias, as well as ventricular tachycardia owing to postoperative left ventricular dysfunction.
- Bipolar blindness is a phenomenon during electroanatomic mapping where bipolar recordings document activation wavefronts that travel parallel to an electrode pair but ignore the activation wavefronts that travel perpendicular to an electrode pair. This directional sensitivity causes “bipolar blindness” to nonparallel wavefronts that could prevent delineation of the arrhythmia circuit.
- High-definition orthogonal mapping allows data point collection in difficult anatomic orientations in adult congenital patients. It also allows for collection of low-voltage signals, thereby improving likelihood of successful mapping of the arrhythmia circuit and precise ablation, improving long-term success.

isthmus was an obligatory limb of the tachycardia circuit. Radiofrequency lesions placed at the cavotricuspid isthmus in the pulmonary venous atrium (PVA) terminated tachycardia, after which bidirectional block was demonstrated.

Burst pacing on dobutamine induced a third tachycardia (cycle length of 190 ms). A high-density map was performed demonstrating 100% of the tachycardia cycle length within the PVA. This activation map demonstrated an area of slow conduction and reentry at the superior PVA with the isthmus at the base of the PVA appendage (Figure 1). Entrainment pacing confirmed this region as an obligatory limb of the circuit. The irrigated ablation catheter was used to deliver 35 watts of radiofrequency energy to create 14 lesions across this isthmus region (Figure 2d). During ablation along the atrial roof, the tachycardia was terminated (Figure 2c). Following termination there was evidence of bidirectional block and no further tachycardias could be induced. The patient continues to be arrhythmia-free during routine follow-up visits over a 2-year period.

Discussion

Adult patients with complex congenital heart defects have often undergone multiple surgical procedures that, in turn, may produce complex arrhythmia substrates. These patients may also have altered hemodynamics, which affect their ability to tolerate scar-related tachyarrhythmias. Transcatheter ablation can be curative, but electroanatomic mapping is often hindered by low-voltage substrates and complex anatomies.⁴ This case illustrates the additional benefits of orthogonal mapping, in addition to traditional bipolar mapping, in uncovering a scar-related critical isthmus in our patient.

During electroanatomic mapping, bipolar recordings document activation wavefronts that travel parallel to an electrode pair, but ignore the activation wavefronts that travel perpendicular to an electrode pair.⁵ This directional sensitivity causes “bipolar blindness” to nonparallel wavefronts that could prevent delineation of the arrhythmia circuit.⁶ In postoperative congenital heart disease, with low-voltage arrhythmia substrates and complex anatomies that limit catheter positioning, bipolar blindness could result in a failed ablation procedure.

In this example, the HD Grid was positioned at the base of the pulmonary venous atrial appendage and the signals within the isthmus were recorded (Figure 3). The HD Grid takes advantage of the fixed 3-mm spacing of the bipoles, down and across the splines, permitting recordings of the

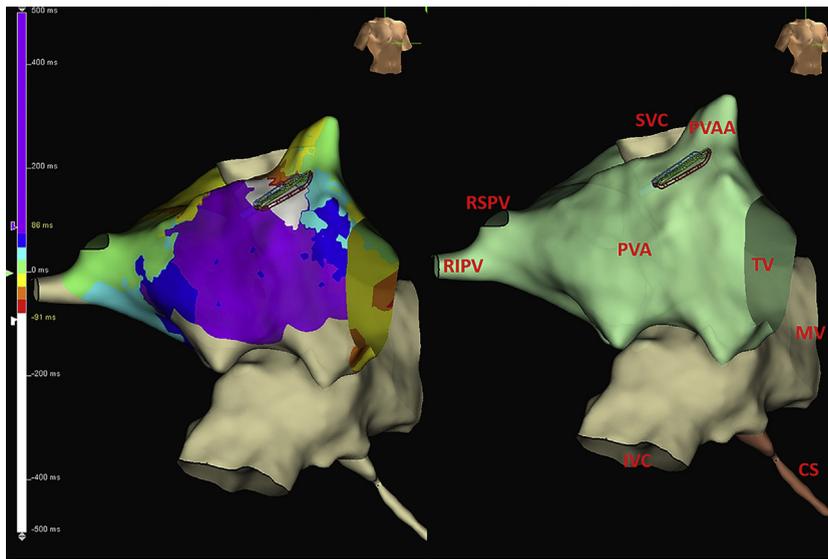


Figure 1 Double-switch atrial anatomic orientation (right anterior oblique): mitral valve (MV), tricuspid valve (TV), superior vena cava (SVC), inferior vena cava (IVC), coronary sinus (CS), pulmonary venous atrium (PVA), pulmonary venous atrial appendage (PVAA), right superior pulmonary vein (RSPV), right inferior pulmonary vein (RIPV).

orthogonal wavefronts (displayed as white electrograms in [Figure 3](#)), thereby providing additional and potentially important information that can help define an arrhythmia circuit.⁷ Without these additional data, the linear “bipolar” splines (displayed as blue and green electrograms in [Figure 3](#)) failed to detect the perpendicular propagation through the isthmus.

Postprocessing of the mapping data after the procedure revealed that without the orthogonal electrograms, the full extent of the isthmus along the pulmonary venous atrial roof where termination was achieved was not apparent

([Figure 2a](#)). Importantly, the “Bipolar Only” and the “Bipolar + Orthogonal” maps within [Figure 2](#) have the same point density and point collection, but the “Bipolar + Orthogonal” map takes advantage of the best duplicate algorithm (EnSite Precision, Abbott). This algorithm allows for automatic comparison of the bipolar and orthogonal electrograms associated with the catheter electrodes and determines the better electrogram to be annotated for a given cathode. Utilizing the best duplicate algorithm in conjunction with the HD Grid catheter to eliminate “bipolar blindness” allowed the full isthmus to be identified, where tachycardia termination was achieved ([Figure 2c](#)).

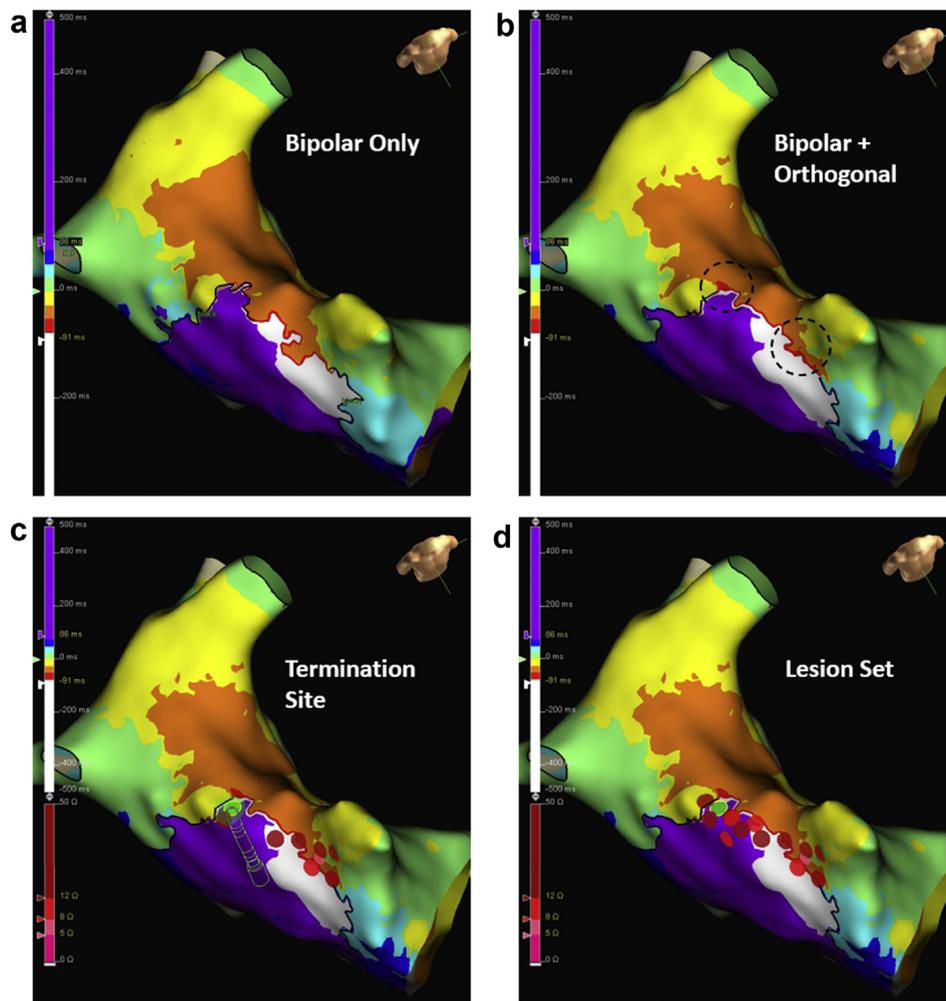


Figure 2 **a:** Arrhythmia map composed of only bipolar data along the splines of the HD Grid catheter (Abbott, St Paul, MN). **b:** Arrhythmia map composed of bipolar and orthogonal data from along the splines and across the splines of the HD Grid. The 2 dashed, black circles display areas of isthmus conduction that were improperly identified as block in the “Bipolar Only” map. **c:** Ablation catheter position and lesion set when arrhythmia termination was achieved. Of note, the portion of the isthmus where ablation termination occurred was only identified in the “Bipolar + Orthogonal” map. **d:** Final lesion set with colors corresponding as follows: dark red (>12 ohm impedance drop), bright red (>8 ohm impedance drop), pink (<8 ohm impedance drop), and green (termination site).

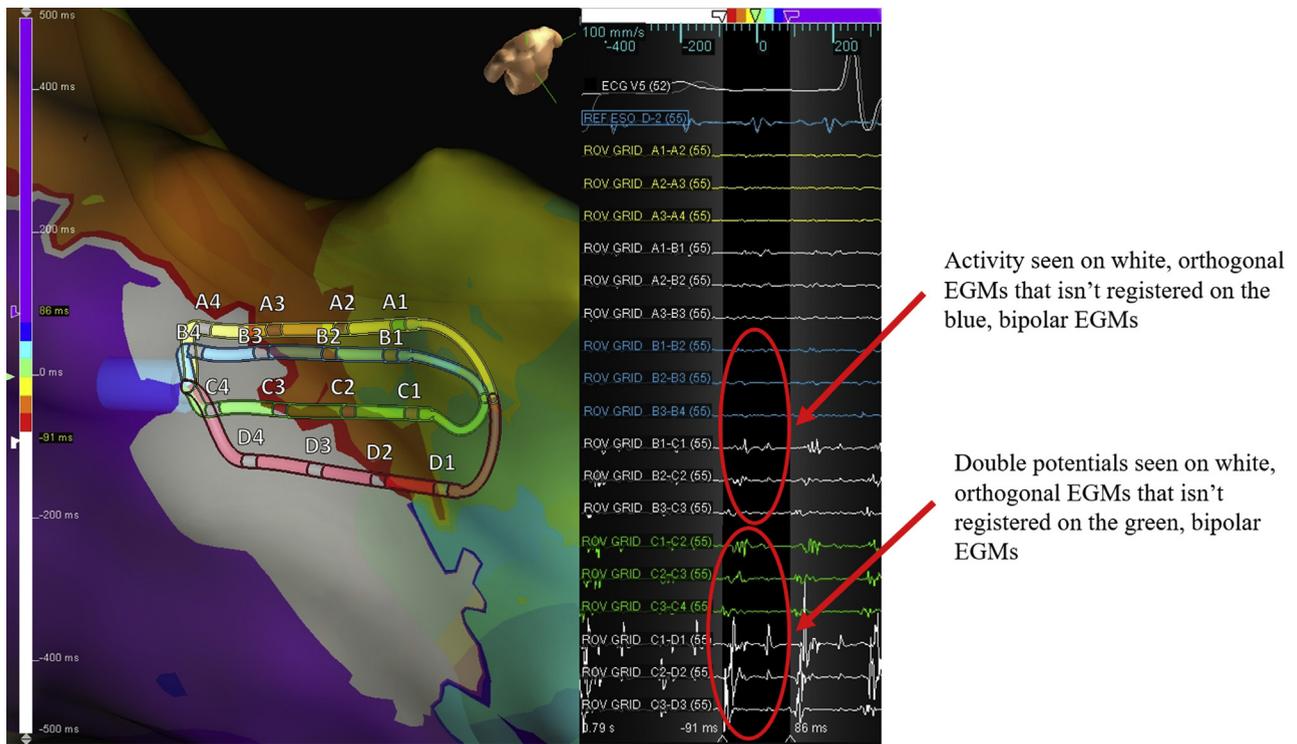


Figure 3 HD Grid (Abbott, St Paul, MN) signals recorded in the isthmus at the base of the pulmonary venous atrial appendage. EGM = electrogram.

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