Diopathic ventricular arrhythmias not infrequently arise from the left ventricular summit, the superior most portion of the epicardium near the bifurcation of the left main coronary artery. These arrhythmias have an inferior axis and usually demonstrate a negative to isoelectric configuration in lead II. Typically, there is a left bundle branch block pattern in lead V1, with early precordial transition. Often there is a pattern break in lead V2, with a more net negative complex compared with leads V1 and V3. The epicardial left ventricular summit is best accessed for catheter ablation via the coronary venous system. However, ablation within the anterior interventricular vein (AIV) can be limited by inability to advance the catheter to the site of interest, inadequate power delivery secondary to impedance/temperature rises, and most importantly, proximity to the left anterior descending (LAD) coronary artery. When one of these limitations is present, ablation can be successful from nearby structures, including the left coronary cusp or left ventricular endocardium. We describe for the first time successful elimination of ventricular arrhythmias arising from the left ventricular summit AIV region with ablation from the nearby right ventricular outflow tract (RVOT).

**Case No. 1**

A 30-year-old woman presented with palpitations. Holter monitoring revealed a 17% burden of monomorphic ventricular premature depolarizations (VPDs). Transthoracic echocardiogram was normal. After treatment with a β-blocker failed, baseline rhythm was sinus with frequent VPDs (right bundle branch block morphology in lead V1, right inferior axis). Activation mapping was performed in the AIV, left coronary cusp, and left ventricular endocardium. Activation was earliest in the AIV (25 ms pre-VPD; Figure [A]). However, coronary angiography revealed immediate proximity of the site of earliest activation to the LAD (Figure [C]). The ablation catheter (Thermocoool, Biosense Webster, Diamond Bar, CA) was withdrawn slightly and a low power lesion delivered within the AIV, without VPD suppression. The earliest activation within the left coronary cusp was 5 ms later than in the AIV, and the anatomic separation between the two was 15 mm. Ablation in the left coronary cusp had no effect. The earliest activation within the left ventricular endocardium was 10 ms later than the AIV. Ablation at that earliest left ventricular site suppressed the VPD late into the lesion, with immediate return of VPDs after the lesion.

Next, the RVOT was mapped. Activation in the leftmost aspect of the septal RVOT was just 2 ms later than the AIV, with an initial far-field component (Figure [A]). Pacemap from the RVOT was a poor match. The anatomic separation between the earliest RVOT and AIV sites was 9 mm (Figure [C]). Ablation in the RVOT (20–30 W, 60 seconds) eliminated the VPD within 4 seconds, and she remains arrhythmia free 2.5 years later.

**Case No. 2**

A 56-year-old woman presented with recurrent episodes of palpitations and presyncope and was documented to have ventricular tachycardia on event monitoring. Echocardiogram, nuclear stress test, and cardiac MRI were all normal. Catheter ablation was performed.

Monomorphic ventricular tachycardia was induced with burst pacing during isoproterenol infusion (left bundle branch block morphology, inferior axis, precordial transition V3, and cycle length 310 ms; Figure [B]). The ventricular tachycardia was not hemodynamically tolerated. Pacing was performed in the leftmost aspect of the septal RVOT, with a good, although not perfect match (Figure [B]). Pacing was performed in the AIV, with a perfect match for the ventricular tachycardia (note precordial lead V3). Coronary angiography revealed immediate proximity of the site of the best AIV pacemap to the LAD, prohibiting safe ablation. Ablation (Thermocoool, 20–40 W, 60 seconds) was instead delivered at the closest site within the RVOT, just 8 mm away. After several ablation lesions in the RVOT, ventricular tachycardia...
could no longer be induced and has not recurred during 1.5-year follow-up.

**Discussion**
To our knowledge, this is the first report of ablation in the RVOT to eliminate ventricular arrhythmias originating from the AIV. In both cases, the site of origin of the ventricular arrhythmia within the AIV was too close to the LAD for safe ablation, a commonly encountered problem. When this is the case, rather than risk injury to the LAD, mapping should be performed in adjacent structures. In addition to mapping the left coronary cusp and left ventricular endocardium, the RVOT can be mapped as well. It is important to advance the catheter as leftward as possible within the RVOT to minimize separation from the AIV. Left anterior oblique fluoroscopy and intracardiac echocardiography can facilitate optimal catheter positioning. If activation and pace mapping are only slightly worse than in the AIV and the anatomic separation is minimal, consideration can be given to ablating within the RVOT. In both our cases, the anatomic separation was <10 mm. For comparison, we have previously reported an anatomic separation of <13.5 mm between the left coronary cusp and the best site in the AIV or great cardiac vein, to permit successful ablation from the left coronary cusp.1

Operators should be mindful of the proximity of the RVOT to not only the AIV but also the LAD. Although to the best of our knowledge coronary injury has not been reported with ablation in the RVOT, a study of gross pathology and computed tomography angiography measured a mean of 2.0 mm minimum distance between the RVOT and LAD.2 The anatomic distance to the LAD is at a minimum 2 to 3 cm below the plane of the pulmonic valve. Thus, when targeting the AIV from the leftmost aspect of the septal RVOT, care should be taken to define the LAD location and to confirm a more superior site of ablation within the RVOT, using the right anterior oblique fluoroscopic projection (Figure [C]).

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**Disclosures**
None.

**References**

**Figure.** Ventricular arrhythmia morphologies, activation and pace mapping, and anatomic relationship among the anterior interventricular vein, left anterior descending coronary artery, and right ventricular outflow tract. **A.** Activation during ventricular premature depolarization of patient No. 1 was earliest in the anterior interventricular vein (AIV), and only slightly later in the right ventricular outflow tract (RVOT). Note the initial far-field component to the electrogram recorded from the RVOT. The pacemap from the RVOT is a poor match for the ventricular premature depolarization, with a left bundle branch block morphology in lead V1, rather than a right bundle branch block morphology. **B.** Pacemapping from the AIV was a closer match to the ventricular tachycardia (VT) of patient No. 2 than pacemapping from the RVOT. The difference is most pronounced in lead V3. Note the pattern break in both patients, with a more net negative complex in lead V2 compared to leads V1 and V3. **C.** Right anterior oblique (RAO) and left anterior oblique (LAO) projections show left coronary angiography with an ablation catheter in the AIV of patient No. 1. Superimposed are the images of the ablation catheter in the successful ablation site in the RVOT. Note the immediate proximity of the ablation catheter in the AIV to the left anterior descending coronary artery (LAD), prohibiting safe ablation. Also, note that the ablation catheter in the RVOT (just beneath the pulmonic valve), while close to the arrhythmia site of origin in the AIV, is above the level of the LAD. LCX indicates left circumflex coronary artery; and LMCA, left main coronary artery.
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