Mechanisms and Utility of Discrete Great Arterial Potentials in the Ablation of Outflow Tract Ventricular Arrhythmias

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Background—Outflow tract ventricular tachycardia originating above the semilunar valves has been reported in a small number of studies. Discrete potentials in the great arteries (above the semilunar valves) have been rarely described in patients undergoing electrophysiology evaluation and radiofrequency ablation for ventricular arrhythmias. The mechanisms of these discrete potentials in the great arteries and the utility of such potentials in guiding radiofrequency ablation are unknown.

Methods and Results—Twelve patients with outflow tract ventricular arrhythmia originating above the semilunar valves with discrete arterial potentials were studied. The clinical characteristics, properties of the arterial potentials, electrophysiological evaluation and ablation, and short- and long-term outcomes were reviewed. Of the twelve patients, 8 (67%) were women. The patients’ average age was 41±14 years. The average ejection fraction was 0.52±0.16 (range: 0.16 to 0.75). Contact mapping in the great artery demonstrated discrete near-field electrograms that were separate from far-field ventricular electrograms in all patients (8 above the pulmonary valve and in 4 the aortic valve). One or more of the following electrophysiological characteristics, supportive of an arrhythmogenic substrate, were observed in 10 of 12 patients: (1) A fixed or reproducibly variable pattern of discrete potential–ventricular arrhythmia relationship was present at baseline or during pacing; (2) the discrete potential–ventricular electrogram relationship during sinus rhythm was the reverse of that during the ventricular arrhythmia; (3) during sustained ventricular tachycardia, spontaneous variation of the ventricular (V-V) cycle length was preceded by a similar variation of arterial spike potential–spike potential cycle length; and (4) ablation guided by the discrete arterial potential successfully eliminated the clinical arrhythmia. Ablation was successful in these patients. In the remaining 2 patients, the potentials were believed to be bystanders. Over 10±4 months (range: 5 to 32 months) of follow-up, there have been no recurrences of the premature ventricular complex or ventricular arrhythmia.

Conclusions—Discrete potentials are present in the great arteries of a select group of patients with outflow tract ventricular tachycardia originating above the semilunar valves. When an arrhythmogenic relationship can be demonstrated, discrete potentials are useful in guiding ablation within the great vessels, despite significant anatomic complexity. (Circ Arrhythmia Electrophysiol. 2008;1:30-38.)

Key Words: arrhythmias, cardiac, ventricular ▪ aorta ▪ tachycardia, ventricular ▪ pulmonary artery ▪ electrophysiology ▪ catheter ablation, radiofrequency

I diopathic ventricular tachycardia (VT) accounts for ≈10% of patients referred for evaluation of VT. In this subset of patients, outflow tract VT is the most common form. This type of VT typically arises below the semilunar valves in the region of the right or left ventricular outflow tracts, along multiple sites of the septum, near the His bundle, and on the epicardial surface of the ventricles. VT originating above the level of the semilunar valves has also been described in a small number of case studies.4–8,11–14 Ventricular myocardium extends up to the semilunar valves and is circumferential in the pulmonary root but incomplete in the aorta because of the intervalvular fibrosa.15,16 Recent studies have shown that ventricular myocardial extensions extend into the pulmonary artery and aorta beyond the

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semilunar valves. Ventricular myocardium extending into the great vessels above the semilunar valves may be a trigger for the arrhythmia, similar to that observed from the superior vena cava and pulmonary veins in patients with atrial fibrillation.

**Clinical Perspective p 38**

Radiofrequency ablation is a highly successful means to treat outflow tract VT. Prior studies have typically involved patients in whom the VT was triggered from sites below the semilunar valves. Less is known about treatment and mapping of idiopathic outflow tract VT originating above the semilunar valves.

**Methods**

**Study Population**

All patients undergoing ventricular outflow tract VT and/or premature ventricular complex (PVC) ablation between 2001 and 2006 at the Mayo Clinic were studied. Patients were included if the clinical arrhythmia was found to be at or above the semilunar valves. Patients were excluded if the clinical arrhythmia was found to be below the semilunar valves. Procedure notes, intracardiac electrogram, electroanatomic and noncontact mapping data, and intracardiac ultrasound data were reviewed. Discrete potentials at or above the semilunar valves during either sinus rhythm or VT/PVCs were further analyzed. The authors had full access to and take full responsibility for the integrity of the data. All authors have read and agree to this manuscript as written.

**Diagnosis of Discrete Potentials**

Bipolar recordings were filtered at 30 to 500 Hz. Discrete sharp electrograms separated from far-field ventricular electrograms by an isoelectric period were considered discrete potentials. The presence of such potentials, as well as the extent of the isoelectric period between ventricular and atrial activation in sinus rhythm and atrial and ventricular pacing, was noted. The site at which these electrograms were recognized was further localized with fluoroscopic and intracardiac ultrasound data. When intracardiac ultrasound was available, the distances from the potential to the semilunar valves were noted. The relationship of the potentials in the great arteries and the QRS complex/local ventricular electrograms during premature beats and other ventricular arrhythmia was noted. Pace mapping data from these sites were collected.

**Mapping Technique**

Deflectable electrophysiological catheters (EP Technologies, Biosense Webster) were advanced via the femoral veins to map the right ventricular outflow tract. A retrograde transaortic approach was used to map the left ventricular outflow tract. The catheters were guided with fluoroscopy and intracardiac ultrasound. Point-to-point mapping identified the earliest site of origin for the ectopic beats or VT. The exact anatomic site with reference to the semilunar valve and coronary arteries was noted with either intracardiac ultrasound or coronary angiography.

**Electroanatomic Mapping**

The CARTO System (Biosense Webster, Johnson & Johnson, Diamond Bar, Calif) was used with standard techniques in cases in which frequent ventricular arrhythmias (spontaneous or induced) were present to aid accurate localization and to allow redeployment of a catheter to a site of good pace mapping or early electrogram during arrhythmia.

**Noncontact Mapping**

In 2 cases, standard techniques of noncontact mapping (Endocardial Solutions, St. Paul, Minn) were used, with a multielectrode array placed in the right ventricular outflow tract. The technique was as described elsewhere. In these cases, either the VT was unstable, or rare premature beats were noted during the electrophysiological study.

**Intracardiac Ultrasound**

A linear phased-array probe was placed in the right atrium, tricuspid annulus, or the right ventricular outflow tract via the right femoral vein. A 10-French or 8-French catheter with bidirectional deflection was used (Acuson Siemens Corp, Mountain View, Calif). Ultrasound imaging was used to exclude aortic arch thrombus/debris, identify the location of the semilunar valve in relation to the ablation catheter (Figure 1), and estimate proximity of the ostia of the main coronary arteries to the ablation catheter.

**Ablation Procedure**

The primary ablation target was to identify the earliest local activation of the discrete potential to the onset of surface QRS.
complex during PVCs or VT and when the discrete potential was clearly associated with the clinical arrhythmia. If the discrete potential was not associated with the clinical arrhythmia, ablation was targeted to sites where the local electrogram preceded the onset of the surface QRS by 15 ms. Pace mapping was attempted in all cases to reproduce the exact morphology of the PVC/VT, although this was frequently limited by noncapture even at high output (20 mAmp) when pacing was conducted above the semilunar valves. When the site of earliest local activation or best pace map was found at a site unsuitable for ablation (for example, near a coronary artery origin [<5 mm] or on the semilunar valve), coronary angiography or intracardiac ultrasound guidance was used to monitor the energy delivery. If the coronary artery ostium was believed to be too close to the target site, other strategies were used. In one instance, ablation of the focus involved isolation of the aortic outflow trunk, with dissociation of the local electrogram in the outflow tract and great arteries from the rest of the ventricle.

Ablation was performed by delivering radiofrequency energy with a standard deflectable 4- or 5-mm-tip ablation catheter. The output was adjusted to between 5 and 50 W to achieve a target temperature of 45 to 60°C. When the ablation site was near a coronary artery origin (between 5 and 20 mm), energy was started at low output (5 to 10 W) and titrated upward according to the catheter tip temperature and patient’s clinical response, such as chest pain or hemodynamic or electrocardiographic changes.

Results

Patient Characteristics and Procedures

Of the total of 120 patients undergoing outflow tract tachycardia ablation between 2001 and 2006, 12 patients met the inclusion criteria (Table). The average age was 41 ± 14 years (range: 18 to 61 years), and 8 (67%) were women. The average ejection fraction was 0.52 ± 0.16 (range: 0.16 to 0.75). Five patients had dilated cardiomyopathy (ejection fractions: 0.45, 0.45, 0.43, 0.40, and 0.16). Four had New York Heart Association Class II heart failure, and one had Class III heart failure (ejection fraction 0.16). The patients’ symptoms, clinical history, and arrhythmia characterization are summarized in the Table.

Presence of Great Arterial Potentials

In all 12 patients, contact mapping in the great artery demonstrated a discrete near-field electrogram that was separate from a far-field ventricular electrogram. In 8 patients, these electrograms originated above the pulmonary valve. The region in which the electrogram was earliest was variable (3 right anterior cusp, 3 posterior cusp, 2 between the right anterior and posterior cusps). In 4 patients, the potentials were above the aortic valve. The site of the discrete potential was <5 mm away from the left main coronary artery in 2 (Figure 2). In 3 patients, a Lasso catheter was placed in the aortic root to further characterize the activation. The potentials tended to be earliest near the left coronary cusp, with the exception of one case in which they were seen at the junction of the left and noncoronary cusps. In the case with potentials earliest near the noncoronary cusp, electrograms were present along two thirds of the aortic vessel circumference.

In those patients with potentials arising above the aortic valve, the ECG was characterized by a left bundle-branch morphology (7 of 8), with an inferior axis (R in III similar to II) and a Rs or QS in V1. In those with electrograms arising from above the pulmonary valve, the ECG was characterized by a left bundle-branch morphology (7 of 8), with an inferior axis (R in III similar to II) and a Rs or QS in V1.

At baseline, we observed 2 patterns to describe these potentials:

1. A discrete electrogram was present during sinus rhythm, with an apparent association to the normal QRS or far-field ventricular electrogram. For example, the patient shown in Figure 3 has a right bundle-branch block during sinus rhythm. The mapping catheter was placed 2 cm above the pulmonary valve (confirmed by intracardiac echocardiogram). The interval from the onset of surface QRS complex to the local spike potential (QRS-S) was 250 ms. As the catheter was advanced further up in the pulmonary artery (away from the valve), the amplitude of the discrete potential decreased before the potential eventually disappeared. After the catheter was pulled back to 5 mm above the pulmonary valve, the amplitude of the potential increased, while the delay between the near-field spike potential and the far-field ventricular electrogram (QRS-S) was less than the delay recorded further above the valve. These observations suggest that the spike potential is associated with the QRS during sinus rhythm. During sinus rhythm, ventricular depolarization proceeded from below the valve to above the valve tissue, with recordable potentials extended to 2 cm above the pulmonary valve.

2. A discrete potential was not apparent during sinus rhythm, but appeared only during the ventricular arrhythmia, as shown in Figure 4. In the patient shown in Figure 4, the mapping catheter was positioned above the pulmonary valve. A far-field atrial electrogram was present during sinus rhythm, whereas no discrete near-field potential was apparently present. In association with spontaneous clinical PVCs, a discrete potential preceded the local ventricular electrogram by 30 ms. The absence of a discrete potential during sinus rhythm could be explained if the near-field discrete potential was fused with the far-field ventricular electrogram. This pattern was seen in 5 patients, whereas a discrete electrogram was noted in sinus rhythm in the other 7 patients.

In Figure 5, the discrete potential begins within the far-field signal from the local ventricle. A clinical PVC is shown, with the relationship of the discrete potential to far-field signal reversed. This was followed by pacing from the distal ablation catheter tip and capture of the discrete potential and a QRS similar to that of the clinical PVC.

Arterial Potential–Ventricular Arrhythmia Relationship: Evidence for Arrhythmogenic Substrate

Spontaneous Reversal of the Discrete Potential–Ventricular Electrogram Relationship

During sinus rhythm, spontaneous discrete potentials after the normal QRS complex were noted in 4 patients. The spontaneous reversal of the relationship of the spike potential to the local ventricular electrogram during ventricular arrhythmia is shown in Figure 6. In this figure, the ablation catheter is positioned above the pulmonary valve. During sinus rhythm, the spike is late and buried in the far-field electrogram. The third beat is a fusion beat between the VT and sinus. Here, the spike comes earlier than sinus but not as early as the nonfused
### Table. Clinical and Procedural Characteristics of Patients With Discrete Electrograms Above the Semilunar Valves

| Patient | Age, y | Sex | Clinical History | Intracardiac Echocardiography | Left Ventricular Outflow Tract Potential to Ventricular Electrogram, ms | Right Ventricular Outflow Tract Potential to Ventricular Electrogram, ms | Potential in Sinus Rhythm | Clinical Arrhythmia | Type of Mapping | Ablation Site | Ablation Attempts (Total) | Follow-Up, mo |
|---------|--------|-----|------------------|------------------------------|-----------------------------|-----------------------------|-----------------------------|--------------------------|-----------------|----------------|----------------|----------------|----------------|-------------|
| 1       | 43     | F   | 50,000 PVCs per day; palpitations | Yes                          | . . .                        | Yes                          | Yes                         | PVC                      | Noncontact mapping  | RA             | 29                        | 32            |
| 2       | 18     | F   | Frequent symptomatic PVCs; dizziness/ lightheadedness | Yes                          | . . .                        | Yes                          | Yes                         | PVC and atrioventricular nodal reentrant tachycardia | Electroanatomic mapping | RA             | 9                         | 10            |
| 3       | 54     | M   | VT; syncope       | No                           | 34                          | . . .                        | Yes                         | VT                       | Electroanatomic mapping | LCC            | 22                        | 10            |
| 4       | 45     | F   | Exercise-induced VT | Yes                          | . . .                        | 18                          | No                          | VT                       | Electroanatomic mapping | RA             | 15                        | 14            |
| 5       | 24     | M   | Frequent PVCs; family history of sudden cardiac death | Yes                          | 20                          | . . .                        | Yes                         | PVC                      | Noncontact mapping  | LCC/NCC        | 12                        | 8             |
| 6       | 43     | F   | 60,000 PVCs per day; idiopathic cardiomyopathy, ejection fraction 0.45 | Yes                          | . . .                        | 26                          | Yes                         | PVC                      | Noncontact mapping  | RA/P           | 3                         | 6             |
| 7       | 61     | F   | Nonsustained VT   | Yes                          | 21                          | . . .                        | No                          | PVC                      | Electroanatomic mapping | LCC            | 1                         | 5             |
| 8       | 37     | F   | PVCs in bigeminy; dilated cardiomyopathy, ejection fraction 0.40 | Yes                          | . . .                        | 28                          | Yes                         | PVC                      | Noncontact mapping  | P              | 33                        | 14            |
| 9       | 61     | M   | VT, ejection fraction 0.16; prosthetic aortic valve | Yes                          | . . .                        | 30                          | No                          | VT, 4 morphologies        | Electroanatomic mapping | P              | 20                        | 8             |
| 10      | 38     | F   | Syncope, VT       | Yes                          | . . .                        | 57                          | Yes                         | VT, 3 morphologies        | Electroanatomic mapping | RA/P           | 33                        | 6             |
| 11      | 25     | M   | Frequent PVCs; dilated cardiomyopathy, ejection fraction 0.45 | Yes                          | . . .                        | 75                          | Yes                         | PVC                      | Electroanatomic mapping | LCC            | 16                        | 18            |
| 12      | 38     | F   | Incessant VT      | Yes                          | . . .                        | 63                          | No                          | VT                       | Electroanatomic mapping | P              | 6                         | 5             |

M indicates male; F, female; LCC, left coronary cusp; NCC, noncoronary cusp; RA, right anterior cusp; and P, posterior cusp.
PVC, which is seen at the end of the tracing (during sinus rhythm, QRS-S=50 ms; during VT, QRS-S=−18 ms). In a second patient, a Lasso catheter was positioned at the root of the aorta (2 cm above the aortic root), while the mapping catheter was in the left aortic sinus (Figure 7). During sinus rhythm, the spike potential (on the mapping catheter) was late in relationship to the local ventricular electrogram (QRS-S=50 ms). During the PVC, the spike potential was significantly earlier (QRS-S=−25 ms). A similar reversal of the relationship of the spike to the ventricular electrogram also could be seen on Lasso electrodes. The relationship reversal during ventricular arrhythmia does not merely support the association of the discrete potential to the ventricular depolarization; a cause–effect relationship of the discrete potentials in the great arteries to the clinical arrhythmias is strongly implicated.

**Relationship of S-S Interval to V-V Interval During VT**

Among the 4 patients with clinical sustained ventricular arrhythmia, spontaneous variation of the tachycardia cycle lengths was observed repeatedly in 1 patient (Figure 8). During tachycardia, spike potential–spike potential (S-S) and interventricular (V-V) intervals were 356 and 354 ms, respectively. A shortening of the S-S interval from 356 to 341 ms during ventricular arrhythmia does not merely support the association of the discrete potential to the ventricular depolarization; a cause–effect relationship of the discrete potentials in the great arteries to the clinical arrhythmias is strongly implicated.
was followed by a prolongation of the V-V interval from 354 to 341 ms. The change in the S-S interval preceding the change in the V-V interval supports the notion that the spike potentials drive the tachycardia.

Bystander Potentials

In 2 patients, the discrete potentials were believed to be bystanders and not involved in the clinical arrhythmia. For example, in Figure 9, the potentials occurred late compared with the onset of the QRS, were not present during all QRS complexes, and did not change location in sinus rhythm versus the clinical arrhythmia.

Response to Ablation and Procedural Outcome

The targeted discrete electrograms, excluding those considered bystanders, occurred on average 44±10 ms (range: 26 to 75) before the onset of the QRS of the clinical PVC or VT. They were successfully ablated in all cases, with an average of 2.8±3.0 ablation attempts (range: 1 to 9) for 125±91 seconds once the target was the discrete potential above the

Figure 5. Discrete potentials are seen on the ablation catheter. The fourth QRS complex demonstrates a local reversal of the far-field and discrete potential recorded at the distal ABL site. Pacing from the distal ABL catheter shows capture of this discrete potential with a similar QRS morphology as that recorded on the nonpaced beat (fourth QRS complex). Abbreviations as in Figure 3.

Figure 6. The discrete potentials are shown late (arrowheads) at the ablation catheter tip in the normal tracings and then magnified on the right (A and B). At A, there is a far-field ventricular signal followed by the discrete potential. The third QRS complex is that of the clinical tachycardia. The relationship of the discrete potential to the far-field ventricular electrogram reverses. This observation was repeated during subsequent runs of the clinical VT. Abbreviations as in Figure 3.

Figure 7. Electrograms in sinus rhythm and with a clinical PVC are shown. Electrograms are displayed that were recorded by a Lasso catheter positioned in the aortic root. Discrete spike potentials (arrows) recorded by a Lasso catheter are present after the QRS in sinus rhythm but before the QRS during the clinical PVC. A similar change is noted with the discrete potential recorded by the distal ablation catheter. These potentials were captured when paced locally and were observed repeatedly in the same relationship with spontaneous PVCs. RVp indicates proximal right ventricle catheter; RVd, distal right ventricle catheter. Other abbreviations as in Figures 2 and 3.

Figure 8. The ablation catheter is superior to the pulmonary valve in a region between the right anterior and posterior pulmonary cusps. The discrete potentials shown are the probable drivers for the tachycardia. There is a delay between the spike and the ventricular electrogram. When this delay decreases from 356 to 341 ms, the tachycardia cycle length also decreases from 354 to 341 ms. This phenomenon was observed repeatedly. Abbreviations as in Figure 3.
Follow-up by echocardiogram. During follow-up, there was no recurrence of the PVC or VT. In the case of the second patient with multiple VTs and PVCs, point isolation of the aorta and left ventricle were guided by contact mapping catheters. In this case, circumferential ablation and ultimately isolation of the aorta and left ventricle were guided by insertion of a Lasso catheter into the aortic root. In the second patient with multiple VTs and PVCs, point isolation above the pulmonary valve terminated one arrhythmia. A second linear ablation line was drawn along the left ventricular septum, to a scar most likely related to prior aortic valve surgery, to terminate a second distinct left ventricular outflow tract VT. In all cases, the clinical arrhythmia was noninducible or the PVC did not recur after ablation.

Periprocedural complications included a vascular access site complication in 1 patient. No stroke, cardiac perforation with tamponade, coronary arterial damage, myocardial infarction, or valvular damage was seen in any of these cases.

Long-Term Follow-Up

The patients were followed up for an average of 10 ± 4 months (range: 5 to 32 months). The targeted PVC or VT did not recur during the follow-up period. In the 1 case in which isolation with circumferential ablation of the aortic root was performed, no evidence of stenosis was noted at 14 months’ follow-up by echocardiogram. During follow-up, there was no evidence of new-onset, symptomatic coronary artery disease in any of the patients.

In the subset of patients with dilated cardiomyopathy, the ejection fraction improved in 2 (ejection fraction 0.43 to 0.55, 0.45 to 0.59). The patient with a severely depressed ejection fraction of 0.16 did not improve his cardiac function or heart failure symptoms.

Discussion

In this report, contact mapping catheters were used to locate discrete electrograms in the great vessels above the semilunar valves. This case series study has provided electrophysiologic evidence to support the notion that these great arterial discrete potentials are involved in the ventricular arrhythmia substrate, analogous to pulmonary venous potentials and atrial fibrillation. Finally, in treatment of these potentials, we report a novel method in which a Lasso catheter is used to isolate the aortic root in a patient in whom the ideal ablation site was too close to the left main coronary artery.

An anatomic basis exists for the presence of these potentials. The truncus arteriosus initially arises entirely from the right ventricle, with a sleeve of myocardium separating the semilunar endocardial cushions from the atroventricular endocardial cushions. The truncus arteriosus then divides into the aortic and pulmonary artery trunks, and an infundibular septum forms between the developing aortic and pulmonary valves. This process results in a conus with a “figure-8” orifice. Gradually, the conus beneath the aortic valve regresses to allow the aorta to shift into the left ventricle. Retention of some of this conal muscle could allow myocardium to persist up along the aortic sinus. In comparison, the pulmonary valve normally sits in a crater of myocardium, which can extend variable distances above the annular insertion of the cusps. This myocardial extension may be particularly pronounced in patients with congenital right ventricular outflow tract obstructions, hypoplastic pulmonary valves, and hypoplastic pulmonary arteries. Recently ventricular myocardial extensions have been shown in both the pulmonary artery and aorta.

Foremost in the evaluation of these potentials is to separate them from the electrical signals from nearby structures such as the ventricle and atrium. This becomes particularly important when near-field and far-field signals are fused. Several maneuvers were used to delineate the origin of these potentials. For example, pacing the ventricle close to the semilunar valve but in the ventricular myocardium resulted in an earlier stimulus-to-electrogram time of the ventricular potential, with no significant effect on the near-field discrete potential. Next, the ventricle was paced at increasingly rapid rates, and the potential was seen to occur with either a similar or longer delay from the far-field ventricular electrogram. Then, pacing above the valve resulted in either capture or near-simultaneous occurrence of the pacing stimulus and the near-field great arterial electrogram. Finally, the atrium was paced with atrial capture, and no direct relationship was identified between the atrium and the discrete electrograms.

We next sought to determine the relationship of these potentials and the clinical arrhythmia. This relationship was supported by a fixed or reproducibly variable pattern of the
discrete potential–ventricular arrhythmia relationship at baseline or during pacing; a reversal of the discrete potential–ventricular electrogram relationship during sinus rhythm versus that during the ventricular arrhythmia; and during sustained VT, spontaneous variation of V–V cycle length being preceded by a similar variation of S–S cycle length. In the absence of inducibility of the clinical arrhythmia or spontaneous PVCs, pace mapping can provide potential evidence that the targeted site is appropriate. When attempting to perform pace mapping at a site where a discrete potential is found, it is important to pace at both high and low output. If the high and low output–generated surface QRS complexes are identical, the data do not allow a clear differentiation between the pacing and arrhythmia origin sites. If high-output pacing and low-output pacing differ in QRS morphologies, pace mapping can provide potential evidence that the ventricular myocardium at the site of pacing is responsible for the tachycardia and the discrete potential is a bystander. Conversely, if low-output pacing reproduces the clinical arrhythmia whereas capture at higher output of the surrounding myocardium does not, a “fascicular” origin for the arrhythmia can be surmised (presumably the captured arrhythmogenic fascicle exits to depolarize the myocardium with conduction delay; Figure 10).

The relationship between the discrete potentials and VT or PVC was also supported by elimination of the arrhythmia on ablation at the site of the potentials. Over long-term follow-up, the outcomes were favorable, with no clinical recurrence of the arrhythmia. Furthermore, the impact of treatment of dense PVCs in 2 patients was notable for improvement in the ejection fraction.

Although ablation above the semilunar valves was successful, it must be viewed in the context of safety. The site of origin of these discrete potentials in the aorta was often not close enough to the coronary arterial system to prohibit ablation. Nonetheless, an angiogram is required to determine the exact distance. If there is not enough distance to provide a comfortable safety margin, we report a novel means to circumvent this problem. With insertion of a Lasso catheter in the aorta, 1 patient received segmental isolation of the aorta inferior to the coronary artery ostium. This maximized the distance from the coronary artery ostium to the catheter tip and provided acceptable efficacy.

Limitations

This study data analysis was retrospective, and not all pacing and imaging information was available in each patient in whom these potentials were found. Prospective studies that use a standardized protocol in all patients undergoing outflow tract ablation are required. The prevalence of discrete potentials in patients with clinical ventricular arrhythmias or in general originating above the semilunar valves cannot be deduced from this study because a search for discrete potentials was not routinely performed in all patients during the study period. The actual cell of origin for the discrete potential is not established from this series, and further detailed investigation is required to ascertain this. Long-term follow-up in a larger group of patients will be needed to assess the safety of ablation in the great arteries.

Conclusion

Discrete potentials above the semilunar valves in the great arteries are seen in selected patients with outflow tract tachycardia. In the majority of cases reported herein, these potentials were an arrhythmogenic source for the patient’s clinical arrhythmia, and targeting these potentials, either with direct ablation or by using these potentials to guide isolation of the great arterial trunks, was useful. Nevertheless, in some cases, these potentials may result from bystander tissue getting passively activated from the ventricle. The precise mechanism of these potentials and their role in the causation of arrhythmia need to be explored further.

Disclosures

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Figure 10. The clinical PVC is displayed with a QRS obtained with both low- and high-output pacing. The origin of the PVC was ultimately located at the aortic valve. High-output pacing results in a narrower QRS than that with a shorter stim-S. In comparison, the low-output QRS morphology is wider with a slightly longer pacing stimulus–S interval. The low-output morphology is very close to that of the clinical PVC, suggestive of a fascicular origin of the discrete potential found in this region. AVR indicates augmented unipolar right arm lead; AVL, augmented unipolar left arm lead; and AVF, augmented unipolar foot lead.
Idiopathic ventricular tachycardia accounts for ~10% of all patients referred for evaluation of ventricular tachycardia. Of these ventricular tachycardias, those originating from the outflow tract are the most common. Despite being labeled outflow tract tachycardias, they have been shown to originate from many sites, including regions above the semilunar valves and within the great arteries. The identification of early discrete potentials within the great vessels is an important finding in the effort to completely map these tachycardias. In addition to the identification of these potentials, the demonstration that they are involved in the tachycardia is essential in establishing the site to ablate. A reproducible arrhythmogenic relationship rules out the possibility that these potentials may only be bystanders. The arrhythmogenic relationship was established by noting one or more of the following: A fixed or reproducibly variable pattern of discrete potential–ventricular arrhythmia relationship was present at baseline or during pacing; the discrete potential–ventricular electrogram relationship during sinus rhythm was the reverse of that during the ventricular arrhythmia; during sustained ventricular tachycardia, spontaneous variation of the interventricular cycle length was preceded by a similar variation of arterial spike potential–spike potential cycle length; and ablation guided by the discrete arterial potential successfully eliminated the clinical arrhythmia. The present study, by report of these potentials above the great vessels and their relationship with idiopathic ventricular tachycardia, should assist in the mapping and ablative treatment of these arrhythmias.
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