Characterization of Aortic Valve Closure Artifact During Outflow Tract Mapping
Correlation With Hemodynamics and Mechanical Valves

Jorge Romero, MD; Olujimi Ajijola, MD, PhD; Kalyanam Shivkumar, MD, PhD; Roderick Tung, MD

Background—Premature ventricular contractions originating in the left ventricle outflow tract represent a significant subgroup of patients referred for catheter ablation. Mechanical artifacts from aortic valve leaflet motion may be observed during mapping, although the incidence and characteristics have not been reported.

Methods and Results—Twenty-eight consecutive patients with left ventricle outflow tract premature ventricular contraction were included. Electric signals recorded on the ablation catheter not coincident with atrial or ventricular depolarization were analyzed on the recording system. Correlation with invasive hemodynamic aortic pressure tracings was performed. Additionally, 4 patients with mechanical aortic valves, who underwent scar-related ventricular tachycardia ablation, were analyzed to correlate the timing of the observed artifacts with native aortic valves. Aortic valve artifact was observed while mapping within the coronary cusps in 11 patients (39%; 73% men; age, 41±25 years; left ventricular ejection fraction 49±16%) with high incidence from the left coronary cusp. This artifact was consistently observed with timing coincident with the terminal portion of the T wave. The average interval between the end of the T wave and the aortic valve artifact was 19±37 ms. The duration of the aortic valve artifact was 39±8 ms with amplitude of 0.12±0.07 mV (range, 0.06–0.36 mV).

Conclusions—In patients referred for left ventricle outflow tract premature ventricular contraction ablation, an aortic valve closure artifact is observed in up to one third of cases during mapping within the aortic cusps. The timing of this artifact correlates with invasive hemodynamics and mechanical aortic valve artifacts. Recognition of this physiological phenomenon is useful when assigning near-field activation.

Key Words: aorta ■ aortic valve ■ arrhythmia, cardiac ■ artifact ■ catheter ablation

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WHAT IS KNOWN
- Recognition of prepotentials during activation mapping is useful to localize the site of origin of left ventricular outflow tract premature ventricular contractions.
- Catheter artifacts from mechanical motion or movements frequently occur when the catheter tip contacts valve structures and myocardial walls.

WHAT THE STUDY ADDS
- An aortic valve closure artifact is frequently observed during mapping within the aortic cusps, particularly the left coronary cusp.
- This artifact timing consistently coincides with the terminal portion of the T-wave and correlates with invasive hemodynamics and mechanical aortic valve artifacts.
- This aortic valve artifact is clinically relevant since during activation mapping this might misrepresent an early site of activation by at least 30 to 40 seconds, timing that could make the difference between a successful or unfavorable premature ventricular contraction ablation.

Results

Baseline Characteristics
Among 28 patients undergoing LVOT PVC mapping and radiofrequency ablation, 11 patients (39%; 73% men; age, 42±25 years) were observed to have an aortic valve artifact while mapping in the cusps. These patients had a mean left ventricular ejection fraction of 49±16%. PVC burden obtained by 24-hour Holter monitoring was 20±10%. In 55% of these 11 cases, previous ablation and mapping was performed at an outside institution. Remote magnetic system (Stereotaxis, St. Louis, MO) was used in 9% of the cases (Table 1). PVC morphology, axis, and R-wave precordial transition are described in Table 2. The average PVC width was 156±26 ms with a mean PVC coupling interval of 470±71 ms. The successful ablation sites were located in the left coronary cusp (LCC; n=2), junction of the right coronary cusp/LCC (n=2), aortomtral continuity (n=3) left ventricular summit (n=2), coronary venous system (n=1), and subaortic valvular region (n=1). Acute success was obtained in 91% of cases. In patient number 4, radiofrequency ablation could not be delivered because of the fact that the earliest site of activation was in the anterior interventricular vein within 5 mm of the left anterior descending coronary artery (<5 mm; Table 1). No tamponade, coronary injury, stroke, or deaths were observed.

Aortic Valve Closure Artifact
Artifact was consistently seen surrounding the terminal portion of the T wave in the surface 12-lead ECG. The duration of the aortic valve artifact was 39±8 ms with a mean amplitude of 0.12±0.07 mV (Table 3). In 4 patients (36%), this artifact was coupled just before onset of the PVC, which was coincident with early activation measurements (Figure 1). In all patients, the artifact was absent after a PVC. In patients who underwent hemodynamic correlation, the timing of the artifact consistently followed the aortic dicrotic notch, which indicates closure of the aortic valve in the cardiac cycle (Figure 2). In all patients, artifact was observed in the LCC, and only in 1 patient, it was seen in the right coronary cusp. The median interval between the end of the T wave and the aortic valve artifact was 28 ms (Q1–Q3: 2–46 ms; Table 3). Similarly, the distance between the onset of the surface QRS and this artifact was 399±44 ms. Figure 3 shows the patient with the longest intervals in this cohort, with valve closure artifact that occurs just before the local mapping electrogram. Moreover, if the QT interval (369±35 ms) is subtracted from QRS-to-artifact distance, the result would be the distance from the end of the T wave to the aortic valve closure artifact (ie, 30 ms). After correction of the QT and QRS-to-artifact intervals by the patient’s heart rate, the distance between the end of the T wave and the aortic valve closure artifact was extremely similar (ie, QRS to artifact: 467±32 and QTc=439±35 ms; Table 3). In 4 patients (36%), this artifact was coupled just before onset of the PVC, obscuring activation measurements (Figure 1A). In this cohort, the corrected QRS-to-artifact interval in this group of patients was 463±37 ms, and the average PVC coupling interval was 470±71 ms, which highlights the importance of identifying this artifact and distinguishing it from the true onset of the PVC being studied during activation mapping when mapping in the LCC.

Mapping and Ablation
Patients underwent the procedure in the fasting state under moderate sedation. Atrophicventricular nodal blockers and antiarrhythmic medications were stopped before the procedure in all cases. Mapping of the aortic root was performed by retrograde approach. Isoproterenol infusion was administered starting at 2 μg/min in cases where PVCs were infrequent or absent after conscious sedation.

After vascular access with a standard Seldinger wire technique, a quadripolar catheter was placed into the right ventricular apex. All patients had spontaneous or inducible PVCs during isoproterenol infusion. A 3.5- or 4-mm open-irrigation ablation catheter was used for mapping with the Carto electroanatomic mapping system (Navistar Thermocool; Biosense Webster, Diamond Bar, CA). Mapping in the aortic cusps was performed via the retrograde aortic approach using intracardiac echo and fluoroscopy. During aortic cusp or left ventricular endocardial mapping, intravenous heparin was given to maintain a target activated clotting time of >300 seconds. Detailed electroanatomic mapping of the coronary cusps, left ventricle, coronary sinus, and epicardium was obtained. Activation mapping was performed using a selected surface ECG lead as reference. Pace mapping was also performed for all cases to assess the morphological match at early sites of activation. In all cases, ablation was performed based on the earliest electric activation. Ablations were performed with a 3.5-mm tip-externally irrigated catheter (Navistar Thermocool; Biosense Webster) or 4-mm open-irrigated catheter (Navistar RMT Thermocool; Biosense Webster) using the Niobe II remote magnetic system (Stereotaxis, St. Louis, MO) with a maximum power of 50 W and a maximum temperature of 42°C. After ablation was performed, patients were monitored for a minimum of 30 minutes for recurrence of PVCs. The ablation was considered successful if no clinical PVCs were inducible after a waiting period of 30 minutes after isoproterenol infusion.

Statistical Analysis
Clinical characteristics were summarized as frequencies and percentages for categorical variables and by mean/SD or as median and quartiles for continuous variables. Spearman rank correlation analysis was used to correlate different variables in the study, and correlation coefficients were generated. All tests were 2-tailed, and a P value <0.05 was considered statistically significant. Analyses were performed using SAS 9.2 (SAS Institute, Inc, Cary, NC).
Correlation analysis showed that the interval from the QRS to the artifact positively correlates with the interval from the end of T wave to the artifact (correlation coefficient: 0.61; \( P = 0.04 \)). Additionally, the longer QT and R-R intervals were correlated with longer QRS-to-artifact intervals (correlation coefficient: 0.72; \( P = 0.01 \) and correlation coefficient: 0.77; \( P = 0.005 \), respectively). Finally, we found that the interval from the end of the T wave to the artifact inversely correlates to the QTc (correlation coefficient: \( r = -0.64; P = 0.03 \)).

### Mechanical Aortic Valves

A total of 4 patients with a mechanical aortic valve undergoing scar-related VT ablation were mapped for proof of concept of the artifact. All these 4 patients had a bileaflet mechanical aortic valve (St. Jude), and a higher frequency, higher amplitude artifact was observed in all cases when mapping at the LVOT close to the mechanical aortic valve. In 2 patients, a double component artifact was observed with a high-amplitude low-frequency initial component follow by a lower amplitude high-frequency component, which was interpreted a fluttering.
of the leaflet before final closure (Figure 4). The duration of the aortic valve artifact caused by a mechanical valve had a mean artifact duration of 123±28 ms, and the average artifact amplitude was 11±3 mV. The average interval between the end of the T wave and the mechanical aortic valve artifact was 6±15 ms (range, −15 to 20 ms).

### Table 3. Aortic Valve Artifact Characteristics

<table>
<thead>
<tr>
<th>Patient Number</th>
<th>Artifact Amplitude, mV</th>
<th>Artifact Duration, ms</th>
<th>QRS to Artifact, ms</th>
<th>QRS to Artifact Corrected by HR, ms</th>
<th>End T Wave to Artifact, ms</th>
<th>Corrected QRS to Artifact—QTc Interval, ms</th>
<th>Presence of Artifact in LCC</th>
<th>Presence of Artifact in RCC</th>
<th>Presence of Artifact in NCC</th>
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<tr>
<td>1</td>
<td>0.06</td>
<td>52</td>
<td>442</td>
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<td>50</td>
<td>53</td>
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<td>2</td>
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<td>46</td>
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<td>403</td>
<td>80</td>
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<tr>
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<td>14</td>
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<td>Mean</td>
<td>0.1</td>
<td>39.1</td>
<td>398.7</td>
<td>467.3</td>
<td>28*</td>
<td>29</td>
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<tr>
<td>SD</td>
<td>0.07</td>
<td>7.7</td>
<td>43.8</td>
<td>32.1</td>
<td>2–46†</td>
<td>36</td>
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</table>

LCC indicates left coronary cusp; NCC, noncoronary cusp; and RCC, right coronary cusp.

*Median.
†Q1–Q3.

**Discussion**

Idiopathic LVOT PVC represent ≤10% of all ventricular arrhythmias referred to electrophysiology laboratories. As mapping and ablation is increasingly being performed within the aortic root, methods to optimize the efficacy of this approach are necessary. As commonly observed in the

![Figure 1](http://circep.ahajournals.org/)

**Figure 1.** Example of valve closure (VC) artifact during mapping from the left coronary cusp with the latest timing in the cohort. Sample measurements from the QRS and T wave end are shown. The artifact occurs just before local activation of the premature ventricular contraction and could potentially be misinterpreted as a prepotential. A indicates atrial electrogram; ABL, ablation catheter; and V, ventricular electrogram.
cardiac catheterization laboratory when performing intra-cardiac or arterial pressure measurements, catheter artifacts from mechanical motion or movements frequently occur when the catheter tip contacts valve structures and myocardial walls. Although artifacts on the recording channel have been observed in every day practice, to our knowledge, this is the first report that systematically characterizes these aortic valve artifacts with hemodynamic and mechanical valve correlation.

Of note, the artifact was observed during mapping of the LCC in all patients. One potential explanation for a greater
frequency of this phenomenon in the LCC may be because of the orientation of the mapping catheter, which is directed anteriorly and leftward to engage the LCC. Subsequently, with every heart beat, the aortic valve opens and shifts this catheter more lateral (leftward), and when it closes, brings the catheter more medially. Early in diastole right after aortic valve closures, there is reversal of arterial flow in the ascending aorta, which builds a recoil force in the aortic cusps (dicrotic notch). Subsequently, the aortic cusps, particularly the LCC in this case, relaxes and hits (dicrotic wave) the catheter a few seconds later, which generates the artifact presented in this article (Figure 5; Movie I in the Data Supplement). On the contrary, the ablation catheter has minimal flexion when mapping the right coronary cusp, and this leaflet does not significantly move it. Our theory of the mechanism of this artifact is similar to the one described by Sabbah and Stein for the origin of the second heart sound. Despite the fact that the second heart sound is thought to be related to the closure of the semilunar valves, they published an elegant study in 1976, demonstrating that the aortic heart sound originates from an initial differential pressure between the aorta and the left ventricle, which stretches the semilunar valve after closure. As a result of this stretch and the following valvular recoil, expansion, and compression of blood, the sound is created.

In this study, we demonstrate that aortic valve closure artifact was independent from atrial and ventricular electrograms and was absent after early coupled PVCs, as reduced stroke volume results in less valve excursion to produce the artifact. The reproducible artifact observed in control patients with mechanical aortic valves provides evidence for the present findings. The artifact has similar timing but greater amplitude and duration as expected because most mechanical valves are made from pyrolytic carbon (Figure 5). The use of arterial pressure measurements allows for the correlation of this phenomenon with the dicrotic notch. Just as the ventricles enter into diastole, the brief reversal of flow from the aorta back toward the left ventricle causes the aortic valves to shut. This results in a slight increase in aortic pressure caused by elastic recoil of the aortic and semilunar valves, which may be the primary mechanism of the closure artifact. This mechanistic hypothesis is supported by the fact that the mechanical valve artifact exactly corresponds to the actual closure of the aortic valve leaflets because this artifact is observed earlier right after the dicrotic aortic notch (6 ms after T wave) as compared with the artifact in native aortic valves, which is somewhat later right after the dicrotic aortic wave (19 ms after T wave). This aortic valve artifact is clinically relevant in some cases of LVOT ablation primarily because of the fact that during activation mapping this might misrepresent an early site of activation by at least 30 to 40 seconds, time that makes the difference between a successful or unfavorable PVC ablation. In 4 cases in this cohort, the closure artifact was initially misinterpreted as local pre-potential activation in real-time. Ablation was not delivered after careful review of previous beats and these observed phenomenon prompted the present systematic analysis. If this artifact is unrecognized without careful analysis of preceding sinus rhythm beats (at fast sweep speeds [200 ms]), radiofrequency ablation might be delivered at sites unnecessarily, exposing the patient to unnecessary ablation and the risks associated with it in the aortic sinuses. The duration of the aortic valve artifact in this study was 39±8 ms with an amplitude of 0.12±0.07 mV (range, 0.06–0.36 mV). The real local electrogram of the PVC is much larger than the artifact with a duration of 118 to 372 ms and with an amplitude ranging from 0.4 to 1.1 mV. Even though they are significantly different, the artifact electrogram usually precedes the PVC local electrogram and can be taken as
part of the latter and create inaccurate activation measurements during LVOT mapping.

**Limitations**
The study is limited by small sample size from a single institution. We acknowledge that the observance of this artifact is likely a function of catheter orientation and duration spent mapping within a given cusp, which is operator dependent. Hemodynamic correlation was not performed in all cases as angiography of a catheter location in the aortic root requires double arterial access.

**Conclusions**
In patients referred for LVOT PVC ablation, an aortic valve closure artifact is observed in up to one third of cases during mapping within the aortic cusps. The timing of this artifact correlates with invasive hemodynamics and mechanical aortic valve artifacts. Recognition of this physiological phenomenon may be clinically useful when assigning near-field activation during mapping within the aortic root.

**Disclosures**
None.
References


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SUPPLEMENTAL MATERIAL

**VIDEO 1.** Intracardiac echocardiography illustrating mapping catheter movement in the LCC.