



New electrocardiographic criteria for predicting successful ablation of premature ventricular contractions from the right coronary cusp



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ABSTRACT

Background: ECG features for predicting successful ablation sites of outflow tract (OT) premature ventricular complex (PVCs) have been previously presented, but effective predictors of right coronary cusp (RCC) remain elusive.

Methods: 106 patients (59 males, 56 ± 14 years) who underwent successful PVC ablation were studied. Various ECG patterns and measurements were analyzed to identify the unique features of RCC PVC origins. The R-wave duration index (RWDI) was calculated as a percentage by dividing the QRS complex duration by the longest R-wave duration in lead V_1 or V_2 .

Results: Successful ablation sites were the RCC in 18 patients, the left coronary cusp (LCC) in 20, the RCC/LCC junction (RLJ) in 22, the AIV/GCV in 11 and the right ventricular outflow tract in 35. Forty-seven patients had dominantly positive forces in lead I. Among these 47 patients, 19 were ablated from the RCC (18/18, 100%), eighteen from the RVOT (18/35, 51%), five from the LCC (5/20, 25%), and five from the RLJ (6/22, 27%). The S-wave amplitude in lead aV_L was significantly smaller in RCC than LCC or RLJ PVCs (0.1 ± 0.3 mV vs. 1.1 ± 0.5 mV, $p < 0.001$). The V_{1-2} RWDI was significantly greater in RCC than RVOT PVCs ($51.8 \pm 20.5\%$ vs. $30.8 \pm 13.9\%$, $p < 0.001$). The optimal cut-off values of <0.95 mV for S-wave (area under the curve, AUC: 0.76, $p < 0.01$) and $>43.6\%$ for R-wave duration index in V_1 or V_2 (AUC: 0.83, $p < 0.001$) were determined by ROC analysis.

Conclusions: The presence of a dominant positive lead I, RWDI $>43.6\%$ and S-wave amplitude in $aV_L <0.95$ mV predicted RCC PVCs with a sensitivity of 83% and specificity of 94%.

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1. Introduction

Most idiopathic premature ventricular contractions (PVCs) originate from the outflow tract sites of both ventricles. Several electrocardiogram (ECG) characteristics of PVCs originating from the aortic sinuses of Valsalva (ASOV), left coronary cusp (LCC), right coronary cusp (RCC), RCC/LCC commissural junction (RLJ), and epicardial sites have been reported [1–9]. Ventricular arrhythmias (VAs) arising from the ASOV account for up to 21% of idiopathic VAs [10]. These arrhythmias

commonly arise from the LCC and RCC, and rarely arise from the non-coronary cusp (NCC) [1,2]. This arrhythmogenicity is thought to be related to the myocardial extensions projecting above the aortic valve into the ASOV. Ouyang et al. [1] explained that PVCs from the ASOV have a QRS morphology similar to those arising from the right ventricular outflow tract (RVOT), and suggested that the R-wave duration and R/S-wave amplitude indices in leads V_1 and V_2 can be used to differentiate these two sites of origin. Our group previously described a QR or Qr in V_1 , small broad R-wave in V_2 and variable precordial transition as typical features of PVCs originating from the RCC region on 12-lead ECG using intracardiac echocardiography (ICE) and electroanatomic mapping. Differentiating RCC PVCs from LCC and RVOT PVCs is difficult due to their close anatomic relationship [3]. Useful ECG criteria for differentiating RVOT and left ventricular (LV) OT sites of origin have also been described [11,12]. Recently, Betensky et al. reported a useful new ECG criterion, the V_2 transition ratio, for distinguishing LVOT from RVOT PVCs [13]. However, differentiating RCC and septal RVOT (sRVOT) PVCs remains a challenge, despite the use of these criteria. We performed systematic electrocardiographic analysis of PVCs originating from the outflow tract area in order to describe features specifically identifying the site of origin of RCC PVCs.

Abbreviations: PVC, premature ventricular contraction; RVOT, right ventricular outflow tract; LVOT, left ventricular outflow tract; RCC, right coronary cusp; LCC, left coronary cusp; RLJ, RCC/LCC junction; AIV/GCV, anterior interventricular vein/great cardiac vein; ASOV, aortic sinuses of Valsalva; RWDI, R-wave duration index; MDI, maximum deflection index.

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2. Methods

2.1. Patient population

We identified all patients who underwent successful ablation of PVCs, at the Samsung Medical Center in Korea, between January 1st, 2008 and December 31st, 2014. We analyzed all patients who underwent successful OT (outflow tract) PVC ablation from the septal RVOT, ASOV, or great cardiac vein (GCV)/anterior interventricular vein (AIV) region. All had frequent PVCs, defined as >5000 PVCs/24 h on a Holter monitor. Successful ablation was defined as at least an 80% reduction in the 24-hour burden of PVCs based on our previously published experience [14]. Patients with active ischemia or prior infarction as a cause of cardiomyopathy were excluded based upon history and either coronary angiography or stress testing. Structurally normal hearts were defined by a normal nuclear perfusion scan, normal transthoracic echocardiogram (TTE), and/or normal coronary angiography. Cardiac catheterization was performed due to equivocal perfusion scans in four patients and resulted in normal coronary angiography. Informed consent was obtained from each patient and the study protocol conforms to the ethical guidelines of the 1975 Declaration of Helsinki as reflected in a priori approval by the institution's human research committee [15].

2.2. Mapping and RF catheter ablation

The RVOT was initially mapped with a 7Fr, 4-mm-tip NaviStar catheter placed through the right femoral vein. Detailed activation and pace mapping were performed in the RVOT, and sinus rhythm electrograms were analyzed. If a diffuse activation pattern was noted on the three-dimensional electroanatomic mapping system (CARTO, Biosense Webster) and/or pace mapping resulted in a poor pace-map match, attention was turned to the LVOT region. The NaviStar catheter was inserted through the right femoral artery and advanced to the aortic cusp region in a retrograde fashion. Intravenous heparin was administered to maintain an activated clotting time >250 s during aortic cusp and/or LV endocardial mapping. An electroanatomic shell of the aortic cusp region was created. The mapping catheter was located on ICE. Pace mapping was performed at a threshold output from multiple sites in each cusp and from the RLJ. The location of each pacing site was tagged on the electroanatomic map. Detailed activation mapping during PVCs was performed in the aortic cusp region, and the earliest area of activation was marked on the electroanatomic map and confirmed on ICE. RF ablation was first performed with a 4-mm, standard, non-irrigated catheter. An irrigated catheter was only used if inadequate power delivery occurred with a standard 4-mm-tip catheter. The maximum power delivery was 30 W with the irrigated ablation catheter. Successful catheter ablation was defined as the absence of spontaneous or inducible PVCs with an isoproterenol infusion (2–20 µg/min) and burst pacing from the RV apex or high right atrium for 30 to 60 min after ablation of the final lesion.

2.3. Comparison of PVC morphology

The site of origin of the PVCs was determined based on activation mapping and successful elimination with RF energy application. The anatomic position of the catheter tip at the site of origin was evaluated using a combination of ICE imaging, electroanatomic mapping data, and orthogonal fluoroscopy (Fig. 1A). The site of origins were classified as RCC, LCC, RLJ, AIV/GCV, antero-septal RVOT (sRVOT-a), and postero-septal RVOT (sRVOT-p) based on both the site-specific pace-mapping and the anatomic definition of the successful catheter ablation site (Fig. 1B).

2.4. ECG measurements (Fig. 2)

The sinus rhythm and PVC ECG morphologies were measured using the same 12-lead ECG with electronic calipers on the Prucka CardioLab® recording system (version 6.5.4.1858, GE Medical Systems). The lead gain was uniform with a paper speed of 100 mm/s. During spontaneous clinical PVCs, the following measurements were obtained: 1) total QRS duration (ms); 2) lead I vector and S-wave amplitude (mV); 3) lead II and III R-wave amplitude (mV) and lead II/III ratio; 4) lead a_VL and a_VR S-wave amplitude (mV) and a_VL/a_VR ratio; 5) R-wave duration (RWD, ms) in leads V₁ and V₂; 6) R-wave duration index (RWDI) in lead V₁ or V₂; 7) R/S-wave amplitude ratio (RSWAR, %) in leads V₁ and V₂; 8) R/S-wave amplitude index (RSWAI) in lead V₁ or V₂; 9) maximum deflection index (MDI); and 9) V₂ transition ratio. Finally, the global ECG patterns, including the bundle branch block pattern and precordial R-wave transition during the PVCs and normal sinus beats, were examined.

The QRS duration was measured from the site of earliest initial deflection at the isoelectric line in any lead to the time of last activation in any lead. The RWD in leads V₁ and V₂ was measured from the onset of the QRS complex to the transition point between the R-wave and the isoelectric line. The RWDI was calculated as a percentage by dividing the QRS complex duration by the longest R-wave duration in lead V₁ or V₂. The R/S-wave amplitude ratio in leads V₁ and V₂ was measured from the QRS complex peak or nadir to the isoelectric line and expressed as a percentage, and the R/S-wave amplitude index was calculated from the greater percentage R/S-wave amplitude ratio in lead V₁ or V₂ [1]. The MDI was calculated by dividing the shortest time to maximum deflection in any precordial lead based on the QRS duration [5]. The V₂ transition ratio was calculated in lead V₂ by computing the percentage of the R-wave during the PVCs (R-wave / R-wave + S-wave) divided by the percentage of the R-wave in normal sinus rhythm (NSR) (R-wave / R-wave + S-wave) in all patients without considering the precordial transition during the PVCs or the bundle branch block (BBB) pattern during sinus rhythm. In this study, we measured the V₂ transition ratio without considering the respiratory variation in the R- and S-waves during either sinus rhythm or the PVCs, as described in the original paper [13]. All ECG measurements were reviewed by one of two authors (KMP or SII), who were blinded to patient outcomes. Assessment of inter-observer agreement for these measurements was performed on a subset of 20. All ECG measurements were repeated five times. The mean of the five measurements was used for analysis to minimize the influence of measurement error.

2.5. Follow-up

The majority of patients were followed in our Arrhythmia Center with routine ECGs at six weeks and with follow-up echocardiograms four to 12 months after ablation. For patients not followed at the Samsung Medical Center, the referring cardiologists were contacted, and the medical records were reviewed. All patients had a follow-up echocardiogram three to 12 months post-ablation. Arrhythmia burden was assessed based upon symptoms, ECG and Holter monitoring.

2.6. Statistical analysis

Pearson's product moment correlation coefficient was calculated to quantify inter-rater variability of the ECG measurements. Descriptive data were presented as means ± SDs or medians (ranges). Between-group comparisons were made using the Student's t-test, whereas within-group comparisons were achieved using paired t-tests. Categorical variables expressed as numbers and percentages in the different groups were compared via the Chi-square test. The characteristics of the RCC, LCC, RLJ,

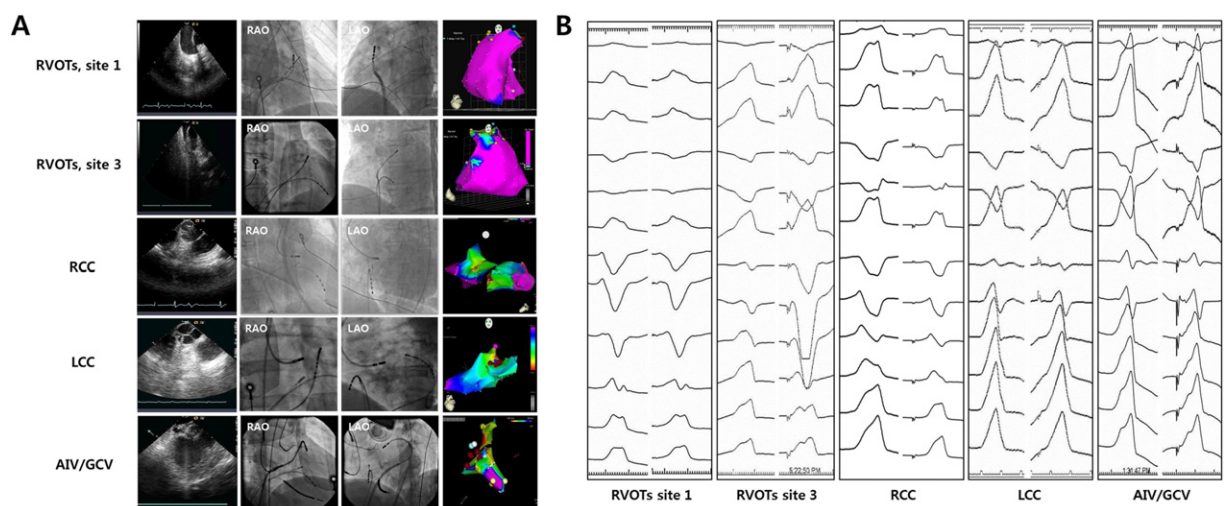


Fig. 1. A. Images of electro-anatomical mapping, intra-cardiac echo, and fluoroscopy along with the morphology of each PVC. B. Comparison of PVC morphologies in the 5 districts and the site-specific pace mapping on successful ablation sites.

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