Electrocardiographic Comparison of Ventricular Arrhythmias in Patients With Arrhythmogenic Right Ventricular Cardiomyopathy and Right Ventricular Outflow Tract Tachycardia

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Objectives
The purpose of this study was to determine whether electrocardiographic characteristics of ventricular arrhythmias distinguish patients with arrhythmogenic right ventricular dysplasia cardiomyopathy (ARVD/C) from those with right ventricular outflow tract tachycardia (RVOT-VT).

Background
Ventricular arrhythmias in RVOT-VT and ARVD/C-VT patients can share a left bundle branch block/inferior axis morphology.

Methods
We compared the electrocardiographic morphology of ventricular tachycardia or premature ventricular contractions with left bundle branch block/inferior axis pattern in 16 ARVD/C patients with that in 42 RVOT-VT patients.

Results
ARVD/C patients had a significantly longer mean QRS duration in lead I (150 ± 31 ms vs. 123 ± 34 ms, p = 0.006), more often exhibited a precordial transition in lead V6 (3 of 17 [18%] vs. 0 of 42 [0%] with RVOT-VT, p = 0.005), and more often had at least 1 lead with notching (11 of 17 [65%] vs. 9 of 42 [21%], p = 0.001). The most sensitive characteristics for the detection of ARVD/C were a QRS duration in lead I of ≥120 ms (88% sensitivity, 91% negative predictive value); QRS transition at V6 was most specific at 100% (100% positive predictive value, 77% negative predictive value). The presence of notching on any QRS complex had 79% sensitivity and 65% specificity of (55% positive predictive value, 85% negative predictive value). In multivariate analysis, QRS duration in lead I of ≥120 ms (odds ratio [OR]: 20.4, p = 0.034), earliest onset QRS in lead V6 (OR: 17.0, p = 0.022), QRS notching (OR: 7.7, p = 0.018), and a transition of V5 or later (OR: 7.0, p = 0.030) each predicted the presence of ARVD/C.

Conclusions
Several electrocardiographic criteria can help distinguish right ventricular outflow tract arrhythmias originating from ARVD/C compared with RVOT-VT patients.

Arrhythmogenic right ventricular dysplasia cardiomyopathy (ARVD/C) is an inherited disease characterized by a progressive replacement of myocytes of the right ventricle with fibrous and fatty tissue (1,2). The clinical spectrum is diverse, and ventricular tachycardia (VT) and sudden cardiac death are hallmarks of the disease (1–4).

In contrast, right ventricular outflow tract tachycardia (RVOT-VT) is the most common cause of idiopathic VT and is a relatively benign condition. Treatment with ablation is frequently recommended, with a success rate >90% (5,6). VTs from this region also share a left bundle branch block (LBBB) QRS morphology/inferior axis pattern, and differentiation between these 2 distinct disease states is paramount. The presence of T-wave inversion in V1 to V3 in normal sinus rhythm may aid the diagnosis of ARVD/C, but recent data showed that these changes may be present in only 32% of ARVD/C patients as well as 1% to 3% of normal young patients (7–10). Similarly, other popular noninvasive studies show a 50% to 60% false-negative rate (echocardiography), and a 70% false-positive rate (magnetic resonance imaging [MRI]) for the diagnosis of ARVD/C (10,11). It is therefore important for the clinician to distinguish ventricular arrhythmias of an LBBB QRS morphology/inferior axis RVOT ventricular arrhythmia from those that occur due to ARVD/C.

The purpose of our study was to determine whether surface electrocardiographic characteristics could distinguish patients with RVOT-VT from patients with ARVD/C-VT with an LBBB and an inferior QRS axis pattern by analysis of electrocardiographic characteristics.

Methods
Patients. Electrocardiograms (ECGs) from patients with ARVD/C were selected from 2 cohorts: 13 patients from the North American ARVD Registry with confirmed ARVD/C by the core laboratory and 3 patients from the Madras Medical Mission, Chennai, Tamil Nadu, India. All patients had confirmed ARVD/C based on the revised task force criteria (10,12). Only patients presenting with a ventricular arrhythmia characterized by an LBBB pattern and inferior axis were included. Electrocardiographic tracings were excluded if they were of poor quality, if they displayed fewer than 10 leads, or if VT was too rapid (preceding T-wave interfering with subsequent QRS) to allow precise measurements of the QRS onset. Because ARVD/C patients may have several morphologies of VT or premature ventricular contractions (PVCs), we analyzed each morphology as a distinct data entry. One of the ARVD/C patients had 2 distinct morphologies. Thus, there were 17 different data entries for ARVD/C patients.

The RVOT-VT cohort consisted of 42 consecutive patients with RVOT-VT successfully treated with ablation. All patients with RVOT-VT had structurally normal hearts as assessed by physical examination and
Echocardiography (and/or MRI). Patients with a diagnosis of RVOT-VT were included if they had at least one 12-lead electrocardiographic tracing with spontaneous PVCs or VT.

**Electrocardiographic analysis.** We used ECGs that were recorded with at least 10 simultaneous leads. The amplitudes were 1 mV/cm. For digital analysis, hard copies of the ECGs were scanned on a flat bed scanner at a resolution of 600 dpi and processed in Adobe Photoshop CS version 8 (Adobe Systems Incorporated, San Jose, California) by changing the size to 200 pixels/cm, rendering the ECGs to 500 pixels/r if they were originally recorded at 25 mm/s. Two investigators made measurements blinded to the diagnosis. The time difference from the lead with earliest QRS onset to the onsets for each lead was measured. If more than 1 lead shared a simultaneous onset with the lead determined to be earliest, they were all determined to be of earliest onset. We also measured earliest onset of QRS to initial peak/nadir of each lead and QRS duration for each lead (Fig. 1). We measured QRS notching (a deflection of $\geq 0.5$ mV on the QRS that did not cross baseline) (Fig. 2) and the precordial R-to-S wave transition, defined as the precordial lead where R-wave amplitude was equal to or greater than the S-wave amplitude.

**Data analysis.** Continuous variables are expressed as mean ± SD if normally distributed and median and interquartile range if not normally distributed. The Wilcoxon rank sum test was used to compare continuous variables. Categorical variables were compared using the chi-square test statistic. We assessed interobserver agreement for binary outcomes using the kappa statistic and for continuous outcomes using intraclass correlation. Standard definitions were used for testing characteristics including sensitivity, specificity, positive predictive value, and negative predictive value (13). Multivariable logistic regression was used to identify a subset of electrocardiographic predictors best able to discriminate ARVD/C ventricular arrhythmias from RVOT. For electrocardiographic predictors that examined the same measurement in all 12 leads, a Bonferroni correction was performed to address multiple hypothesis testing (a p value cutoff of $< 0.004$ was therefore considered statistically significant for these univariate analyses). For individual electrocardiographic characteristics with a priori evidence suggesting utility as a predictor (lead I QRS duration $\geq 120$ ms [14]), a 2-tailed p value $< 0.05$ was considered statistically significant in univariate analysis. Beginning with 6 predictors with p $< 0.05$ in single-predictor models, we screened models with 2-, 3-, and 4-predictor models using 5-fold cross-validation of the C-statistic, a measure of discrimination. STATA version 10.0 (StataCorp, College Station, Texas) was used for all statistical analyses.

**Results**

A total of 59 ECGs demonstrating ventricular arrhythmias were analyzed. Seventeen were from the ARVD/C cohort. Eleven of the 17 ECGs (65%) had revealed VT, and 6 of the 17 ECGs (35%) showed PVCs. Forty-two ECGs were from RVOT-VT patients, and 7 of the 42 ECGs (17%) of the RVOT-VT group showed VT, whereas the other 35 ECGs (83%) showed PVCs. The baseline noninvasive imaging characteristics and presence of T-wave inversions of the ARVD/C cohort are shown in Table 1. Only 4 of the 16 patients (25%) had transthoracic echocardiographic findings consistent with ARVD/C compared with 12 of 16 MRI scans, computed tomography scans, or right ventricular angiograms (75%) showing right ventricular changes consistent with ARVD/C. The diagnosis of the other patients included a strong family history (such as autopsy confirmation) together with abnormalities on the ECG and/or positive gene mutations associated with ARVD/C.

The mean and SD duration of the QRS complex in lead I was greater in ARVD/C patients than in those with RVOT-VT ($150 \pm 31$ ms vs. $123 \pm 34$ ms, p = 0.006) (Fig. 3). We did not find any significant difference between ARVD/C and RVOT-VT ECGs in QRS durations in other leads or the earliest onset of QRS to peak/nadir measurements.

Notching in at least 1 lead was more often observed in ARVD/C ECGs compared with RVOT-VT ECGs (11 of 17 [65%] vs. 9 of 42 [21%], p = 0.001). We also found a significant preponderance of notching during the upstroke of the QRS complex in ARVD/C ECGs compared with RVOT-VT ECGs (8 of 17 [47%] vs. 5 of 42 [12%], p = 0.003). The presence of multiple notches across several leads was seen more often in the ARVD/C ECGs compared with RVOT-VT ECGs (6 of 17 [35%] vs. 5 of 42 [12%], p = 0.04).

Precordial transition at $V_6$ or later was observed in 3 of 17 patients (18%) with ARVD/C and in none of the 42 patients with RVOT-VT (p < 0.005). If $V_5$ was used for transition, 8 of 17 of the ARVD/C cohort (47%) had a transition at $V_5$ or later compared with only 4 of 42 patients with RVOT-VT (9.5%) (p = 0.001).

The most sensitive test was duration of QRS in lead I of $\geq 120$ ms (88%). The QRS transition at $V_6$ was the most specific (100%). The individual test characteristics are shown in Table 2.

The interobserver agreement between the 2 ECG readers showed an intraclass coefficient with regard to the QRS onset to $V_6$ measurement of 0.60 (95% confidence interval: 0.43 to 0.77) and a kappa value of 0.74 for the assessment of QRS notching.
Using these electrocardiographic test characteristics, we used univariate and multivariate logistic regression to determine which variables were independent predictors of ARVD/C. Because transition at V6 was had specificity of 100% for ARVD/C, this variable fell out of the model. For multivariate analysis, duration of QRS in lead I < 120 ms (odds ratio [OR]: 20.4, p = 0.034), earliest onset QRS on lead V1 (OR: 17.0, p = 0.022), QRS notching (OR: 7.7, p = 0.018), and transition V5 or later (OR: 7.0, p = 0.030) were all statistically significant. The specific ORs, 95% confidence intervals, and p values are shown in Table 3. A 5-fold cross-validation of the C-statistic, a measure of discrimination, was 0.49 to 0.64 for the single-predictor models and 0.85 for the multivariate model.

Using electrocardiographic criteria to distinguish the site of origin of VT/PVCs previously published by Dixit et al. (15) as well as the observed successful site of ablation for the RVOT-VT group, we showed that ARVD/C patients are more likely to have a free wall (nonseptal) location (70.6% vs. 16.7%, p < 0.001) (Table 4) and the location of the free-wall site was mostly anterior (52.9% vs. 9.5%, p < 0.001). RVOT-VT patients were more likely to have septal location (83.3% vs. 29.4%, p < 0.001).

**Discussion**

It is important to distinguish ventricular arrhythmias of an LBBB QRS morphology/inferior axis ARVD/C ventricular

![Figure 1](image1.png)

*Figure 1 Examples of Measurements*

Simultaneous recordings of a 12-lead electrocardiogram in a patient with right ventricular outflow tract ventricular tachycardia demonstrating an example of our measurements. The initial onset was in lead V6 (arrow), and it was used as a baseline (long vertical line) for our measurement of initial onset to local onset, where local onsets are marked as small vertical lines in the other leads in the left column. The duration was 66 ms in lead I, 13 ms in lead II, and 13 ms in lead III. In the middle column, we measured the initial onset to peak/nadir in each lead. This duration was 97 ms in aVR and 94 ms in aVL. In the right column, we show examples of measurements of QRS duration. QRS duration in lead I measured 58 and 129 ms in V6.

![Figure 2](image2.png)

*Figure 2 Examples of Notching*

Example of multiple notching (arrows) during ventricular tachycardia in a patient with arrhythmogenic right ventricular dysplasia/cardiomyopathy.
arrhythmia from those that occur due to RVOT-VT to assist in differentiating these 2 conditions. To our knowledge, this report consists of the largest cohort of patients with ARVD/C with ventricular arrhythmias consisting of an LBBB with an inferior axis morphology. In this study, we found that certain features of the QRS complex during ventricular arrhythmias could assist in distinguishing ARVD/C from idiopathic RVOT-VT. This is based on several findings, such as a precordial transition at V5 or later, the presence of notching on the QRS complex, and a QRS duration in lead I of $\leq 120$ ms. The most specific findings were late precordial transition (100% specificity at V6 and 90% specificity at V5 or later), and the most sensitive was duration of QRS in lead I of $\leq 120$ ms, followed by the presence of any notching on the QRS complex (Fig. 4).

The differences observed in the QRS complexes in our ARVD/C study population compared with our RVOT-VT population may be due to 2 factors. The most important is the replacement of normal right ventricular myocardial tissue with fibrous and fatty tissue, which may delay cell-to-cell conduction and facilitate the development of re-entrant ventricular arrhythmias (16). We hypothesize that this results in a greater delay from earliest onset to local onset of the QRS complex, greater duration of the QRS complex, and irregularities of conduction manifest as notching of the QRS complex. Another factor may be related to a greater frequency of the site of origin of the right ventricular free wall for patients with ARVD/C sites more remote from the normal His-Purkinje conduction tissue than in those patients with RVOT-VT. These factors also explain the late precordial transition and possibly notching of the QRS. A number of studies have found that VT

<table>
<thead>
<tr>
<th>Patient #</th>
<th>Echocardiogram (Transthoracic)</th>
<th>Magnetic Resonance Imaging</th>
<th>T-Wave Inversions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Right atrial enlargement</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Within normal limits</td>
<td>V2-V3</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Mild biatrial enlargement</td>
<td>V2-V3</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Within normal limits</td>
<td>V2-V3</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Severe right ventricular dilation, left ventricular dysfunction</td>
<td>V2-V3</td>
<td>None</td>
</tr>
<tr>
<td>6</td>
<td>Within normal limits</td>
<td>Distal septum fibrosis and thinning, no fatty tissue, enlarged right ventricle with dyskinesia</td>
<td>V2-V4</td>
</tr>
<tr>
<td>7</td>
<td>Within normal limits</td>
<td>Right ventricular wall thinning and trabeculation, no fatty infiltration</td>
<td>None</td>
</tr>
<tr>
<td>8</td>
<td>Within normal limits</td>
<td>V2-V3</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>Within normal limits</td>
<td>Focal thinning of the free wall of the right ventricle with associated hypokinesia; dilation of the right atrium and right ventricular outflow tract with mild dilation of the right ventricle.</td>
<td>V2-V6, II, III, aVF</td>
</tr>
<tr>
<td>10</td>
<td>Moderate right ventricular dilation with moderate right ventricular dysfunction</td>
<td>Right ventricular wall thinning with areas of wall thinning; findings compatible with right ventricular aneurysms; transmural right ventricular fat in free wall with focal thinning; hypokinesia and akinesia of the right ventricle.</td>
<td>V2-V5</td>
</tr>
<tr>
<td>11</td>
<td>Severe left atrial enlargement</td>
<td>No fatty infiltrates, right ventricular apex dyskinesia and right ventricular apical aneurysm</td>
<td>V2-V4</td>
</tr>
<tr>
<td>12</td>
<td>Within normal limits</td>
<td>Severe right ventricular dilation, wall thinning, and hypokinesia with dysplasia</td>
<td>None</td>
</tr>
<tr>
<td>13</td>
<td>Within normal limits</td>
<td>Within normal limits* (computed tomography)</td>
<td>V2-V5, II, III, aVF</td>
</tr>
<tr>
<td>14</td>
<td>Within normal limits</td>
<td>Not available†</td>
<td>V2-V4</td>
</tr>
<tr>
<td>15</td>
<td>Dilated right ventricular, moderate dysfunction</td>
<td>Not available†</td>
<td>V2-V4</td>
</tr>
<tr>
<td>16</td>
<td>Biventricular dysfunction, right ventricular enlargement</td>
<td>Not available†</td>
<td>V2-V6</td>
</tr>
</tbody>
</table>

*Patient underwent computed tomography, not magnetic resonance imaging. †Patients 14 through 16 had right ventricular angiograms performed that were consistent with ARVD/C by modified task force criteria (12). All other patients (1 to 13) were from the ARVD registry with confirmed diagnosis made by the core laboratory.

ARVD/C = arrhythmogenic right ventricular dysplasia cardiomyopathy.

**Figure 3**

Boxplot showing the duration of lead I QRS in right ventricular outflow tract tachycardia (RVOT-VT) versus arrhythmogenic right ventricular dysplasia cardiomyopathy (ARVD/C). The mean duration of the QRS complex in lead I was greater in ARVD/C patients than those with RVOT-VT (150 ± 31 ms vs. 123 ± 34 ms, p = 0.006). The middle line represents the median (50th percentile), each box boundary represents the 25th to 75th percentiles (interquartile range).
Our study, we were able to confirm that the duration of the QRS in lead I was helpful in distinguishing between the 2 groups. Other discriminators were QRS notching and precordial transition at V5 or later.

Multiple criteria are required to diagnose ARVD/C. These include characteristic electrocardiographic as well as morphological criteria (10,12,17). Our observations suggest that electrocardiographic parameters may help guide the clinician to the proper diagnosis. In addition, it is clear that the initial echocardiographic evaluation (even when using the recent revised suggestive criteria [18]) was very limited in supporting the diagnosis of ARVD/C. The echocardiogram was normal or equivocal in 12 of 16 patients (75%). Of interest, the finding that T-wave inversions V1 to V3 of the 12-lead ECG in sinus rhythm was found in 12 of 16 patients (75%), suggesting that both the electrocardiographic findings described for VT/PVCs as well as the surface ECG can be used for useful noninvasive screening.

Study limitations. The sample size is relatively small because this is a rare disease and we studied a subset of patients with LBBB/inferior axis. To increase the sample size for a more robust analysis, we included patients with both VT and PVCs. Ainsworth et al. (14) analyzed a subset of patients with PVCs and found identical findings compared with those with VT. They concluded that measuring PVC duration might represent a practical method of differentiating the 2 entities and warrants future study. Because our sample size is relatively small, there are limitations to our multivariate analysis for electrocardiographic predictors of ARVD/C, and the model is prone to overfitting. We also

| Table 2 | Test Characteristics for Identifying ARVD/C |
|------------------------------------------------|
| Test Algorithm | Specificity, % | Sensitivity, % | Positive Predictive Value, % | Negative Predictive Value, % |
| Duration of QRS in lead I of ≥120 ms | 48 | 88 | 41 | 91 |
| Notching on QRS complex | 79 | 65 | 55 | 85 |
| Notching on upstroke of QRS | 88 | 47 | 62 | 80 |
| Multiple notching of the QRS across several leads | 88 | 35 | 55 | 77 |
| Earliest onset of QRS on lead V1 | 90 | 35 | 60 | 77 |
| Transition at V5 or later | 100 | 18 | 100 | 77 |
| Transition at V6 or later | 90 | 47 | 67 | 81 |

ARVD/C = arrhythmogenic right ventricular dysplasia/cardiomyopathy.
recognize that the 3 covariates related to QRS notching are at least in part colinear and that may be the main reason that statistical significance for each is lost after multivariable adjustment. We realize that having 4 covariates violates the rule of 1 covariate per 10 outcomes, although a recent paper suggests that this criterion may be too stringent (19); however, we acknowledge that our ratio is quite small. However, ARVD/C is a rare disease, and enrolling the necessary number to comply with this rule is not feasible. In addition, the C-statistic, a measure of discrimination, for the 4-variable multivariate model is 0.85 compared with 0.49 to 0.64 for the univariate models, signifying an improved prediction of the multivariate model. Although our findings are statistically significant, many of the 95% confidence intervals are wide. However, even in the setting of these wide confidence intervals and even if the lowest value is used, the odds of predicting ARVD/C are still clinically useful.

For example, the odds of having ARVD/C are at least 38% greater if the duration of QRS in lead I is at least 120 ms (lower limit of 95% confidence interval OR: 1.38, p = 0.034).

The methodology used in our report may be challenging for routine application for some clinicians, but the measurements can be done on any software system that has image editing software with an appropriate selection tool. In addition, the VT/PVC Anatomic Localization by Electrocardiography table provides a detailed analysis of the localization of arrhythmogenic right ventricular dysplasia/cardiomyopathy arrhythmias.

### Table 4 VT/PVC Anatomic Localization by Electrocardiography

<table>
<thead>
<tr>
<th>Location</th>
<th>ARVD/C (n = 17)</th>
<th>RVOT-VT (n = 42)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Free wall (total)</td>
<td>12 (70.6)</td>
<td>7 (16.7)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Anterior</td>
<td>9 (52.9)</td>
<td>4 (9.5)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Posterior</td>
<td>2 (11.8)</td>
<td>2 (4.8)</td>
<td>0.333</td>
</tr>
<tr>
<td>Inflow tract/His bundle region</td>
<td>1 (5.9)</td>
<td>1 (2.4)</td>
<td>0.501</td>
</tr>
<tr>
<td>Septal (total)</td>
<td>5 (29.4)</td>
<td>35 (83.3)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Anterior septal</td>
<td>4 (23.5)</td>
<td>16 (38.1)</td>
<td>0.128</td>
</tr>
<tr>
<td>Mid septal</td>
<td>0</td>
<td>9 (21.4)</td>
<td>0.038</td>
</tr>
<tr>
<td>Posterior septal</td>
<td>1 (5.9)</td>
<td>10 (23.8)</td>
<td>0.109</td>
</tr>
</tbody>
</table>

Values are n (%). Data from Dixit et al. (15).

ARVD/C = arrhythmogenic right ventricular dysplasia/cardiomyopathy; PVC = premature ventricular contraction; RVOT-VT = right ventricular outflow tract ventricular tachycardia; VT = ventricular tachycardia.

Values are n (%). Data from Dixit et al. (15).

ARVD/C = arrhythmogenic right ventricular dysplasia/cardiomyopathy; PVC = premature ventricular contraction; RVOT-VT = right ventricular outflow tract ventricular tachycardia; VT = ventricular tachycardia.

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**Figure 4 Characteristic Features**

Twelve-lead electrocardiograms from patients with right ventricular outflow tract tachycardia (RVOT-VT) (A to C) and arrhythmogenic right ventricular dysplasia/cardiomyopathy (ARVD/C) (D to H) showing characteristic features. (A) RVOT-VT from an anterior-septal location showing precordial transition at V2 and narrow QRS duration in lead I (78 ms). (B) RVOT-VT originating superior to His bundle region showing precordial transition at V4, positive R-wave in aVL, and narrow QRS in lead I (86 ms). (C) RVOT-VT from a posterior-septal location showing precordial transition at V3, wide QRS duration in lead I (124 ms), and earliest onset QRS in V1 (vertical line). (E) ARVD/C-VT shows very late precordial transition V5 and wide QRS duration in lead I (126 ms). (F) ARVD/C-VT shows very late precordial transition V5 and wide QRS duration in lead I (150 ms). (G) ARVD/C-VT shows wide QRS duration in lead I (128 ms) and notching of the QRS (II, III, aVF, V4 to V6).
addition, some of the newer commercial electrocardiographs include the options to line up all lead tracings and include digital calipers for measurements (Philips Tracemaster MD, Andover, Massachusetts). These measurements are readily available with standard equipment used in the electrophysiology laboratory. Even in the absence of these tools, other criteria such as notching and precordial transition can be readily appreciated.

**Conclusions**

Several electrocardiographic criteria can help distinguish ventricular arrhythmia originating from ARVD/C from RVOT-VT. Precordial transition at lead V₆ was exclusively seen in ARVD/C patients. QRS duration of ≥120 ms in lead I was sensitive for the diagnosis of ARVD/C, whereas the presence of notching in the QRS and precordial transition at lead V₆ are specific for the diagnosis of ARVD/C. Multivariate logistic regression revealed that the duration of QRS in lead I of ≥120 ms, earliest onset QRS in lead V₁, notching, and transition V₅ or later all significantly increased the odds of ARVD/C. A combination of these factors helps in differentiating ARVD/C from RVOT-VT.

**REFERENCES**


**Key Words:** arrhythmogenic right ventricular cardiomyopathy - electrocardiography - right ventricular outflow tract.

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