Ablation at Right Coronary Cusp as an Alternative and Favorable Approach to Eliminate Premature Ventricular Complexes Originating From the Proximal Left Anterior Fascicle

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BACKGROUND: Premature ventricular complex (PVC) with narrow QRS duration originating from proximal left anterior fascicle (LAF) is challenging for ablation. This study was performed to evaluate the safety and feasibility of ablation from right coronary cusp (RCC) for proximal LAF-PVC and to investigate this PVC's characteristics.

METHODS: Mapping at RCC and left ventricle and ECG analysis were performed in 20 patients with LAF-PVC.

RESULTS: The earliest activation site (EAS), with Purkinje potential during both PVC and sinus rhythm, was localized at proximal LAF in 8 patients (proximal group) and at nonproximal LAF in 12 patients (nonproximal group). The Purkinje potential preceding PVC-QRS at the EAS in proximal group (32.6±2.5 ms) was significantly earlier than that in nonproximal group (28.3±4.5 ms, \( P = 0.025 \)). Similar difference in the Purkinje potentials preceding sinus rhythm QRS at the EAS was also observed between proximal and nonproximal groups (35.1±4.7 versus 25.2±5.0 ms, \( P < 0.001 \)). In proximal group, the distance between the EAS to left His bundle and to RCC was shorter than that of nonproximal group (12.3±2.8 versus 19.7±5.0 mm, \( P = 0.002 \), and 3.9±0.8 versus 15.7±7.8 mm, \( P < 0.001 \), respectively). No difference in the distance from RCC to proximal LAF was identified between the 2 groups. PVCs were successfully eliminated from RCC for all proximal groups but at left ventricular EAS for nonproximal groups. The radiofrequency application times, ablation time, and procedure time of nonproximal group were longer than that of proximal group. Electrocardiographic analysis showed that, when compared with nonproximal group, the PVCs of proximal group had narrower QRS duration; smaller S wave in leads I, V 5, and V 6; lower R wave in leads I, aVR, aVL, V 1, V 2, and V 4; and smaller q wave in leads III and aVF. The QRS duration difference (PVC-QRS and sinus rhythm QRS) <15 ms predicted the proximal LAF origin with high sensitivity and specificity.

CONCLUSIONS: PVCs originating from proximal LAF, with unique electrocardiographic characteristics, could be eliminated safely from RCC.

VISUAL OVERVIEW: A visual overview is available for this article.

Key Words: bundle of His • catheter ablation • coronary cusp • heart ventricles • humans

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WHAT IS KNOWN?

- Catheter ablation is challenging for premature ventricular complexes with narrow QRS duration originating from proximal left anterior fascicle (LAF). It frequently results in change of QRS morphology and high recurrence.

WHAT THE STUDY ADDS?

- Far-field tiny Purkinje potentials can be mapped at right coronary cusp above the proximal LAF.
- The anatomic correlation between the right coronary cusp and the proximal LAF provides an alternative approach for eliminating LAF premature ventricular complexes at right coronary cusp.
- LAF premature ventricular complexes have unique ECG features: narrower QRS duration; smaller S wave in leads I, V₅, and V₆; lower R wave in leads I, aVR, aVL, V₁, V₂, and V₄; smaller q wave in leads III and aVF; and smaller QRS duration difference between premature ventricular complexes and sinus rhythm.

Nonstandard Abbreviations and Acronyms

| EAS | earliest activation site |
|-----|--------------------------|
| LAF | left anterior fascicle |
| PVC | premature ventricular complex |
| RCC | right coronary cusp |
| SR | sinus rhythm |

Radiofrequency ablation has been demonstrated to be a low-risk and effective treatment for eliminating premature ventricular complexes (PVCs). Many studies demonstrated that ablation was also effective for those PVCs or ventricular tachycardia originating from or around the His-Purkinje system. However, due to the anatomic characteristics, ablation of those arrhythmias may potentially increase the risk of injuring the special conduction system, such as atioventricular block, left bundle branch block, etc. Furthermore, ablation of PVC with narrow QRS duration originating from proximal left anterior fascicle (LAF) was still challenging for clinical practice. We previously reported that a case of PVC with narrow QRS duration originating from proximal LAF was successfully eliminated by ablation at the right coronary cusp (RCC) without complications after failed ablation (with transient left bundle branch block) at left ventricle. Therefore, we presumed that RCC could be an alternative and favorable ablation target for proximal LAF-PVC due to the short anatomic distance. In this study, we prospectively investigated whether Purkinje potential during sinus rhythm (SR) and PVC can be recorded at RCC, what the anatomic relationship between the proximal LAF and RCC was, whether proximal LAF-PVC can be eliminated from RCC, and what was the electrocardiographic characteristic of proximal LAF-PVCs.

METHODS

The data that support the findings of this study are available from the corresponding authors upon reasonable request.

Study Population

From January 2015 to February 2019, a total of 507 patients with frequent PVCs underwent catheter ablation. A number of 20 (3.9%) patients with LAF-PVC were enrolled in the present study. LAF-PVC was defined as PVC-QRS with prominent inferior frontal plane QRS axis (qR in the inferior leads and rS in leads I and aVL) and with typical or atypical right bundle branch block. All patients provided written informed consent before ablation. This study was approved by the Ethics Committee of Shanghai General Hospital.

Anatomic Study of the RCC and LAF

LAF extends from the common trunk of left bundle branch to the anterosuperior left ventricle below the RCC and travels into the lateral free wall with terminal branch into the myocardial around the anterolateral papillary muscle. Therefore, the proximal LAF was anatomically defined as the LAF part running underneath RCC (ie, from the common trunk of left bundle branch to the anterior side of the interleaflet triangle between RCC and left coronary cusp; Figure 1A and 1B), whereas the lateral part was defined as nonproximal (middle and distal) LAF. For evaluation the anatomic characteristics and relationship between RCC and proximal LAF, mapping of the abovementioned area was prospectively performed in another 20 consecutive patients who underwent catheter ablation needed left-sided approach for left ventricular arrhythmia (but without LAF-PVC).

Electrophysiological Procedure for LAF-PVC

After discontinuation of antiarrhythmic drugs for at least 5 half-lives, all patients underwent electrophysiological study. Under the guidance of 3-dimensional electroanatomic mapping system (CARTO 3), a 7.5F irrigated catheter with distal electrode of 3.5 mm was used for mapping and ablation via femoral artery. Data were recorded by CARTO system and simultaneously by a digital multichannel electrophysiology recording system. Bipolar and unipolar signals were filtered at 30 to 500 Hz and 0.05 to 500 Hz, respectively. Intravenous heparin was administered to maintain the activated clotting time >250 seconds. Isoproterenol was administered to provoke clinical PVC if clinical PVC did not occur spontaneously.

Based on surface ECG morphology, electrophysiological mapping was initially performed by retrograde aortic approach. Three-dimensional electroanatomic reconstruction and mapping of the aortic root, especially the 3 coronary cusps, was performed before the catheter was introduced into the left ventricle (Figure 1A). Detailed activation mapping was performed to identify the earliest activation site (EAS) of PVCs, primarily to search for a sharp, high-frequency, and
presystolic Purkinje potential preceding PVC-QRS at RCC and left ventricle. For evaluating the relationship between the EAS and His-Purkinje system, right ventricular septum mapping was also performed to identify and locate the right His bundle before ablation, if necessary. Ablation attempt was performed from the adjacent RCC if the EAS was located at the proximal LAF (ie, underneath the RCC) or was performed at the left ventricular EAS if it was located at the nonproximal LAF. Before ablation attempt from RCC, angiography through the irrigated hole of ablation catheter and a pigtail catheter was performed to identify the position of ablation catheter and coronary arteries and to evaluate the ablation safety (Figure 1C). Therefore, the proximal LAF-PVC was defined as LAF-PVC with EAS originated from proximal LAF, whereas the nonproximal LAF-PVC was defined as LAF-PVC with EAS originated from nonproximal LAF.

Radiofrequency energy was delivered with an initial power of 20 W, uptitrated to a maximum power of 35 W with an irrigation rate of 17 to 30mL/min for 60 to 180 seconds while carefully monitoring the surface ECG and conduction intervals. Surface ECG, catheter position within RCC or left ventricle, temperature, and impedance were continuously monitored during ablation. Radiofrequency application will be terminated if PVCs did not disappear within 20 seconds or the catheter was dislodged. Mapping was repeated to search EAS. Successful ablation was defined as PVC elimination and no PVC recurrence with isoproterenol infusion, during at least a 30-minute observation period.
ECG Analysis
Before catheter ablation, the simultaneous 12-lead ECG of the SR and PVC was obtained. The analysis of the ECG pattern was focused on the following characteristics of SR-QRS and PVC-QRS: (1) QRS morphology, (2) QRS duration, and (3) maximal R-wave and S-wave amplitude of the 12 leads. All parameters were measured with electronic calipers by 2 experienced investigators blinded to the originating site. If there were discrepancies between those results, they were adjudicated by a third investigator.

Follow-Up
Surface ECG and 24-hour Holter ECG were taken immediately after procedure and the day at discharge, patients were scheduled for our outpatient clinic at 1, 3, 6, 9, and 12 months for the first year and every 6 months thereafter. All patients underwent 24-hour Holter monitoring during follow-up. If arrhythmia-related symptoms occurred, 12-lead ECG and 24-hour Holter monitoring was performed to document the cause of symptoms.

Statistical Analysis
Data for all continuous variables are presented as mean±SD. Data for categorical variables are presented as number and percentage. Categorical variables were compared using the χ² test or Fisher exact test. Continuous variables were compared using the Student t test or Mann-Whitney U test. A 2-tailed P of <0.05 was considered statistically significant. All statistical analyses were performed using SPSS 20.0.

RESULTS

Mapping Purkinje Potential at RCC in Patients Without LAF-PVC
Mapping of RCC and LAF was prospectively performed in another 20 patients without LAF-PVC (14 men; mean age, 40.9±12.3 years) who underwent catheter ablation (5 for idiopathic left ventricular tachycardia and 15 for PVCs originating from left ventricle but not from LAF). Notably, in all 20 patients, a small but significant and stable Purkinje potential was mapped at RCC (Figure I in the Data Supplement). The maximum Purkinje potential amplitude at RCC was 0.05±0.02 mV. The area that Purkinje potential could be mapped at RCC was 1.71±0.29 cm². The shortest distance from RCC to proximal LAF was 3.5±0.7 mm (2.4–5.2 mm). There was no significant difference in Purkinje potential amplitude and Purkinje potential mapped area at RCC during SR among the 2 groups (Table 2).

Notably, in all the 8 patients of proximal group, presystolic Purkinje potential during PVC was recorded at RCC (Figure 3). The maximum Purkinje potential amplitude at RCC was 0.05±0.02 mV. The area that Purkinje potential could be mapped at RCC was 1.66±0.54 cm². The shortest distance from RCC to proximal LAF was 3.6±0.8 mm (2.6–5.5 mm).

Characteristics of Patients With LAF-PVC
Twenty patients with LAF-PVC were enrolled in this study. Among the 20 patients, 8 (40.0%) patients, who’s PVCs originated from proximal LAF, were defined as proximal group (Figure 2). The remaining 12 (60.0%) patients, who’s PVCs originated from nonproximal LAF, were defined as nonproximal group (Figure II in the Data Supplement). Baseline characteristics were presented in Table 1. There was no significant difference in patients' characteristics among the 2 groups.

Mapping and Ablation of LAF-PVC
During electrophysiological procedure, spontaneous LAF-PVCs frequently occurred in all 20 patients. The major electrophysiological mapping parameters of PVCs are listed in Table 2.

In all 20 patients with LAF-PVC, the EAS was confirmed at the left ventricle, with a presystolic Purkinje potential (ie, fascicular potential) that preceded the PVC-QRS by 30.1±4.3 ms and preceded the SR-QRS by 29.2±6.9 ms. Presystolic Purkinje potential at EAS preceded the SR-QRS by 35.1±4.7 ms (31–44 ms) in proximal group and by 25.2±5.0 ms (16–30 ms) in nonproximal group (P<0.001). The Purkinje potential amplitude during PVC at the EAS of proximal group was smaller than that of nonproximal group (P=0.040). In the proximal group, the distances between the EAS to left His bundle and to RCC were both shorter than that of the nonproximal group (P=0.002 and P<0.001, respectively), further indicating the proximal LAF origin at the proximal group.

In all 20 patients, a small but constant and stable Purkinje potential during SR was mapped at RCC (Figure 3). During procedure, spontaneous PVC morphology change was observed in 2 (10.0%) patients of nonproximal group, preceding the PVC-QRS only by 14.±5.3 ms (Figure 3). However, presystolic Purkinje potential during PVC was recorded at RCC in 8 of 12 (66.7%; P=0.117) patients of nonproximal group, preceding the PVC-QRS by only 5.3±3.3 ms, which was significantly later than the timing in the proximal group (P<0.001).

Interestingly, for all proximal group patients, PVCs were successfully eliminated from RCC without ablation attempt at left ventricle (Figure 3). Furthermore, 1 patient (12.5%) of proximal group underwent her repeated ablation after a failed ablation at the left ventricle (transient success but recurrence soon) at other center (Figure 4). However, for all nonproximal group patients, PVCs were eliminated at left ventricular EAS without ablation attempt at RCC. During procedure, PVC morphology change was observed in 2 (10.0%) patients (both in nonproximal group; P=0.495). However, the requirement of repositioning mapping catheter was found only in 1 patient from nonproximal group (P=0.020). Therefore, the radiofrequency application times (P=0.003), ablation time (P=0.005), and procedure time (P=0.018) of nonproximal group were longer.
than that of proximal group. Radiofrequency-induced left anterior hemiblock, which was shown in Figure 4, indicated the changes of SR-QRS morphology to a deeper S wave in the inferior leads and a higher R wave in leads I and aVL. Left anterior hemiblock was observed in 10 (50.0%) patients, including 1 (12.5%) of proximal group and 9 (75.0%) of nonproximal group ($P=0.020$; Table 2). No junctional rhythm or atrioventricular conduction block was observed.

**Electrocardiographic Characteristics**

The PVC morphology of all the 20 patients has the feature of left posterior fascicular block, which indicated a qR or qRs complex in the inferior leads and an rS or RS complex in leads I and aVL. Typical right bundle branch block morphology (a terminal R wave in lead V1 and a prolonged S wave in leads I and V6) was observed in 13 (65.0%) patients, including 1 patient (12.5%, patient 3 in Figure 2) of proximal group and all nonproximal group patients (100%, $P<0.001$).

There are other significant differences of PVC morphology between the proximal group and nonproximal group (Table 3). The PVC-QRS duration of proximal group was narrower than that of nonproximal group ($P=0.004$). Notably, the proximal group had smaller QRS duration differences between PVC-QRS and SR-QRS ($P<0.001$).
The R-wave amplitude in leads I, aVR, and aVL and the S-wave amplitude in lead I of proximal group were smaller than that of nonproximal group ($P=0.042$, $P=0.006$, $P=0.029$, and $P=0.002$, respectively).

The q-wave amplitude in lead III and aVF of proximal group were smaller than that of nonproximal group ($P=0.011$ and $P=0.043$).

The R-wave amplitude in leads $V_1$, $V_2$, and $V_4$ of proximal group was lower than that of nonproximal group ($P=0.003$, $P=0.026$, and $P=0.015$, respectively). The S-wave amplitudes in leads $V_5$ and $V_6$ of proximal group were both smaller than that of nonproximal group ($P=0.010$ and $P=0.009$). When compared with nonproximal group, proximal group had lower R/S ratio on lead $V_2$ ($P=0.002$) but had higher R/S ratio on leads $V_5$ and $V_6$ ($P=0.046$ and $P=0.009$, respectively).

The sensitivity, specificity, and positive and negative predictive accuracies of the electrocardiographic characteristics for PVCs originating from proximal LAF are shown in Table 4. The QRS duration difference (between PVC-QRS and SR-QRS) <15 ms was a good predictor of proximal LAF origin site with high sensitivity and specificity.

### Follow-Up

During a mean follow-up of 26.5±12.0 months, 2 patients of nonproximal group experienced PVC recurrence at the first month after ablation. One patient became asymptomatic and PVC burden reduced from 13.2% to 2.7% with previous failed antiarrhythmic (diltiazem) and the other refused a repeated ablation procedure. The total successful rate of one procedure was 90.0% (18 of 20) and no difference between the 2 groups ($P=0.495$; Table 2). No procedure-related complications occurred during hospitalization or follow-up. No iatrogenic aortic regurgitation was revealed by echocardiography during follow-up.

| Table 1. Basic Characteristics |
|------------------------------|
| Total (n=20) | Proximal Group (n=8) | Nonproximal Group (n=12) | $P$ Value |
| Age, y | 42.3±17.7 | 45.3±16.7 | 40.5±18.7 | 0.577 |
| Sex (male), n (%) | 10 (50.0%) | 3 (37.5%) | 7 (58.3) | 0.850 |
| Symptoms, n (%) | 17 (85.0%) | 7 (87.5%) | 10 (83.3) | 1.000 |
| Hypertension, n (%) | 3 (15.0%) | 1 (12.5%) | 2 (16.7) | 0.798 |
| LVEF, % | 63.3±9.9 | 59.1±13.2 | 66.1±6.1 | 0.126 |
| PVC history, mo | 43.6±61.9 | 59.6±80.7 | 32.8±46.5 | 0.357 |
| PVC burden, % | 16.6±5.5 | 18.1±2.4 | 15.7±6.8 | 0.351 |
| Antiarrhythmic drugs | 1.3±0.7 | 1.0±0.8 | 1.5±0.7 | 0.139 |

LVEF indicates left ventricular ejection fraction; and PVC, premature ventricular complex.

| Table 2. Mapping and Ablation Parameters |
|------------------------------|
| Total (n=20) | Proximal Group (n=8) | Nonproximal Group (n=12) | $P$ Value |
| Purkinje potential preceded PVC-QRS at EAS, ms | 30.1±4.3 | 32.6±2.5 | 28.3±4.5 | 0.025 |
| Purkinje potential preceded SR-QRS at EAS, ms | 29.2±6.9 | 35.1±4.7 | 25±5.0 | <0.001 |
| Purkinje potential amplitude during PVC at EAS, mV | 0.33±0.21 | 0.21±0.13 | 0.40±0.22 | 0.040 |
| Purkinje potential amplitude during SR at EAS, mV | 0.56±0.40 | 0.40±0.35 | 0.86±0.42 | 0.159 |
| Purkinje potential amplitude during SR at RCC, mV | 0.05±0.02 | 0.05±0.02 | 0.05±0.02 | 0.793 |
| Purkinje potential mapped area during SR at RCC, cm² | 1.7±0.29 | 1.65±0.35 | 1.74±0.26 | 0.510 |
| Shortest distance from RCC to proximal LAF, mm | 3.5±0.7 | 3.5±0.8 | 3.5±0.7 | 0.903 |
| Distance between the EAS and His, mm | 16.7±5.6 | 12.3±2.8 | 19.7±5.0 | 0.002 |
| Distance between the EAS and RCC, mm | 11.0±8.4 | 3.9±0.8 | 15.7±7.8 | <0.001 |
| PVC morphology change during procedure, n (%) | 2 (10.0) | 0 | 2 (16.7) | 0.495 |
| Reposition of ablation catheter, n (%) | 10 (50.0) | 1 (12.5) | 9 (75.0) | 0.020 |
| Radiofrequency application, times | 5.9±1.5 | 2.4±1.2 | 8.2±3.1 | 0.003 |
| Ablation time, min | 7.2±6.2 | 3.1±2.5 | 9.9±6.5 | 0.005 |
| Procedure time, min | 70.6±15.3 | 61.0±6.9 | 76.9±16.2 | 0.018 |
| ECG morphology changed after ablation, n (%) | 10 (50.0) | 1 (12.5) | 9 (75.0) | 0.020 |
| Long-term single-procedure success rate, n (%) | 18 (90.0) | 8 (100.0) | 10 (83.3) | 0.495 |

EAS indicates earliest activation site; LAF, left anterior fascicle; PVC, premature ventricular complex; RCC, right coronary cusp; and SR, sinus rhythm.
The main findings of this study are as follows: (1) during SR, Purkinje potential can be mapped at RCC above the proximal LAF in all patients with and without LAF-PVC; (2) the anatomic correlation between the RCC and proximal LAF can provide the RCC as an alternative and favorable approach for eliminating proximal LAF-PVC;
(3) proximal LAF-PVCs have less electrocardiographic morphology change between the PVC and SR; (4) the QRS duration difference (between PVC-QRS and SR-QRS) <15 ms was a good predictor of proximal LAF origin site with high sensitivity and specificity.

**Anatomic Considerations**

Human anatomy study shows that the His bundle penetrates the right fibrous trigone and emerges between the noncoronary cusp and RCC giving off the sheet of left fascicles, which run down beneath the endocardium from the inferior ring of the membranous septum. Moreover, some studies show that the dead-end tract of the conduction system could be observed and mapped at or beneath the RCC. In the present study, the proximal LAF was anatomically defined as the LAF part running beneath the RCC (Figure 1). From the mapping data, the proximal LAF running below the RCC with a shortest distance to the RCC by 3.5±0.7 mm.

**Figure 4.** The first and second procedure data of proximal left anterior fascicle (LAF) premature ventricular complex (PVC; patient 8 of proximal group).

A. During the first procedure, the earliest activation site (EAS; MP1, blue dot), with Purkinje potential (red arrows) preceded the PVC-QRS (red star) by 31 ms, was mapped and located at the proximal LAF. Several radiofrequency applications, targeted at MP1, eliminated the PVC and changed the ECG morphology (MP2, pink dot). However, 3 h later, the PVC recurred. The yellow dots indicated the route of LAF and left posterior fascicle. The red dots indicated the ablation targets. B. During the second procedure, the EAS (MP3, blue dot), with Purkinje potential (red arrows) preceded the PVC-QRS (red star) by 31 ms, was also mapped and located at the proximal LAF. Single radiofrequency application (red dot), performed from right coronary cusp (RCC), eliminated the PVC without ECG changed. ABL indicates ablation catheter; Ao, aorta; LAO, left anterior oblique; and LV, left ventricle.
Furthermore, in the proximal group, the distance from the proximal LAF origin to RCC (3.9±0.8 mm) is shorter than 6 mm, which theoretically makes the penetration of radiofrequency-induced conductive lesion to the proximal or the branch of proximal LAF.\textsuperscript{14,15} However, the distance from the nonproximal LAF origin to RCC is longer than 6 mm. Hence, the spatial correlation and short distance between the RCC and proximal LAF provides RCC as an available target for PVCs originating from the proximal LAF.

Table 3. Electrocardiographic Characteristics of Left Anterior Fascicle PVC

| Electrocardiographic Characteristics | Total (n=20) | Proximal Group (n=8) | Nonproximal Group (n=12) | P Value |
|-------------------------------------|-------------|----------------------|--------------------------|---------|
| QRS duration, ms                   | 111.4±14.7  | 100.6±11.5           | 118.6±12.1               | 0.004   |
| QRS duration difference between PVC and SR, ms | 18.0±9.2  | 9.0±3.9              | 24.0±5.3                 | <0.001  |
| R-wave amplitude in I, mV           | 0.34±0.18   | 0.24±0.14            | 0.40±0.17                | 0.042   |
| S-wave amplitude in I, mV           | 0.45±0.18   | 0.31±0.13            | 0.54±0.15                | 0.002   |
| R-wave amplitude in aVL, mV         | 0.22±0.11   | 0.15±0.06            | 0.27±0.10                | 0.006   |
| S-wave amplitude in aVL, mV         | 1.00±0.37   | 0.83±0.38            | 1.11±0.34                | 0.097   |
| R-wave amplitude in aVR, mV         | 0.19±0.10   | 0.14±0.08            | 0.23±0.09                | 0.029   |
| q-wave amplitude in II, mV          | 0.13±0.07   | 0.10±0.05            | 0.15±0.07                | 0.126   |
| q-wave amplitude in III, mV         | 0.23±0.12   | 0.16±0.07            | 0.28±0.12                | 0.011   |
| q-wave amplitude in aVF, mV         | 0.18±0.09   | 0.13±0.05            | 0.21±0.09                | 0.043   |
| R-wave amplitude in V_s, mV         | 0.52±0.31   | 0.29±0.16            | 0.67±0.28                | 0.003   |
| R/S ratio on V_1                    | 2.72±2.71   | 1.36±2.29            | 3.62±2.68                | 0.067   |
| R-wave amplitude in V_s, mV         | 0.97±0.49   | 0.68±0.27            | 1.17±0.05                | 0.026   |
| R/S ratio on V_s                    | 1.13±0.54   | 0.71±0.28            | 1.41±0.49                | 0.002   |
| R-wave amplitude in V_s, mV         | 1.42±0.40   | 1.16±0.42            | 1.59±0.30                | 0.015   |
| R/S ratio on V_s                    | 2.83±1.42   | 2.98±1.51            | 2.74±1.42                | 0.717   |
| S-wave amplitude in V_s, mV         | 0.41±0.24   | 0.25±0.11            | 0.51±0.24                | 0.010   |
| R/S ratio on V_s                    | 3.80±2.40   | 5.10±3.15            | 2.94±1.26                | 0.046   |
| S-wave amplitude in V_s, mV         | 0.26±0.14   | 0.17±0.08            | 0.33±0.14                | 0.009   |
| R/S ratio on V_s                    | 4.61±2.35   | 6.10±2.78            | 3.45±1.25                | 0.009   |

Table 4. Sensitivity, Specificity, and Positive and Negative Predictive Accuracies of Electrocardiographic Characteristics for Proximal Left Anterior Fascicle Premature Ventricular Complexes

| Electrocardiographic Characteristics | Sensitivity | Specificity | Positive Predictive Value | Negative Predictive Value |
|-------------------------------------|-------------|-------------|---------------------------|---------------------------|
| QRS duration <115 ms                | 58.3        | 87.5        | 87.5                      | 58.3                      |
| QRS duration difference <15 ms      | 100         | 100         | 100                       | 100                       |
| No right bundle branch block in V_1 | 100         | 92.3        | 87.5                      | 100                       |
| R-wave amplitude in I <0.30 mV      | 62.5        | 75          | 62.5                      | 75                        |
| S-wave amplitude in I <0.40 mV      | 75.0        | 83.3        | 75.0                      | 83.3                      |
| R-wave amplitude in V_s <0.20 mV    | 70.0        | 90.0        | 87.5                      | 75.0                      |
| R-wave amplitude in aVL <0.20 mV    | 58.3        | 87.5        | 87.5                      | 58.3                      |
| q-wave amplitude in III <0.15 mV    | 100         | 75.0        | 50.0                      | 100                       |
| q-wave amplitude in aVF <0.20 mV    | 50.0        | 83.3        | 87.5                      | 41.7                      |
| R-wave amplitude in V_s <0.30 mV    | 100         | 75.0        | 50.0                      | 100                       |
| R-wave amplitude in V_s <0.70 mV    | 100         | 85.7        | 75.0                      | 100                       |
| R/S ratio on V_1 <1.00              | 85.7        | 84.6        | 75.0                      | 91.7                      |
| R-wave amplitude in V_1 <1.20 mV    | 57.1        | 69.2        | 50.0                      | 75.0                      |
| S-wave amplitude in V_s <0.40 mV    | 63.6        | 88.9        | 87.5                      | 66.7                      |
| R/S ratio on V_s >4.00              | 62.5        | 75.0        | 62.5                      | 75.0                      |
| S-wave amplitude in V_s <0.25 mV    | 77.8        | 90.9        | 87.5                      | 83.3                      |
| R/S ratio on V_s >4.00              | 70.0        | 90.0        | 87.5                      | 75.0                      |
Mapping of Purkinje Potential at RCC

In the previous report, the LAF-PVC, with Purkinje potential recorded at RCC during both SR and PVC, was successfully eliminated from RCC after failed ablation (with transient left bundle branch block) at the left ventricle. In the present study, a small but significant and stable Purkinje potential could be identified and mapped at a small area of RCC during SR in all patients with LAF-PVC and in all control patients without LAF-PVC (Figure 3; Figure II in the Data Supplement). Importantly, the amplitude of the Purkinje potential at RCC was 0.05±0.02 mV and smaller than that recorded at the left ventricle. Also, during PVC, the Purkinje potential preceded PVC-QRS was found in 16 of 20 (80.0%) patients with LAF-PVC and also presented as a tinypotential. The mapped tiny Purkinje potential at RCC probably indicates far-field activation from proximal LAF to RCC due to the short anatomic distance.

Mapping and Ablation of LAF-PVC

In our study population, all the cases presented with frequent PVCs of single QRS morphology, which was similar to a previous study. High-frequency and presystolic Purkinje potential preceding the QRS during PVC was mapped in all LAF-PVC cases. Therefore, the underlying mechanism of LAF-PVC was expected to be abnormal automaticity or triggered activity. Some studies have reported that ablation could be performed at RCC for ventricular arrhythmias originating from the RCC or para-Hisian region. However, those QRS morphology characteristics (wide duration and none had q wave in the inferior leads) of the abovementioned studies were different from that of our cases (narrow duration and all had the q wave in the inferior leads). Our data showed that LAF-PVCs have common electrocardiographic characteristics of qR or qRs patterns in the inferior leads, which was consistent with previous studies. Furthermore, the EAS of our cases, with high-frequency and presystolic Purkinje potential preceding the QRS during both PVC and SR (ie, fascicular potential), was located at the LAF of left ventricle and had distance to the left His bundle. Therefore, according to the electrocardiographic morphologies and the electrophysiology mapping results, these PVCs could be clearly confirmed as LAF origin but not myocardial origin close to RCC or para-Hisian origin.

Usually, ablation was performed at the left ventricle for LAF-PVC. However, catheter ablation of LAF-PVC with narrow QRS duration is challenging and may be associated with a risk of inadvertent conduction block. The complications may due to several reasons, such as poor contact between the catheter and targeted site, unstable position associated with deep respiration during ablation, catheter displacement, and so on. In another study, for ablation PVCs originating near left His bundle, a transseptal approach using a reversed S curve was performed. Indeed, that can provide a stable position. However, it needs transseptal puncture and required a long learning curve and may increase the cost, procedure time. In our study, for those PVCs originating from proximal LAF, ablation at RCC can eliminate the proximal LAF-PVC although the activation of RCC during PVC was significantly later than that of left ventricular EAS (further excluded the RCC origin of proximal group). Nevertheless, ablation at left ventricle may eliminate our proximal LAF-PVC. However, ablation from RCC makes less catheter reposition and less QRS morphology change in the proximal group. Furthermore, 1 patient of proximal group achieved successful ablation from RCC after a failed ablation at left ventricle (transient success but recurrence soon; Figure 4). Therefore, ablation at RCC, with the improvement of clinical practice (better catheter stability, easier manipulation, and less risk), could be an alternative and favorable target for eliminating PVCs originating from the proximal LAF.

QRS Morphology Characteristics of LAF-PVC

In our study, there are some significant differences in electrocardiographic characteristics between different origin sites of LAF-PVC (Figure 2; Figure I in the Data Supplement; Table 3). PVCs originating from the proximal LAF have less electrocardiographic morphology change between the PVC and SR, that is, smaller S wave in leads I, V_1, and V_6; lower R wave in leads I, aVR, aVL, V_1, V_2, and V_6; and smaller q wave in leads III and aVF and smaller QRS duration difference (between PVC-QRS and SR-QRS). Furthermore, the QRS duration difference (between PVC-QRS and SR-QRS) <15 ms was a good predictor of proximal LAF origin site with high sensitivity and specificity (Table 4). Due to the isolated conduction within the left His-Purkinje system, ectopic rhythm from proximal LAF would activate the whole His-Purkinje system faster than that from nonproximal LAF. As a result, the biventricular depolarization time of ectopy originating from proximal LAF would be smaller than that of nonproximal LAF. This may help explaining the electrocardiographic characteristics of proximal LAF-PVC.

Limitations

In this study population, pace mapping was not performed at RCC to mimic the morphology of PVCs. However, it was very difficult to perform pace mapping because the stable and significant tiny Purkinje potential was mapped. Ablation from RCC also required a special caution to avoid the injury of His-Purkinje system or right coronary artery. The ablation target at the RCC was confirmed by the 3-dimensional anatomic mapping, catheter morphology and the contact force, and angiography in this study population. Using of intracardiac echocardiography can...
provide more information regarding the anatomy, mapping catheter, and RF-induced lesion. However, it is infrequently used due to the high cost. Also, remap at RCC and left ventricle after successful ablation was not systematically performed to clarify whether the Purkinje potential during SR exists or disappears. However, it strongly suggested that Purkinje potential may come from the small branch of proximal LAF in the patients without change of QRS morphology during SR after successful ablation. The number of patients enrolled in this study was small, therefore, the accuracy of electrocardiographic characteristic may be overestimated, and further evaluation may be needed. The result should be used with caution in those patients who presented with conduction block or aberrant ventricular conduction during SR.

Conclusions

Purkinje potential can be recorded at RCC during SR in all patients with and without LAF-PVC and can be recorded at RCC during PVC in all patients with PVC originating from proximal LAF. RCC provides an alternative and favorable anatomic target for ablating those PVCs originating from proximal LAF due to the close relationship between the RCC and proximal LAF. Idiopathic PVCs originating from proximal LAF have unique electrocardiographic characteristics. The QRS duration difference (between PVC-QRS and SR-QRS) <15 ms was a good predictor of proximal LAF origin site with high sensitivity and specificity.

ARTICLE INFORMATION

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Disclosures

None.

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