CASE REPORT

Catheter ablation of premature ventricular contractions originating from the His bundle region

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We describe a 35-year-old man with idiopathic frequent premature ventricular contractions (PVCs) originating from the His-bundle region, where a low frequency ventricular potential was recorded in the bipolar recording and QR pattern in the unipolar recording. Electro-anatomical mapping revealed a confined low voltage zone (<1.2 mV) in the His-bundle region, and the activation spread out radially to the entire right ventricle. The PVCs were successfully abolished without any atrioventricular block by a step-wise incremental radiofrequency energy application to the His-bundle region. The patient has remained free from PVCs or any symptoms without medication during a 23 month follow-up period.

KEYWORDS
Premature ventricular contractions;
The His-bundle region;
Electro-anatomical mapping;
Radiofrequency catheter ablation

Introduction

We report a case of idiopathic premature ventricular contractions (PVCs) originating from the His-bundle region in which the electrophysiologic characteristics were examined in detail with the use of conventional and electro-anatomical mapping, and in which radiofrequency catheter ablation (RFCA) abolished the PVCs.

Case report

A 35-year-old man with recurrent palpitations at rest and during exercise was referred to our institution to treat his symptomatic PVCs that were refractory to several class I anti-arrhythmic agents. The ECG during sinus rhythm exhibited a normal QRS morphology and ST-T segment. An extensive investigation including echocardiography, left ventriculography, and coronary angiography revealed no structural heart disease. At baseline, frequent PVCs with left bundle branch block morphology and normal axis were observed in a bigeminal fashion with a coupling interval of 400 msec, and were consistent with originating from the His-bundle region (Figure 1).1 A total number of 45 000 PVCs per day were observed on the 24-h ambulatory Holter monitoring in spite of the prophylactic oral administration of mexiletine (300 mg/day).

After obtaining written informed consent, we performed an electrophysiological study using the standard technique with no anti-arrhythmic agents during a fasting non-sedated state. Although PVCs frequently occurred during the baseline, sustained ventricular tachycardia (VT) was not induced by either programmed ventricular stimulation using two different drive cycle lengths with up to three extrastimuli or incremental burst pacing at a cycle length of up to 250 msec delivered from the right ventricular apex (RVA) or right ventricular outflow tract (RVOT).

During activation mapping in the RV with the use of electro-anatomical and conventional mapping (Figures 2 and 3), the earliest activation was identified at the His-bundle region where a small His-bundle electrogram could be recorded during sinus rhythm, and during the PVC a low frequency ventricular electrogram preceding the QRS by 33 msec was recorded on the bipolar recording and QR pattern on the unipolar recording though the ventricular electrogram during sinus rhythm was of a high frequency. The timing of the beginning of the bipolar and unipolar QR electrograms during the PVC was identical, suggesting that the PVC originated from an endocardial site in that region.

In the voltage mapping constructed with the electro-anatomical mapping (Figure 2, right), it was observed that there was a small low voltage zone (<1.2 mV) confined to an area around the His-bundle region while the rest of the RV had a normal voltage (>1.2 mV), and the activation of the PVC spread out radially to the rest of the entire RV (Figure 2, left). The conduction through the atrioventricular node-His-Purkinje system appeared to be normal because the AH and HV intervals during sinus rhythm were 61 and 36 msec, respectively, (Figure 3), and the effective...
The refractory period of the AH and HV intervals were 240 and 250 msec, respectively. These findings appeared to suggest that the substrate of the PVCs consisted of a diseased area of the ordinary endocardial myocardium surrounding the His-bundle region but did not include the atrioventricular node-His-Purkinje system. During pace mapping performed from the ablation catheter in the His-bundle region during sinus rhythm, a perfect pace map was obtained.

RFCA was carefully performed in the His-bundle region according to the method advocated by Haissaguerre, et al., in which a step-wise incremental application of the RF energy was performed with 5, 7, 10, and 15 W, while increasing the energy of each of those steps at 10-s intervals over 40 s. The RF energy application was terminated when a prolongation of the AV interval, AV block, abnormal impedance rise (>30 Ω) or excessive junctional ectopic beats of >5 beats were observed. After the initiation of the RFCA, the PVCs suddenly disappeared and became completely abolished. The RFCA was then continued for 40 s, and during that delivery, five successive junctional ectopic beats were initially observed and subsequently disappeared. No procedure-related complications including impairment of the atrioventricular conduction were observed, and a 24-h

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**Figure 1** A 12-lead ECG on admission. Bigeminal PVCs with a left bundle-branch block configuration and normal axis morphology with a coupling interval of 400 msec were observed.

**Figure 2** Activation map and voltage map using an electro-anatomical mapping system. These panels show the entire right ventricle in the posterior view from the tricuspid valve. The left panel shows an activation map in which the activation originates from the His-bundle region and spreads out to the entire right ventricle. The right panel shows a voltage map in which there is a confined low voltage area (<1.2 mV) only in the His-bundle region.
ambulatory Holter recording performed after the procedure confirmed the abolition of the PVCs. The patient has been free from any PVCs or symptoms without any medications during a follow-up period of 23 months.

Discussion

The main findings of this study were that, firstly, the frequent symptomatic PVCs originating from the His-bundle region were successfully eliminated by the RF ablation guided by both pace mapping and activation mapping with the use of both conventional and electro-anatomical mapping methods. Secondly, a confined diseased area of the ordinary endocardial myocardium, not including the conduction tissue, was observed in the His-bundle region, which might have been the substrate for the PVCs. Thirdly, a step-wise incremental application of the RF energy safely and successfully abolished the PVCs without causing any impairment of the atrioventricular conduction.

Yamauchi et al. have reported that VT/VPCs (ventricular premature contractions) originating from near the His-bundle have distinctive ECG characteristics. The QRS morphology of the VPCs in our case was consistent with that of those originating from the vicinity of the His-bundle, in which the R wave amplitude in the inferior leads was low and that in leads V5 and V6 was high, and an R wave was present in aVL, and lead V1 exhibited a QS pattern.

As mentioned above, the focus of the PVCs appeared to be confined to the ordinary endocardial myocardium and did not include the His-Purkinje system. If activation arose from a deeper region or epicardial site, it would be expected that the morphology of the unipolar electrogram recorded from the electrode on the endocardial surface might have had an rS or RS pattern depending on the amount of the preceding excitation approaching the electrode, and the timing of the beginning of the unipolar electrogram would have preceded that of the bipolar electrogram. In the present case, the timing of the beginning of the bipolar electrogram of the PVC was identical to that of the unipolar electrogram in addition to having a QR morphology in the unipolar electrogram, suggesting that the PVCs originated from an endocardial site. Thus, it was suggested that the application of the RF energy should be limited to only the endocardial surface, and not to the deeper layers which could result in damage to the His-Purkinje system conduction.

RFCA of parahisian accessory pathways has been established in previous studies, where attention was paid to avoid any undesirable effects on the atrioventricular conduction. Those efforts included a step-wise incremental RF energy application, and careful observation of any successive junctional ectopic beats (> 5) which would act as a warning sign for subsequent atrioventricular block. In our patient, a low energy application just enough to ablate the PVC focus which was suggested to be located in the superficial layer of the endocardium might have been all that was needed to abolish the PVCs. Although there have been no previous reports regarding an established method to eliminate VT or PVCs originating from the His-bundle region without creating atrioventricular conduction block, we applied the method advocated by Halissaguerre et al. to the RFCA for the PVCs in the present case and successfully eliminated the frequent PVCs without impairing the atrioventricular conduction.

In the present case, although adequate pressure was applied to the tip of the mapping catheter during the endocardial mapping, and sufficient contact between the mapping catheter and endocardial surface was confirmed at every mapping point, a confined low voltage area in the His-bundle region, not involving the rest of the RV was observed. The reason for that remains unclear.

Conflict of interest: T.T. is a member of the advisory board of Nihon Kohden.
References


