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Trade-off between elimination of premature ventricular complexes and loss of **s**ynchronized left ventricular pacing improved cardiac function in a patient with heart failure





Hideyuki Hasebe ^{a, *}, Masataka Iida ^b, Naoki Hatano ^b, Toshiro Muramatsu ^b

^a Division of Arrhythmology, Shizuoka Saiseikai General Hospital, Shizuoka, Japan
^b Division of Clinical Engineering, Shizuoka Saiseikai General Hospital Shizuoka, Japan

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ABSTRACT

Synchronized left ventricular pacing (sLVP) via adaptive cardiac resynchronization therapy (aCRT) algorithm might enhance the efficacy of CRT. A 71-year-old female was admitted with the diagnosis of heart failure. Electrocardiograms revealed left bundle branch block with QRS width of 144 ms, and frequent premature ventricular complexes (PVCs). A CRT device was implanted, and sLVP via a CRT algorithm was provided. Frequent PVCs arising from the His-bundle suppressed effective sLVP percentage. Radiofrequency application at the His-bundle successfully eliminated the PVCs, but induced complete atrioventricular (AV) block, leading to loss of sLVP. These procedures remarkably improved the patient's cardiac function, suggesting the advantage of PVC elimination overweighed the disadvantage of loss of sLVP.

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1. Introduction

Several studies have shown that left ventricular (LV) only pacing has equivalent or superior to biventricular pacing in some patients who receive cardiac resynchronization therapy (CRT) in both acute and chronic phase [1–3]. In patients with left bundle branch block (LBBB) and preserved atrioventricular (AV) conduction, synchronization of LV pacing with intrinsic activation (sLVP) via right bundle branch might enhance the efficacy of CRT. Adaptive CRT (aCRT) is a novel algorithm that periodically measures intrinsic conduction and provides sLVP with dynamically adjusted timing [2,3].

Frequent premature ventricular complexes (PVCs) can cause a reversible form of cardiomyopathy [4,5]. The management of high PVC burden is especially important for patients who have received CRT from the view point of ventricular pacing percentage and

E-mail address: h153478@siz.saiseikai.or.jp (H. Hasebe).

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cardiomyopathy due to PVCs.

We describe here a case of chronic heart failure due to LBBB and frequent PVCs originating from the His-Purkinje system. After CRT device implantation, the catheter ablation successfully eliminated PVCs. At the same time, the ablation induced complete AV block, resulting in the loss of sLVP. Cardiac function remarkably improved in spite of the loss of sLVP due to AV block.

2. Case report

A 71-year-old female was diagnosed with heart failure (New York Heart Association [NYHA] class III) owing to nonischemic cardiomyopathy despite optimal medication. Serial ECGs revealed sinus rhythm, LBBB morphology wide QRS complex of 144 ms and frequent ectopic beats, the QRS morphology of which was similar with intrinsic conduction (Fig. 1A). Twenty-four-hour Holter ECG disclosed a PVC burden of 25%. The patient had heart failure symptoms including dyspnea, and general fatigue, while she had no symptoms from PVCs such as palpitations, or skipped beat sensation. Echocardiography showed dyssynchronous, impaired LV systolic motion (LVEF, 33%) and LV dilatation (LVDd, 69 mm) without any valve disease. An electrophysiological study revealed that the ectopic beats were PVCs preceded by Purkinje potentials (Fig. 2). The H–V intervals during intrinsic conduction, and PVC were 66,

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Abbreviations: aCRT, adaptive CRT; AV, atrioventricular; CRT, cardiac resynchronization therapy; LBBB, left bundle branch block; LV, left ventricle; NYHA, New York Heart Association; PVC, premature ventricular complex; sLVP, synchronized left ventricular pacing.

^{*} Corresponding author. Division of Arrhythmology, Shizuoka Saiseikai General Hospital, 1-1-1 Oshika, Suruga-ku, Shizuoka, 422-8527, Japan

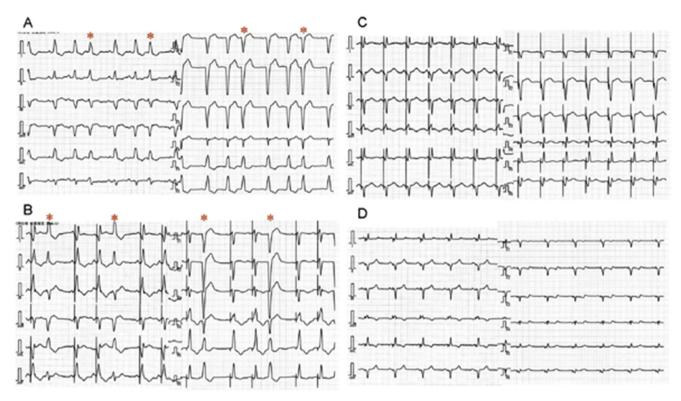


Fig. 1. Time course change in the 12-lead electrocardiograms (ECGs). A: Upon admission, wide QRS complex with left bundle branch block morphology and frequent premature ventricular complexes (PVCs) with similar morphology with that during sinus rhythm (red asterisks). B: After cardiac resynchronization therapy (CRT) introduction, the CRT device provided left ventricular (LV) pacing synchronized with intrinsic activation via the right bundle (sLVP). The pacing polarity of LV lead was LV2 to RV-coil. The frequency of PVCs (red asterisks) remained unchanged. C: After PVC ablation, the CRT device provided biventricular pacing. The pacing polarity of LV lead was LV1 to RV-coil. D: The paced QRS duration was shortened at six months after ablation compared with that just after the ablation.

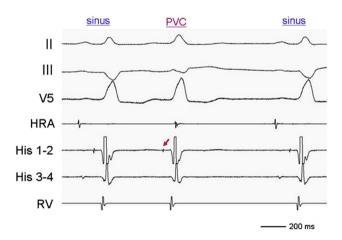


Fig. 2. Intracardiac electrocardiogram. The ectopic beats turned out to be premature ventricular complexes (PVCs) preceded by Purkinje potentials. HRA = high right atrium; PVC = premature ventricular complex; RV = right ventricle.

and 58 ms, respectively. The earliest activation site of the preceding Purkinje potential was in the His region. An attempt of catheter ablation for these PVCs was highly likely to induce complete AV block. Therefore, the patient was implanted with a Viva Quad XT CRT-D device (Medtronic, Inc., Minneapolis, MN, USA) as initial therapy (Fig. 3). The adaptive CRT algorithm provided sLVP that was 96% in all ventricular pacing. The ventricular pacing percentage was only 75%, because the PVC burden persisted (25%) after CRT device implantation despite medication with bisoprolol (5 mg, daily). The PVC-QRS duration was 156 ms, while that generated by sLVP was

135 ms (Fig. 1B). Therefore, catheter ablation proceeded two weeks after CRT device implantation. Focal application at the His region successfully eliminated the PVCs. At the same time, this procedure induced complete AV block, leading to the loss of sLVP. After the session, the PVC burden decreased from 25% to 1%, and hence the device provided 99% of the biventricular pacing.

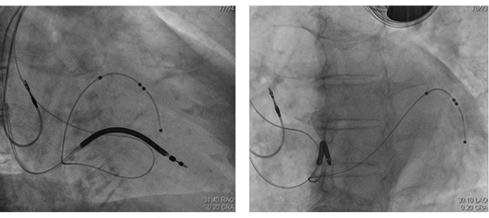
Six months later, the patient reported a remarkable improvement in symptoms and cardiac function. Indeed, the LVEF had increased from 33% to 65%, the LVDd had decreased from 69 to 49 mm, and the NYHA class had improved from III to II. The paced-QRS duration was shortened from 138 (just after the ablation) to 126 ms, indicating electrical remodeling as a result of the CRT and PVC ablation (Fig. 1C and D).

3. Discussion

A subanalysis of aCRT trial suggested that sLVP might be more effective than conventional biventricular pacing [3]. In the present case, focal application at the His region to eliminate PVCs originating from the His-Purkinje system necessarily induced complete AV block, leading to a loss of sLVP. The effect of trade off between loss of sLVP and PVC elimination on cardiac function was unpredictable before the procedure.

The impact of loss of sLVP is obscure based on currently available clinical evidence.

Several studies have shown both acute and chronic benefit of sLVP when the intrinsic conduction through right bundle is preserved [1-3]. There are some speculations for the mechanism of benefit from sLVP. First, maximum resynchronization might be achieved with fusion conduction between the slow LV epicardial pacing and fast intrinsic conduction through the right bundle



<RAO View>

<LAO View>

Fig. 3. Implantation with cardiac resynchronization therapy system. Left and right panels show right anterior oblique (RAO) view, and left anterior oblique (LAO) view, respectively. Atrial and right ventricular (RV) leads were screwed-in at the high right atrial septum, and RV apex, respectively. Left ventricular (LV) quadripolar lead was placed into the lateral branch of coronary sinus. LAO = left anterior oblique; RAO = right anterior oblique.

branch. Second, sLVP may prevent RV pacing-induced dyssynchrony and allow for more simultaneous RV and LV electrical activation. Third, avoidance of RV pacing with sLVP may result in superior RV function when the conduction of right bundle branch is sufficiently preserved.

On the other hand, several studies reported that a high PVC burden and a wide PVC-QRS duration are associated with deterioration in cardiac function and a feasible response after a successful ablation. Carballeira et al. reported that a PVC-QRS duration longer than 153 ms might be useful predictor of PVC-induced cardiomy-opathy [5]. According to Penela et al., there is a positive correlation between PVC-burden and improvement in LVEF after successful ablation [4]. The benefit of PVC elimination in the present case seemed considerable based on a PVC burden of 25% and a PVC-QRS duration of 156 ms. Therefore, we took priority with PVC elimination over sLVP maintenance.

There is a possibility that the LVEF may have improved with adaptive CRT without PVC ablation. The effect of adaptive CRT alone was unknown because we performed the PVC ablation two weeks after the CRT device implantation. This point is one of the important limitations in this case report. The PVCs seemed malignant and to be a hindrance to improve cardiac function for the reasons mentioned above. We took priority with PVC elimination over observation of adaptive CRT effect for early recovery of the patient's cardiac function. In case of applying adaptive CRT alone without PVC ablation, using ventricular sense response (VSR) algorithm could be one of therapeutic options. However, this algorithm only works on ventricular events in the PR interval, when in a tracking mode. We did not apply the VSR algorithm, because almost PVCs were not accompanied with preceding atrial sensing events, indicating that this algorithm would not work on the PVCs.

The improvement in cardiac function in chronic phase might imply that the advantage of PVC elimination overweighed the disadvantage of loss of sLVP.

Conflict of interest

The authors declare no conflict of interest.

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