Cardiac Resynchronization Therapy and Atrioventricular Junction Ablation in a Patient With Heart Failure and Atrial Fibrillation: an Analysis of Activation Imaging

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(Received November 12, 2014; Accepted December 4, 2014)

Atrioventricular junctional ablation (AVJA) combined with biventricular (BiV) pacing is an effective treatment for atrial fibrillation (AF) and rapid ventricular response associated with heart failure. We report the case of a 64-year-old man with dilated cardiomyopathy and AF who underwent AVJA and BiV pacing. Activation imaging is a recently developed novel imaging technique that uses threedimensional speckle tracking echocardiography, which visualizes the left ventricular (LV) intra-ventricular propagation pattern of regional contractions. After AVJA and BiV pacing therapy, interventricular mechanical activation time (IVAT) quantified as the time difference from the QRS onset to regional activation in the 16 segments decreased from 209 \pm 92 msec to 177 \pm 88 msec. LV free-wall (basal anterior and lateral wall) IVAT values were shorter than those at baseline. Activation imaging appears to be a feasible technique for assessing LV mechanical dyssynchrony.

Key words: Dilated cardiomyopathy, three-dimensional echocardiography, speckle tracking, biventricular pacing

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INTRODUCTION

Atrioventricular junctional ablation (AVJA) combined with biventricular (BiV) pacing is an effective treatment for atrial fibrillation (AF) and rapid ventricular response associated with heart failure [1, 2]. Cardiac resynchronization therapy (CRT) by BiV pacing was established as therapy for advanced heart failure with left ventricular (LV) dyssynchrony. In patients with left bundle branch block (LBBB), who have the most typical indication for CRT, the initial contraction is observed in the mid to apical septum; next, the contraction propagates to the basal lateral wall, turning at the apex. This propagation pattern has been confirmed as the typical dyssynchrony pattern [3]. Recently, Seo et al. developed a novel activation imaging with threedimensional (3D) speckle tracking echocardiography (3D-STE), which visualizes the time required for the onset of regional contraction from QRS onset based on electro-mechanical coupling [4]. We report the case of a 64-year-old man with dilated cardiomyopathy and AF who underwent AVJA and BiV pacing. Activation imaging with 3D-STE clearly revealed the effectiveness of BiV pacing.

CASE REPORT

A 64-year-old man was referred to our hospital because of heart failure and recurrent ventricular tachycardia (VT) with AF. At age of 50, he had a dyspnea during physical activity; and was diagnosed with dilated cardiomyopathy. He was previously ad-

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mitted to a local hospital because of sudden acute chest pain. Coronary angiography was performed and total occlusion in the right coronary artery was detected. He underwent percutaneous coronary intervention (PCI) and a drug eluting stent was implanted. He had continuous hemodiafiltration because of the contrast-induced nephropathy that occurred after PCI. Five days after PCI, the patient had sudden AF and sustained VT, and direct current cardioversion was performed successfully. VT recurred 9 days after the first VT and atrial flutter (AFL) appeared, and the patient was transferred to our hospital for further therapy.

At admission, patient's body mass index was 29.6 kg/m², blood pressure was 100/80 mmHg, and heart rate was 88 beats/min. No rales or rhonchi were heard over the lung fields. A grade III/VI systolic murmur was audible at the apex. His abdominal examination was normal. He did not have pitting edema in either legs. Treatment included amiodarone, beta-blocker, loop diuretics, aspirin, clopidogrel, statin, and anti-diabetics. The initial laboratory studies indicated renal dysfunction (blood urea nitrogen, 72.3 mg/dL; creatinine, 3.79 mg/dL), an elevated plasma level of B-type natriuretic peptide (1234.1 pg/mL), and hemoglobin of 13.6 g/dL; other labora-

tory data remained normal.

An electrocardiogram (Fig. 1a) showed AF and complete LBBB (QRS interval, 200 msec), and a chest radiograph revealed a cardiothoracic ratio of 67% (Fig.2). Transthoracic echocardiography demonstrated severe LV dysfunction (Table 1). The LV end-diastolic diameter was 86 mm, LV endsystolic diameter was 80 mm, LV end-diastolic volume was 485 mL, and LV end-systolic volume was 433 mL with a LV ejection fraction of 11%. Left atrial diameter was 67 mm. Moderate mitral regurgitation and mild tricuspid regurgitation were demonstrated. The estimated pulmonary artery pressure was 43 mmHg. Because almost flat motions of the interventricular septum (IVS) and posterior wall were observed in the M-mode image (Fig. 3), the septal to posterior wall motion delay was not measured. Activation imaging was performed using the ArtidaTM ultrasound system (Toshiba Medical Systems Co., Tochigi, Japan). Regional deformation was measured by area change ratio (ACR) obtained from the area tracking method by 3D-STE. Onset of the regional activation was defined as the time at which the ACR value exceeded 25% of the maximum ACR value based on the previous validation study against electrical activation mapping [4].

	Before CRT	After CRT
LV end-diastolic diameter (mm)	86	86
LV end-systolic diameter (mm)	80	78
LV end-diastolic volume (mL)	485	448
LV end-systolic volume (mL)	433	359
LV ejection fraction (%)	11	20
LA diameter (mm)	67	65
Interventricular mechanical activation time (msec)	209±92	177±88

Table 1. Echocardiographic findings before and after cardiac resynchronization therapy (\mbox{CRT})

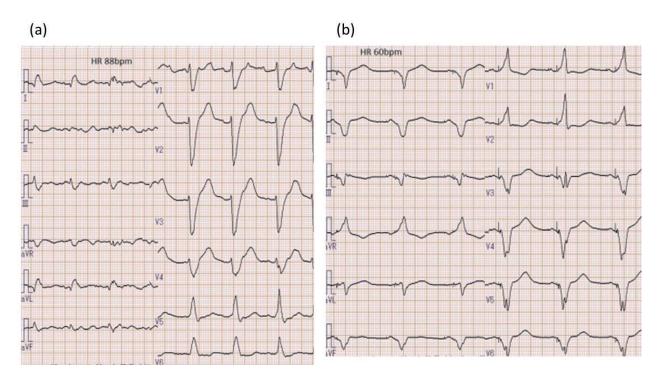


Fig. 1. Electrocardiogram (ECG).

(a) On admission, the ECG showed atrial fibrillation and complete left bundle branch block. (b) After atrioventricular junctional ablation and biventricular pacing combined with cardioverter-defibrillator were performed, the pacing rate is set to 60 beats/min.



Fig. 2. Chest radiograph. The cardiac size is enlarged and the cardiothoracic ratio is 67%.

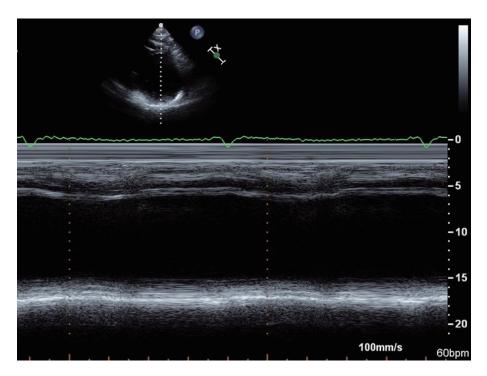


Fig. 3. M-mode echocardiogram.

On admission, the transthoracic echocardiography demonstrated severe left ventricular dysfunction. Almost flat motions of the interventricular septum and posterior wall were observed on the M-mode image.

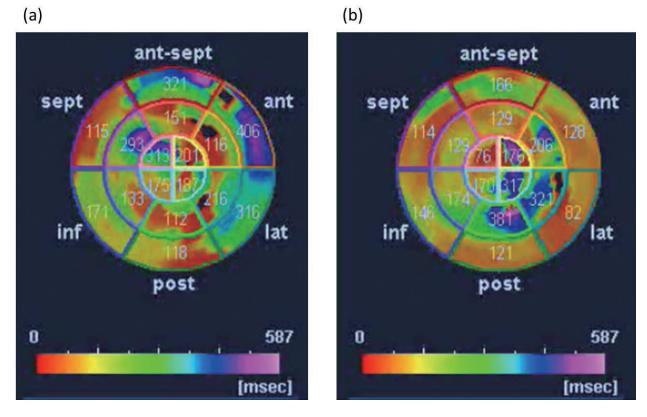


Fig. 4. Activation imaging (a) before and at 3-months after cardiac resynchronization therapy (CRT) (b). The numbers indicate the interventricular mechanical activation time (IVAT) quantified as the time difference from the QRS onset to the regional activation in the 16 segments. After CRT, the free-wall (basal anterior and lateral wall) IVAT values are shorter than those at baseline.

Interventricular mechanical activation time (IVAT) was quantified as the time difference from the QRS onset to the regional activation in the 16 segments, and the mean activation time was 209 ± 92 msec (Fig. 4a). Propagation from the mid-septum to the lateral wall through the apex was detected. Activation did not directly propagate from the septum to the lateral wall through the basal anterior wall.

Treatment included an increased dose of amiodarone (from 200 to 400mg/day) with an angiotensin II receptor blocker for VT as tolerated. Since frequent episodes of AF/AFL were observed, AVJA and BiV pacing combined with a cardioverterdefibrillator was performed (Fig. 1b). Written informed consent was obtained from the patient. LV pre-activation of 30 msec for BiV pacing was programmed to maximize the LV outflow velocity integral by pulsed wave Doppler imaging. The patient's clinical course was stable, and the plasma level of B-type natriuretic peptide decreased to 638.3 pg/mL. After 3 months of treatment, the LV end-diastolic diameter was 86 mm, end-systolic diameter was 78 mm, LV end-diastolic volume was 448 mL, and LV end-systolic volume was 359 mL with an LV ejection fraction of 20% (Table 1). The LV endsystolic volume was decreased to 17% compared to the baseline. The estimated pulmonary artery pressure was 31 mmHg. IVAT by activation imaging decreased to 177 ± 88 msec (Fig. 4b). Propagation from the mid-septum to the lateral wall through the basal anterior wall was detected.

DISCUSSION

CRT is for advanced heart failure with LV dyssynchrony. Compared with two-dimensional STE, 3D-STE provides more accessible images for analyzing LV dyssynchrony by showing temporal changes of strain distribution over the entire LV chamber [5-7]. Activation imaging can visualize the intra-ventricular propagation pattern of regional contractions, and reliability was validated in comparison studies on the electrical 3D mapping system. The concept of activation imaging method was to visualize the wavefront image of mechanical activation modeled after electrical mapping systems. The system was set to present the onset of regional wall deformation most accurately in comparison with studies using 3D electrical voltage mapping systems [4]. Various propagation patterns of LV activation deserve special attention for predicting CRT response. In the present case, propagation from the mid-septum to the lateral wall through the apex was detected by activation imaging, and the activation did not directly propagate from the septum to the lateral wall through the was anterior wall. After CRT, propagation from mid-septum to lateral wall through basal anterior wall was detected and IVAT was decreased.

The IVS motion in LBBB has been reported as a key predictor of the CRT response. The early displacement of IVS correspond to the septal flash [8, 9]. Despite the higher temporal resolution, standard M-mode imaging has limitations to detect the septal flash, as observed in the present case. Activation imaging with 3D-STE may be a feasible modality for assessing LV dyssynchrony and predicting CRT effects.

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