Improved Septal Contraction and Coronary Flow Velocity After Cardiac Resynchronization Therapy Elucidated by Strain Imaging and Pulsed Wave Doppler Echocardiography

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Abstract

Objectives. The effects of cardiac resynchronization therapy (CRT) with various atrioventricular conduction delay settings were investigated on cardiac hemodynamic changes involved in coronary flow velocity using color and pulsed wave Doppler modalities and myocardial regional contractility using a novel echocardiographic technique (strain imaging).

Methods. Seven patients with advanced heart failure (left ventricular ejection fraction $<35\%$) and left bundle branch block (QRS ≥ 140 msec) were treated with CRT. Color and pulsed wave Doppler imaging were performed from the apical four-chamber view to examine the cardiac functions such as stroke volume, cardiac output, mitral regurgitant volume and coronary flow velocity. Strain imaging was performed to quantify the asynchrony of both intraventricular and interventricular time delay between the septum and left ventricular free wall (posterior wall) and to assess the regional contractile function. Wall motion was also evaluated.

Results. Intraventricular and interventricular asynchrony were improved from 173 ± 18 to 60 ± 6 msec, and 69 ± 25 to 12 ± 3 msec, respectively. Stroke volume (55.2 ± 6.2 to 76.8 ± 10.8 m*l*; 39% up), cardiac output (3.9 ± 0.3 to 5.4 ± 0.5 *l*/min; 38% up) and coronary flow velocity (24 ± 3 to 36 ± 5 cm/sec; 50% up) were greatly increased and mitral regurgitant volume (59.7 ± 18.0 to 38.9 ± 11.3 m*l*; 35% down) was clearly decreased. Septal wall shortening was greatly increased from $10.2 \pm 2.3\%$ to $17.0 \pm 1.8\%$ and septal wall motion (radial thickening) was also improved simultaneously. Atrioventricular interval settings influenced all above parameters.

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Conclusions. CRT improved the cardiac hemodynamics involved in coronary flow significantly due to both resynchronization of inter and intra asynchrony, and improvement of the regional myocardial contraction in patients with severe congestive heart failure and complete left bundle branch block.

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Key Words

■**Cardiac surgery** ■**Doppler ultrasound (strain imaging, coronary flow velocity)**
■**Heart failure (cardiac resynchronization therapy)** ■**Myocardial contraction (regional)**

INTRODUCTION

Cardiac resynchronization therapy (CRT) is a recent advance in non-pharmacological therapy for patients with severe heart failure with electro-mechanical delay. The principle of this therapy is to resynchronize the timing of the left ventricular wall contraction. Prior studies reported improved global left ventricular systolic function[1−4], reduced pulmonary capillary wedge pressure[4], and decreased mitral regurgitation[5]. CRT also is effective in regressing left ventricular remodeling, diminishing energy cost, and is more powerful than medical therapy alone[6,7]. As a result of contractile resynchronization, CRT improves the entire left ventricular systolic function, but whether left ventricular myocardial regional contractility and coronary flow velocity are improved or not, is unknown. Several studies[8,9] concerned with the optimal settings for atrioventricular (AV) conduction delay rather than cardiac hemodynamic change have been reported, but no studies have discussed the relationship between AV conduction delay and coronary flow during CRT.

The present study investigated both hemodynamic changes involved in coronary flow velocity and myocardial regional contractility before and after CRT at various AV conduction delay settings.

SUBJECTS AND METHODS

Patients

Seven consecutive patients (three males and four females, mean age 81.0 ± 5.0 years) with an implanted bi-ventricular pacemaker were evaluated. All patients had severe heart failure characterized by left ventricular ejection fraction less than 35% and QRS wave complex duration longer than 140 msec with left bundle branch block pattern, and were still symptomatic (New York Heart Association class Ⅲ or Ⅳ). All patients underwent coronary angiography and none had coronary stenosis of greater than 50%. The etiologies of congestive heart failure were ischemic heart disease in two patients, idiopathic dilated cardiomyopathy in two, hypertensive cardiomyopathy in two and amyloidosis cardiomyopathy in one. All patients were hospitalized for congestive heart failure at least twice in the year. Pharmacological management included diuretics, angiotensin receptor antagonists and beta-adrenergic blocking agents at the maximum tolerated doses in all patients, spironolactone in three and pimobendan in three. After recognizing clinical stability for at least 3 months, a bi-ventricular pacemaker was implanted and the average of the QRS complex duration significantly decreased from 167 ± 20 to 133 ± 10 msec ($p < 0.02$; **Table 1**).

Bi-ventricular pacemaker implantation

Pacemaker catheters were inserted through the subclavian vein and the right ventricle, and right atrium pacing leads were placed at the right atrial appendage to pace the right atrium and at the right ventricle apex to pace the right ventricle. The left ventricular pacing lead was inserted through the coronary sinus into the postero-lateral cardiac vein with the help of a venogram in all patients. The bi-ventricular devices used were PULSAR™ MAX_DR, GUIDANT Japan K.K. in one patient, Actros DR, BIOTRONIK GmbH & Co. in four and Talent DR, ELA MEDICAL in two. After implantation, the AV interval (AV conduction delay: AV-) was changed based on the following four conditions, baseline (pacing off) and during CRT (AV-50, 100 and 150 msec).

Standard echocardiography

Standard echocardiography, including Doppler studies, was performed with a Vivid 7 (GE-YOKOKAWA medical system) equipped with a cardiac M3S (1.9/4.0 MHz) tissue harmonic transducer and a 7S (6.7 MHz) high frequency transducer. Left ventricular ejection fraction (LVEF; %) was assessed using the bi-planar Simpson's method, and left ventricular stroke volume (LVSV

Table 1 Clinical characteristics of the patients

Patient No.	Age (yr)	Sex	Diagnosis	LVEF (%)	QRS width (msec)	
					Before CRT	After CRT
1	79	Female	A	29	160 (CLBBB)	128
2	81	Male	Id	22	155 (CLBBB)	151
3	74	Male	I	18	197 (CLBBB)	160
4	89	Female	Id	19	182 (CLBBB)	116
5	86	Male	I	35	162 (CLBBB)	127
6	78	Female	H	25	159 (CLBBB)	120
7	80	Female	H	27	182 (CLBBB)	158
Mean±SD	81±5			25±6	167±20	133±10*

*$p<0.02$.
LVEF=left ventricular ejection fraction; CRT=cardiac resynchronization therapy; A=amyloidosis cardiomyopathy; I=ischemic heart disease; H=hypertensive cardiomyopathy; Id=idiopathic dilated cardiomyopathy; CLBBB=complete left bundle branch block.

; m*l*) and cardiac output (CO; *l*/min) were both assessed by pulsed wave Doppler echocardiography using the apical two-chamber view. The mitral regurgitation volume (MRV; m*l*) was also assessed by pulsed wave Doppler echocardiography using the apical four-chamber view. MRV is expressed as follows:

MRV (m*l*) = Left ventricular inflow volume per beat − LVSV

Left ventricular inflow volume per beat (m*l*) = $3.14 \times (Rm/2)^2 \times TVIm$

Where Rm (cm^2) is the mitral ring diameter, and TVIm (cm) is the time velocity integral of the trans-mitral flow.

Coronary flow velocity (CFV; cm/sec) of the left anterior descending artery (LAD) was assessed by a transthoracic echo Doppler technique using a 7S high frequency transducer.

Strain imaging

Myocardial pulsed wave Doppler velocity profiles were recorded from the apical two-chamber view with adjustments to filter frequency, gain settings, pulse repetition frequency and color saturation and at the highest possible frame rate (≥ 100 frames/sec). The images were digitized and strain imaging (SI) was analyzed on line. SI is a novel technique that can calculate the compression and expansion of the myocardial regional length quantitatively. SI (%) is expressed as follows:

$SI(\%) = dX/X$

Where dX is the changed myocardial length, and X is the original myocardial length.

Sample volumes (2 × 6 mm) were placed among the basal segment of the interventricular septum (IVS), mid segment of the IVS and basal segment of the posterior. The strain curves of the three segments were examined and both peak systolic strain values and the time (msec) until the peak systolic strain from the QRS complex throughout a cardiac cycle were compared. Those values in other regions of the free wall were also evaluated, but there were no significant differences in the other regions compared with the posterior region. In this study, posterior values were used as representative of the free wall. At least 3 consecutive beats of sinus rhythm were measured in a blinded manner and an average value was obtained.

Study protocol

Investigations were performed just before discharge (mean period after CRT implantation; 9 ± 2 days) in all patients in stable condition. This study evaluated the following factors before and after CRT: intraventricular and interventricular mechanical delay; cardiac hemodynamic changes; and myocardial shortening and radial thickening of regional myocardium.

The intraventricular delay (msec) between the basal IVS and the basal posterior were evaluated as the time lag until peak systolic strain from the QRS complex. The interventricular delay between the left and right ventricular contractions were evaluated as the time lag until the peak velocity of each Doppler wave. Right ventricular ejection flow was assessed by pulsed wave Doppler echocardiography using the parasternal short-axis view of the aortic level.

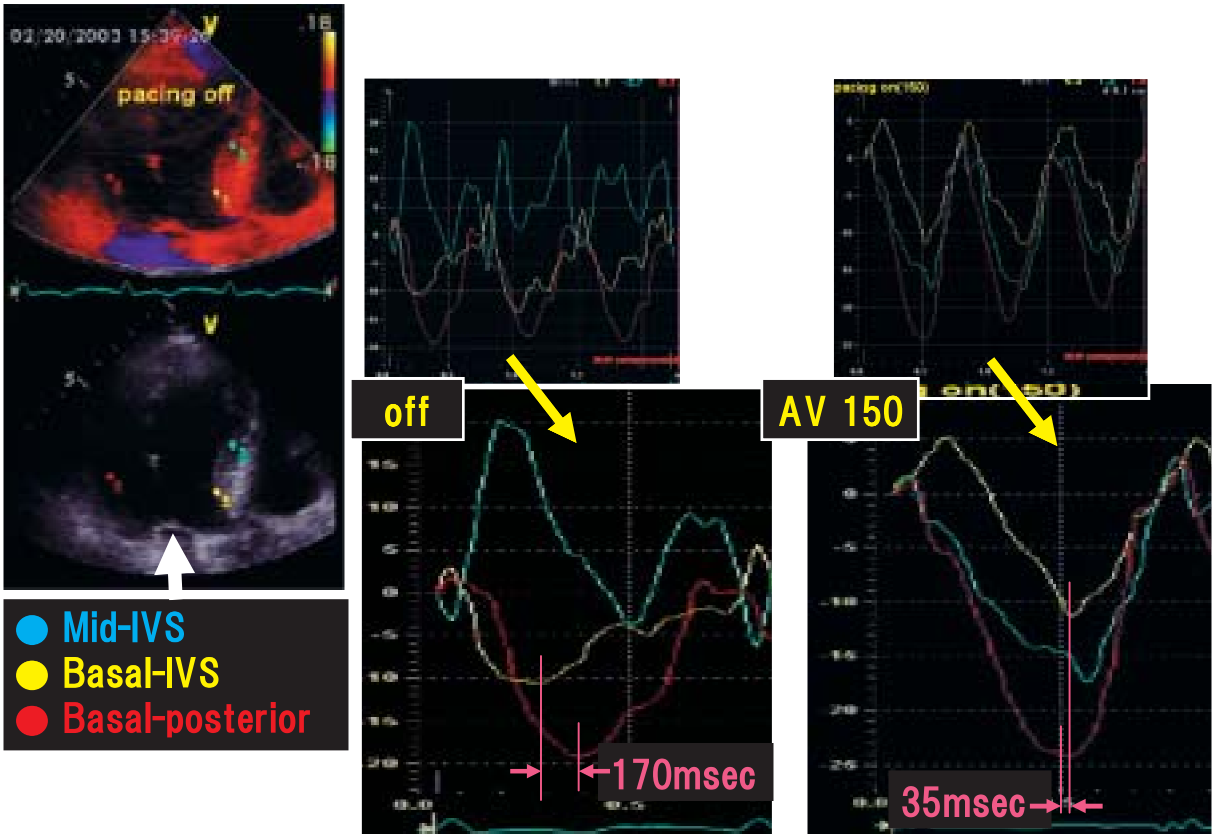

Fig. 1 Mechanical delay before and after cardiac resynchronization therapy
Typical images from the intraventricular delay measurements in the three myocardial regions (basal and mid IVS, basal posterior) using strain imaging before and during CRT (AV-150 msec). The time lag of the two regions (between basal IVS and posterior) until peak systolic strain during CRT (AV-150 msec; 35 msec) shortened to less than the baseline value (170 msec). Wall motion of mid IVS was improved to hypokinesis from dyskinesis by CRT.
IVS = interventricular septum; AV = atrioventricular conduction delay. Other abbreviation as in Table 1.

All echocardiographic Doppler values such as LVSV, CO, LVEF, MRV and CFV were measured at baseline and during CRT (AV-50, 100 and 150 msec). The cardiac hemodynamic effect of the CRT was measured as a cardiac hemodynamic ratio (%; CRT values/baseline value × 100).

Myocardial shortening was evaluated as the peak systolic strain value. Radial myocardial contraction was evaluated by the M-mode method of the parasternal long-axis view as percentage wall thickening. The percentage wall thickening is expressed as follows:

Percentage wall thickening = (EST − EDT)/EDT × 100

Where EST is the end-systolic thickness, and EDT is the end-diastolic thickness.

After continuation of the hemodynamic stability in each of the four conditions (baseline, AV-50, 100 and 150 msec) for at least 15 min, the results of these examinations were studied. Informed consent was obtained from all patients and this study protocol was approved by the hospital's Ethics Committee.

Statistical analysis

Data are presented as mean ± standard deviation (SD). Repeated analysis of variance (ANOVA) was used to compare cardiac hemodynamic (pulse Doppler) values, systolic strain values (%) and radial thickening (%) of both basal IVS and posterior wall at baseline and during CRT (AV-50, 100 and 150 msec). A p value < 0.05 was considered statistically significant.

RESULTS

Intraventricular and interventricular delays

Typical images from the intraventricular delay measurements in the three myocardial regions

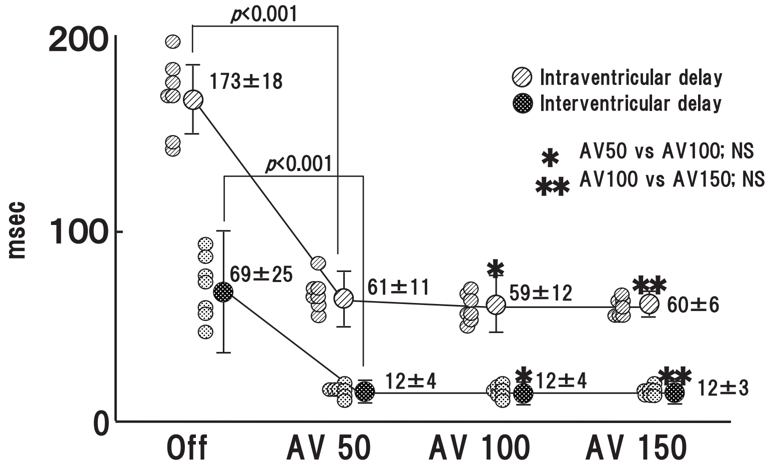

Fig. 2 Intraventricular and interventricular delays under the four conditions
Intraventricular delay improved significantly from the baseline value, as did interventricular delay. However, neither mechanical delay showed any significant differences between the three CRT conditions.
Abbreviations as in Table 1, Fig. 1.

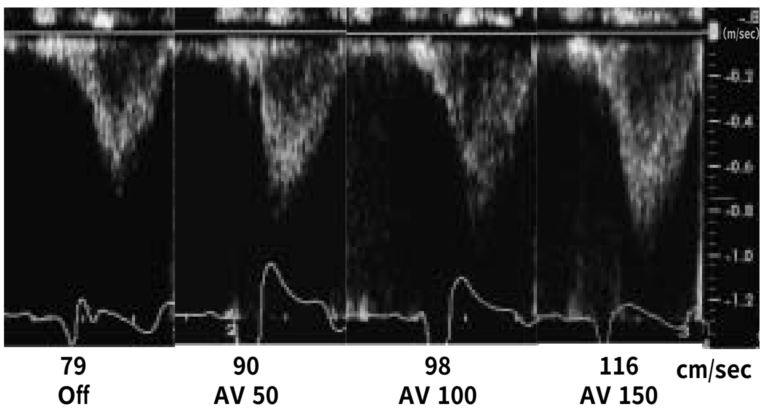

Fig. 3 Pulsed wave Doppler tracings at the left ventricular outflow tract
During CRT (AV-50, 100 and 150 msec), peak velocity accelerated gradually from 79 to 90, 98 and 116 cm/sec, respectively.
Abbreviations as in Table 1, Fig. 1.

(basal and mid IVS and basal posterior) using SI before and during CRT (AV-150 msec) are illustrated in **Fig. 1**. During CRT, wall motion of the mid-IVS improved to hypokinesis from dyskinesis. Intraventricular delay of the two regions (between the basal IVS and posterior) during CRT (AV-150 msec; 35 msec) shortened to less than the baseline value (170 msec).

Fig. 2 shows the intraventricular and interventricular delays of the two regions under the four conditions. CRT (AV-50,100 and 150 msec) improved intraventricular delay significantly from a baseline value of 173 ± 18 to 61 ± 11, 59 ± 12 and 60 ± 6 msec, respectively ($p < 0.001$), and also improved interventricular delay significantly from a baseline value of 69 ± 25 to 12 ± 4 , 12 ± 4 and 12 ± 3 msec, respectively ($p < 0.001$). However, neither mechanical delay showed any significant difference between the three CRT conditions.

Left ventricular systolic function

Fig. 3 shows a typical flow pattern of the left ventricular outflow tract. During CRT (AV-50, 100 and 150 msec), peak velocity accelerated from 79 to 90, 98 and 116 cm/sec, respectively. **Fig. 4** shows a typical image of LVEF measurements using the bi-planar Simpson's method. During CRT (150 msec), LVEF increased from 28% to 40%. Wall motion of the mid-IVS improved to hypokinesis from dyskinesis after CRT (← *arrows*).

Fig. 5 shows a graph of the hemodynamic change ratio. During CRT (AV-50 msec), LVSV, CO and LVEF increased significantly from their baseline values (55.2 ± 6.2 m*l*, 3.9 ± 0.3 *l*/min and 28 ± 5%, respectively) to 68.5 ± 6.0 m*l*, 5.0 ± 0.4 *l*/min and 33 ± 6%, respectively ($p < 0.01$, $p < 0.001$, $p < 0.001$, respectively). During CRT (AV-100 and 150 msec), the respective values of LVSV (74.1 ± 9.6, 76.8 ± 10.8 m*l*), CO (5.4 ± 0.4, 5.4 ± 0.5 *l*/min) and LVEF (40 ± 4%, 41 ± 5%) showed greater improvement ($p < 0.05$, $p < 0.05$, respectively) over CRT (AV-50 msec). However, LVSV, CO and LVEF showed no significant differences between CRT (AV-100 msec) and CRT (AV-

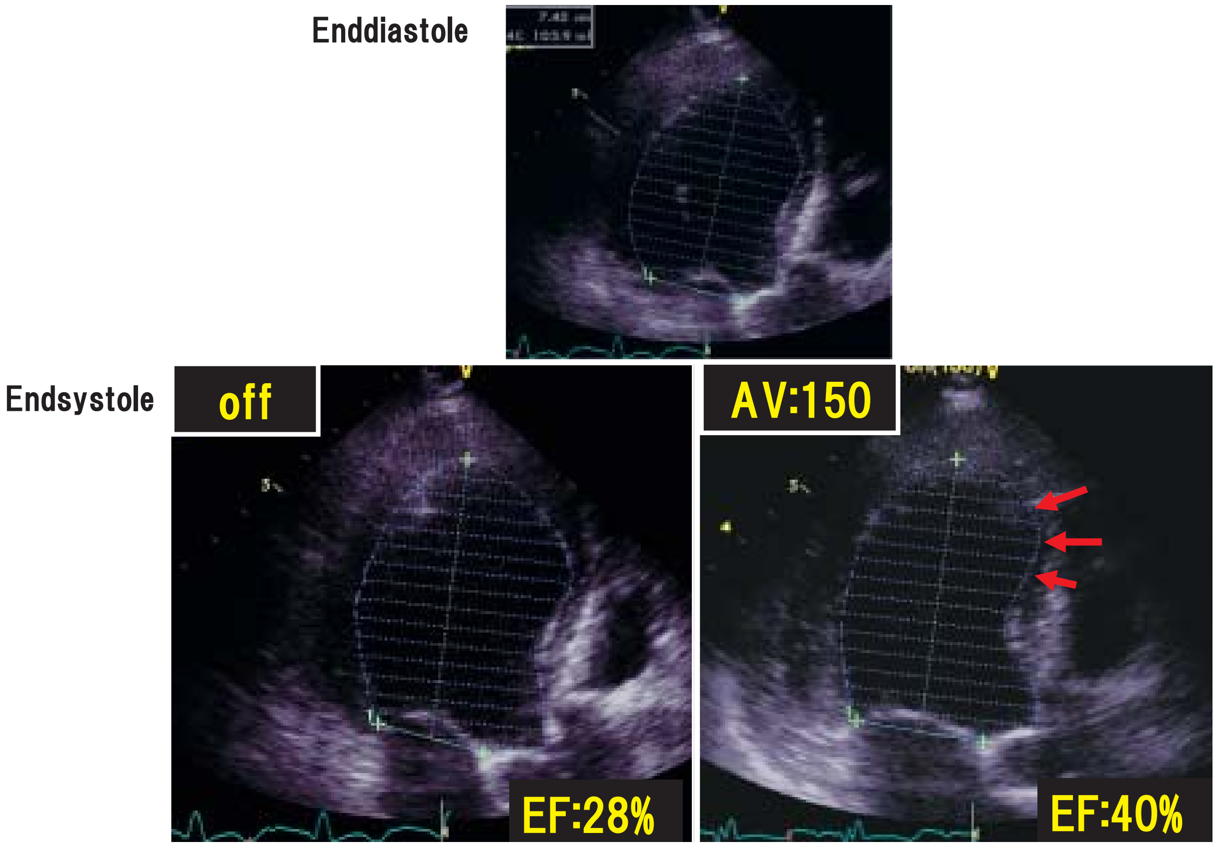

Fig. 4 Left ventricular ejection fraction using the bi-planar Simpson's method
During CRT(150 msec), LVEF increased from 28% to 40%.
Abbreviations as in Table 1, Fig. 1.

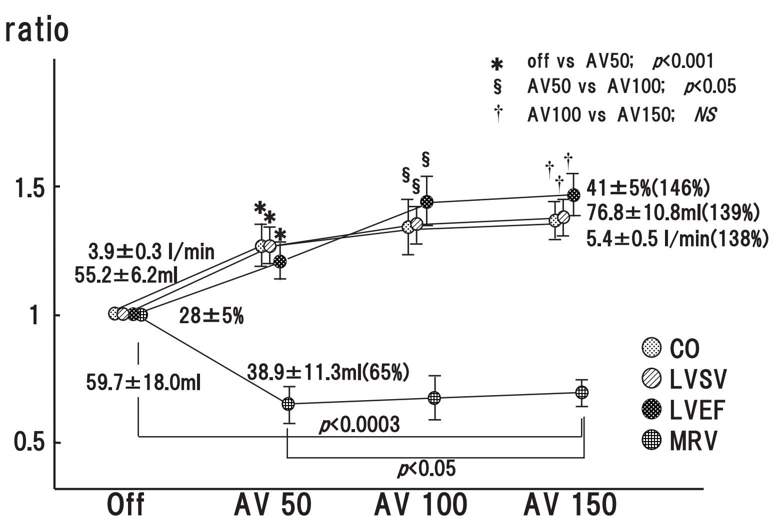

Fig. 5 Changes of cardiac hemodynamics before and during CRT
During CRT, LVSV, CO and LVEF increased significantly from their baseline values. During CRT (AV-100 and 150 msec), all values showed greater improvement over CRT(AV-50 msec). However, no significant differences were observed between CRT(AV-100 msec) and CRT(AV-150 msec). MRV during CRT(AV-50 msec) significantly decreased from the baseline value ($p < 0.0003$). However, no significant differences were observed between the three CRT conditions.
LVSV = left ventricular stroke volume; CO = cardiac output; MRV = mitral regurgitation volume. Other abbreviations as in Table 1, Fig. 1.

150 msec).

On the other hand, MRV during CRT(AV-50 msec) significantly decreased from 59.7 ± 18.0 to 38.9 ± 11.3 m*l* ($p < 0.0003$). However, MRV showed no significant differences between the three CRT conditions.

Coronary flow velocity in the left anterior descending artery

Typical images of color Doppler signals and

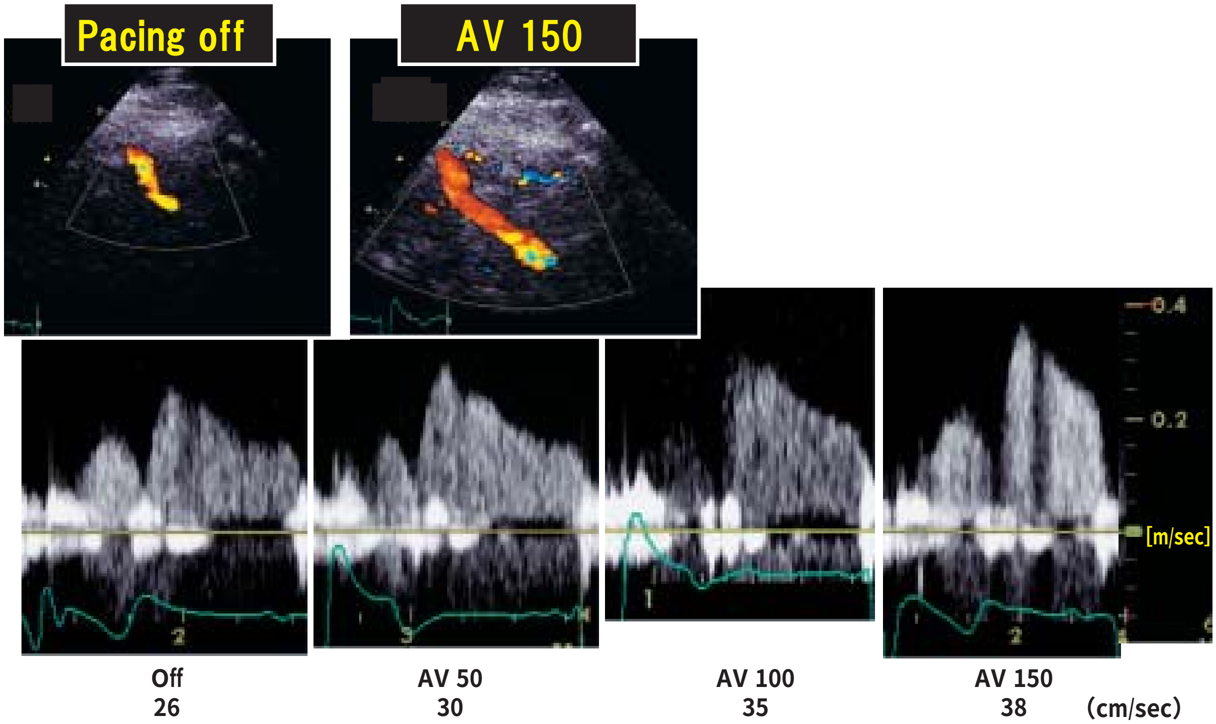

Fig. 6 Color Doppler signals and pulsed wave Doppler tracings in the left anterior descending coronary artery
Peak velocity during CRT(AV-50, 100 and 150 msec) accelerated from 26 to 30, 35 and 38 cm/sec, respectively.
Abbreviations as in Table 1, Fig. 1.

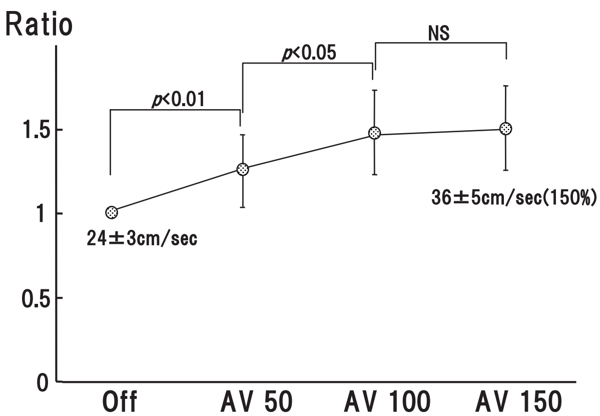

Fig. 7 Coronary flow velocity in the left anterior descending coronary artery
Coronary flow velocity increased significantly from the baseline values with CRT. CRT(AV-100 and 150 msec) showed greater improvement over CRT(AV-50 msec). However, no significant difference was observed between CRT(AV-100 msec) and CRT(AV-150 msec).
Abbreviations as in Table 1, Fig. 1.

pulsed Doppler waves of LAD flow are shown in **Fig. 6**. Peak velocity during CRT(AV-50, 100 and 150 msec) accelerated from 26 to 30, 35, 38 cm/sec, respectively.

Fig. 7 shows the coronary flow velocity change ratio. During CRT(AV-50 msec), CFV increased significantly from their baseline values (24 ± 3 cm/sec) to 31 ± 4 cm/sec ($p < 0.01$). During CRT (AV delay-100 and 150 msec), CFV (35 ± 5, 36 ± 5 cm/sec, respectively) showed greater improvement over CRT (AV-50 msec). However, CFV showed no significant differences between CRT (AV-100 msec) and CRT (AV-150 msec).

Myocardial regional contraction

CRT improved both myocardial shortening and radial contraction. During CRT (AV-50 msec), myocardial shortening of the basal IVS improved significantly from the baseline value (10.2 ± 2.3) to 13.5 ± 3.0 ($p < 0.05$). During CRT(AV-100 and 150 msec), values of 16.7 ± 1.6 and 17.0 ± 1.8, respectively, showed greater improvement over CRT(AV-50 msec) ($p < 0.05$). However, no significant difference was observed between CRT(AV-

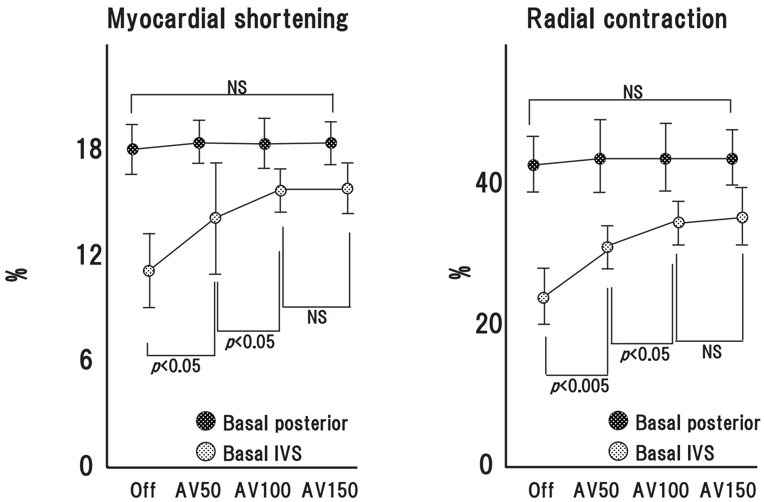

Fig. 8 Myocardial shortening and radial thickening

Left: Myocardial shortening of the basal IVS improved significantly from the baseline value with CRT. During CRT(AV-100 and 150 msec), greater improvement was shown over CRT(AV-50 msec)($p < 0.05$). However, a significant difference was observed between CRT(AV-100 msec)and CRT(AV-150 msec). Myocardial shortening of the basal posterior did not change with or without CRT, regardless of the AV conduction delay.

Right: Radial thickening of the basal IVS improved significantly from the baseline value with CRT. During CRT(AV-100 and 150 msec), greater improvement was shown over during CRT(AV-50 msec)($p < 0.001$). However, as with myocardial shortening, no significant difference was observed between CRT(AV-100 msec)and CRT(AV-150 msec). Radial thickening of the basal posterior also did not change with or without CRT, regardless of the AV conduction delay.

Abbreviations as in Table 1, Fig. 1.

100 msec)and CRT(AV-150 msec). Myocardial shortening of the basal posterior did not change with or without CRT, regardless of the AV conduction delay(**Fig. 8−left**).

Similarly, during CRT(AV-50 msec), radial contraction of the basal IVS improved significantly from the baseline value(22.3 ± 3.5)to 30.0 ± 2.9 ($p < 0.005$). During CRT(AV-100 and 150 msec), values of 34.0 ± 2.5 and 34.3 ± 3.1, respectively, also showed greater improvement over CRT(AV-50 msec)($p < 0.05$). However, no significant difference was observed between CRT(AV-100 msec) and CRT(AV-150 msec). Radial thickening of the basal posterior also did not change with or without CRT, regardless of the AV conduction delay(**Fig. 8−right**).

DISCUSSION

The mechanism by which CRT improves mechanical left ventricular function in patients with heart failure and ventricular asynchrony is not completely understood[10]. Our study was aimed at assessing the relationships between acute hemodynamic improvement and myocardial regional contraction after CRT at varying AV conduction delays in patients with severe heart failure and complete left bundle branch block(CLBBB).

In this study, after CRT, all seven patients showed significant improvement in the electrical delay and mechanical delay and were consequently discharged. The degree and range of mechanical delay is the most powerful predictor[11,12]for cardiac functional improvement after CRT. The optimal pacing site is the most delayed wall[13].

Our seven patients had left ventricular septal asynchrony over a wider range and myocardial viability, and whole cardiac pump function improved after CRT in all patients. Although many reports have evaluated the degree of the mechanical delay and improvement after CRT using the tissue Doppler imaging(TDI)method[12−17], this study used the SI method that can evaluate the initiation of regional wall contraction directly and quantitatively regardless of cardiac translation and tethering[18−20]. Due to these reasons, the SI method was superior for the detection of CRT candidates.

Cardiac hemodynamics in our patients demonstrated improvement in the LVSV, CO, and MRV by 30−40%. Previously, patients who responded to CRT to improve the cardiac function had improved

CO, pulmonary arterial wedge pressure, and MRV by 25−40%[1−4,12]. Similar results were shown in this study. The cardiac output improves because of reduction or disappearance of diastolic mitral regurgitation when the pacing occurs just after the end (AV interval; 75−80 msec) of atrial kick[8,9]. We inferred that the cause of reduced CO during CRT (AV-50 msec) is the time phase at the middle of the left atrial kick and the filling volume is thus insufficient.

Coronary flow of the LAD was measured as an index for hemodynamics evaluation after CRT. Transthoracic evaluation of coronary flow correlates significantly with the value measured using a Doppler flow wire during coronary angiography and its reliability is thus high[21,22]. In this study, the CFV improved equivalently to or more than the LVSV (150% vs 139%) after CRT (AV-150 msec). In an animal experiment in which a pacemaker was implanted to cause CLBBB reversely, the myocardial internal pressure gradient of the IVS changes and myocardial internal pressure during diastole increased equivalently to or more than that during systole in CLBBB. Also, the flow velocity of the septal branch of LAD decreases significantly ($p < 0.05$)[15]. In clinical patients with stenosis at the coronary artery of LAD (≥50%) with CLBBB, the ischemic detectable sensitivity of the LAD was favorable, but the specificity significantly decreased[23−25] as compared to patients without CLBBB. CLBBB can disturb the coronary perfusion of the LAD through shortening of the diastolic flow duration[26]. In the absence of stenosis at the coronary artery, the improvement of the coronary flow velocity is assumed to parallel the improvement of the LVSV as a rule. However, the improvement of the LAD flow velocity was equivalent to or more than the LVSV improvement in this study, possibly because the improvement of the IVS asynchrony may have normalized the septal myocardial internal pressure gradient and promoted the improvement of the LAD coronary flow.

Many studies have investigated wall motion quantitative analysis using strain imaging and strain rate imaging[15−17,19,20]. We evaluated the left ventricular shortening using strain imaging and left ventricular radial thickening using M-mode method. Although the shortening and radial thickening of the basal IVS improved significantly by CRT, those of the basal posterior did not improve. In the animal experiment with CLBBB the septal wall motion decreased significantly due to the change of myocardial internal pressure gradient at the CLBBB but the posterior wall motion did not change[23]. In this study, we considered that the improvement of the asynchrony after CRT might normalize the collapse of the myocardial internal pressure gradient and improve the septal wall motion. Although CRT is reported not to improve myocardial contraction[11,14], cases were evaluated by the TDI method. However, the TDI method is influenced by cardiac translation, so small change of regional improvement cannot be evaluated directly. We considered that strain imaging can evaluate such small changes directly and this method evaluated the septal contraction accurately. The degree of wall motion improvement was significantly small during CRT (AV-50 msec), presumably because the left ventricular preload was so small due to the middle of the atrial kick that improvement of LVSV decreased, based on Laplace's theorem[1].

Limitations

This study evaluated only seven patients with apparent asynchrony (most delayed wall) of the IVS. Regional and whole cardiac function improved in all seven patients after CRT. However, patients who do not respond to CRT are present among patients with severe CHF associated with CLBBB. Additional investigations must be done at the other asynchrony site and different pacing sites. In this study, the acute effectiveness of cardiac hemodynamics and regional myocardial contractile function after CRT was evaluated, but the relationship between the improvement of the acute effectiveness and the long-term prognosis was not evaluated. Among the three AV conduction delays (50, 100 and 150 msec), cardiac hemodynamics with AV delays (100 and 150 msec) showed greater improvement than that of AV-50 msec in this study, but the optimal AV conduction delays in various patients will differ.

CONCLUSIONS

CRT improved cardiac hemodynamics involved in coronary flow significantly due to both resynchronization of inter and intra asynchrony, and improvement of regional myocardial contraction in patients with severe congestive heart failure and

CLBBB. However, the optimal AV conduction delay settings must be considered carefully.

Acknowledgments

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要　約

完全左脚ブロックを有する重症心不全の心臓再同期療法後にStrainエコー法を用いて評価した中隔心筋収縮と冠血流速度の改善

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背景と目的：薬剤抵抗性の重症心不全患者に心臓再同期療法（CRT）が広く臨床応用されている．最近では組織ドップラー法を用いてCRTの適応の決定や効果判定が行われているが，今回我々は，Strainエコー法やパルスドップラー法を用いてCRTの条件設定が心機能，冠血流，そして局所心筋収縮に及ぼす影響を検討する．

方　法：対象は左脚ブロック（QRS幅≥140 msec）を有しCRTを行った重症心不全（左室駆出率＜35%）患者7例である．CRTにおける房室伝導時間の設定（50, 100, 150 msec）が心拍出量，僧帽弁逆流量や左前下行枝冠血流速度に及ぼす変化をパルスドップラー法を用いて測定するとともにstrainエコー法を用いて心室内と心室間の機械的な同期性および心筋局所収縮能に対する影響を検討した．

結　果：CRTにより心室内機械的遅延は173±18 msecからCRT後は60±6 msecへと有意に短縮し，心室間機械的遅延も同様に69±25から12±3 msecへと有意に短縮した．CRT前に比較してCRT後最大で1回拍出量は55.2±6.2から76.8±10.8 m*l*へ，心拍出量は3.9±0.3から5.4±0.5 *l*/minへ，左前下行枝冠血流速度は24±3から36±5 cm/secへと有意に増加し，僧帽弁逆流量は59.7±18.0から38.9±11.3 m*l*へと有意に減少した．また，CRT後最大で中隔心筋収縮率は10.2±2.3%から17.0±1.8%と有意に増大したが，後壁の収縮率に変化はなかった．房室伝導時間を変更することで中隔の局所心筋収縮能が改善し全体的な心機能も向上したうえ，冠血流速度も同様に改善した．

結　論：CRTは心臓全体のポンプ機能の効率を向上させるだけではなく，冠血流速度や局所心筋の収縮能も改善させることが示唆された．

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