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ORIGINAL ARTICLE

Effects of biventricular pacing on right ventricular function assessed by standard echocardiography

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Abstract

Background and aim: The aim of this study was to evaluate the short term effect of cardiac resynchronisation therapy (CRT) on right ventricular (RV) function assessed by standard echocardiography.

Methods: Data from 57 patients (54 men, 95%; three women, 5%), aged 66.4 ± 8.7 years with heart failure (HF) was analysed. All patients were in NYHA III–IV functional classes, despite optimal pharmacological treatment according to the current guidelines, had left ventricular ejection fraction ≤ 35% and QRS complex ≥ 120 ms in a standard electrocardiogram. At baseline and three months after CRT implantation the patients' histories were taken, an anthropometrical examination was made, laboratory tests including the level of NT-proBNP and electrocardiogram were performed, and echocardiographic examination was extended by tissue Doppler imaging techniques and complex RV evaluation.

Results: Three months after CRT implantation in the whole study group, the average NYHA functional class had decreased from 3.11 ± 0.28 to 2.25 ± 0.68 ($p < 0.001$), and the six-minute walk test distance had increased from 298.04 ± 107.42 m to 373.12 ± 127.15 m ($p < 0.001$). CRT improved RV systolic function in the whole study group. Tricuspid annular plane systolic excursion had increased from 13.95 ± 2.80 to 15.79 ± 2.33 mm ($p < 0.001$), and so likewise had systolic excursion velocity (S'), which rose from 8.84 ± 3.45 to 11.00 ± 3.43 cm/s ($p < 0.001$). Tricuspid regurgitation grade decreased from 2.02 ± 0.95 to 1.86 ± 0.91 ($p = 0.013$). RV systolic pressure decreased from 31.07 ± 20.43 to 27.75 ± 17.35 mm Hg ($p < 0.001$). RV fractional area change rose from 31.35 ± 10.30% to 35.40 ± 10.51% ($p < 0.001$).

Conclusions: This study showed that CRT improved RV systolic function evaluated with parameters assessed in standard echocardiographic examination three months after therapy initiation. The observed improvement was consistent among all applied echocardiographic parameters reflecting RV systolic function.

Key words: right ventricle, cardiac resynchronisation therapy, echocardiography, heart failure

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INTRODUCTION

Heart failure (HF) is one of the world's greatest public health problems with not only social but also large economic repercussions. A growing incidence and poor prognosis require development of new therapeutic strategies.

Cardiac resynchronisation therapy (CRT) is a well established treatment for HF with systolic dysfunction. CRT improves functional status, reverses left ventricular (LV) remodelling, and decreases morbidity and mortality in patients with HF [1]. Most of the studies assessing the effects of CRT have focused on LV function, as CRT was primarily designed to improve LV contractility and synchrony. Little is known about how CRT affects right ventricular (RV) function [2]. Some studies have suggested that the RV's role in HF has been underestimated [3]. RV dysfunction is an independent outcome predictor in patients with moderate to severe HF [4]. Some recently conducted studies have proved that the LV response to CRT is determined by the baseline RV function [5]. Impaired RV function may limit the beneficial effects of CRT. It is not clear if CRT improves RV function and what the precise mechanism of CRT's influence on RV is.

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The aim of this study was to evaluate the short term effect of CRT on RV function assessed by standard echocardiography.

METHODS

Patient population

The study group comprised 60 of patients who consecutively underwent implantation of a biventricular (BiV) pacemaker (CRT-P) or a defibrillator (CRT-D) for drug-refractory HF over a 12-month period. All patients were in New York Heart Association (NYHA) class III–IV, despite optimal pharmacological treatment according to the current guidelines, had left ventricular ejection fraction (LVEF) ≤ 35% and QRS complex ≥ 120 ms in a standard electrocardiogram (ECG). Patients in NYHA class IV could not have a history of hospitalisation due to HF exacerbation within a month prior to CRT implantation [6]. Patients with permanent atrial fibrillation (AF) were included in the study if more than 95% of ventricular pacing was achieved with pharmacotherapy. In cases where an adequate level of ventricular pacing was impossible to achieve with medication, atrioventricular (AV) junction ablation was performed. The study was approved by the local ethics committee and all patients provided informed written consent. Patients were followed-up for 12 weeks. Three patients died in the follow-up period — one due to stroke, one due to myocardial infarction and one for an unknown reason. Data from 57 patients (54 men, 95%; three women, 5%, aged 66.4 ± 8.7 years) with HF was analysed. Table 1 shows the demographic and clinical variables of the patient population.

Table 1. Clinical data and pharmacotherapy of the study group (n = 57) — data presented as mean value with standard deviation (SD) or patients' percentage share (%)

Age [years]	66.35 ± 8.69
BMI [kg/m^2]	25.82 ± 4.15
Male gender	54 (94.7%)
History of MI	64.9%
Diabetes	40.4%
Hypertension	63.2%
COPD	19.3%
Hypercholesterolaemia	77.2%
Anaemia	3.5%
Smoking	22.8%
Chronic renal disease	31.6%
Ischaemic background of HF	71.9%
History of CABG	14.0%
History of PCI	17.5%
Beta blockers	96.5%
ACEI/ARB	86.0%
Loop diuretics	87.8%
Digoxin	22.8%
Amiodarone	31.6%

BMI — body mass index; MI — myocardial infarction; COPD — chronic obstructive pulmonary disease; HF — heart failure; CABG — coronary artery bypass graft; PCI — percutaneous coronary intervention; ACEI — angiotensin converting enzyme inhibitor; ARB — angiotensin receptor blocker

Device implantation

All patients received CRT devices with leads placed transvenously. Patients in sinus rhythm received CRT-P with an atrial lead (setting: DDDR; n = 26). In the presence of permanent AF, CRT-P was placed without an atrial lead (setting: VVIR; n = 13). The system with combined cardioverter-defibrillator function (CRT-D; n = 18) was implanted in patients with a history of cardiac arrest and/or malignant ventricular arrhythmias. Atrial leads were placed in the right atrial (RA) appendage. RV lead was placed in the RV outflow tract position in 48 patients and in the RV apex in the other nine patients. RV lead apex position was present only in patients in whom CRT was an upgrade of a previously existing device. Fourteen patients had their previous devices upgraded: VVI — two (3.5%), DDD — nine (15.8%), and DDD-ICD — three (5.3%). The LV lead, guided by venography, was placed in a coronary sinus tributary in a stable lateral (n = 49) or postero-lateral (n = 8) position, with a < 3.5 V capture threshold. Frontal and sagittal chest X-rays were used to verify the lead position. AV delay remained standard programmed (100–130 ms for non-paced atrial rhythm/130–150 ms for paced atrial beat). In patients with continued ventricular conduction, the AV delay was shortened until the ventricles were consistently paced (four cases). Interventricular (VV) timing was left at a nominal value of 5 ms, with LV paced first, unless no signs of BiV stimulation in the body surface ECG were observed. VV was then changed then to elicit the picture of QRS fusion beats in ECG lead V1. Devices used in this study were Medtronic InSync III, St. Jude Medical Frontier and Biotronik LUMAX 300. During the follow-up period, there were no ATP-pacing therapies or ICD shocks in our study group.

Study design

Prior to and three months after CRT implantation (12–16 weeks), study participants were evaluated clinically by NYHA class and six-minute walk test (6-MWT). Their medical history including hospitalisation was taken and an echocardiographic exam was performed (GE-Vingmed Vivid 7 system; GE Vingmed Ultrasound, Horten, Norway). One month after CRT implantation, all patients reported for a routine check-up to assess wound and stimulation parameters. One patient with permanent AF who did not achieve 95% of ventricular stimulation had AV junction ablation performed. In this patient, follow-up was extended to three months after a successful ablation procedure. All patients undergoing CRT also underwent coronary angiography. An ischaemic background of HF was diagnosed when there was at least 50% stenosis of one or more

coronary artery branches or a patient had a history of coronary artery bypass graft or percutaneous coronary intervention.

Echocardiographic assessment

All echocardiographic studies were performed by the same physician and were reported as an average of his measurements. Presentations were acquired with a General Electric Healthcare Vivid 7 device and included typical long- and short-axis and apical views. Assessment of the right heart was performed in concordance with the current guidelines of the European Association of Echocardiography and the American Society of Echocardiography [7]. RA and RV linear dimensions were obtained from a four-chamber view from the apical window at end-diastole. RV free wall thickness was measured at end-diastole by M-mode from the subcostal window. RV systolic function was evaluated using tricuspid annular plane systolic excursion (TAPSE) and tissue Doppler-derived tricuspid lateral annular systolic velocity (S'). TAPSE was acquired in a standard apical four-chamber window, as the distance of systolic excursion of the RV annular segment along its longitudinal plane. The same window was used in S' measurements, with pulsed Doppler sample volume placed in the tricuspid annulus. Tissue Doppler velocities of the tricuspid annulus were also analysed to establish E' and A'. RV fractional area change (RV FAC) was obtained by tracing the RV endocardium in systole and diastole from the annulus to the apex along the free wall and from the apex to the annulus along the intraventricular septum. Trace was led along the free wall carefully, to avoid trabeculations. Right ventricle systolic pressure (RVSP) was calculated from tricuspid regurgitation (TR) velocity and right atrium pressure, which was estimated from the size and collapsibility of inferior vena cava (IVC) [7]. The colour Doppler jet size and jet area corrected for the receiving chamber size were used for assessment of the degree of TR. Analysis of all acquired images was performed offline using commercially available software EchoPAC PC version 6.00 (GE Vingmed Ultrasound).

Statistical analysis

Statistical analysis was performed with SAS System 9.1 (SAS Institute Inc., Cary, NC, USA). All parameters were tested for normal distribution with the Shapiro-Wilk test. The Student's t-test was applied to determine if the parameters' averages before and after the CRT introduction were significantly different. Statistical significance was considered when $p < 0.05$.

RESULTS

In the study group, mean NYHA class dropped and 6-MWT distance rose. The mean QRS duration was also seen to decrease (Table 2). Basic echocardiographic parameters at baseline and at three months follow-up are presented in Table 3.

Effects of CRT on RV function

After three months of follow-up, there was an improvement of RV systolic function in the whole study group. Significant improvements of RV function and size were noted. RV fractional area change and TAPSE increased. Peak systolic annular velocity increased. The major diameter of RA was reduced. The TR assessed with qualitative method decreased. There was also a significant reduction in RVSP (Table 4).

DISCUSSION

Very little data on the effect of CRT on RV systolic function has been published. The effect of CRT on RV function in the short period of time after therapy onset has been even less discussed. Donal et al. [8] found that CRT improves RV tissue

Table 2. Clinical outcomes after cardiac resynchronisation therapy implantation

	At baseline (n = 57)	At three month follow-up (n = 57)	P
New York Heart Association functional classification	3.11 ± 0.28	2.25 ± 0.68	< 0.001
Six-minute walk test distance [m]	298.04 ± 107.42	373.12 ± 127.15	< 0.001
QRS [ms]	184.23 ± 28.31	152.70 ± 19.11	< 0.001

Table 3. Basic echocardiographic parameters in the whole study group

Echocardiographic parameter	At baseline (n = 57)	At three month follow-up (n = 57)	P
Left ventricular end-diastolic diameter [mm]	73.33 ± 8.94	71.54 ± 9.87	0.005
Left ventricular end-systolic diameter [mm]	62.40 ± 10.03	60.60 ± 11.29	0.081
Left ventricular ejection fraction [%]	21.70 ± 4.81	26.05 ± 4.86	< 0.001
Left ventricular end-diastolic volume [mL]	244.30 ± 83.79	226.42 ± 88.61	< 0.001
Left ventricular end-systolic volume [mL]	192.79 ± 71.95	168.67 ± 76.40	< 0.001

Table 4. Echocardiographic parameters of right ventricle at baseline and at three month follow-up

Echocardiographic parameter	At baseline (n = 57)	At three month follow-up (n = 57)	P
RA minor dimension [mm]	37.67±7.39	36.42 ± 6.18	0.116
RA major dimension [mm]	49.79 ± 10.53	48.33 ± 9.47	0.040
RA end-systolic area [cm^2]	17.49 ± 6.30	17.06 ± 5.91	0.155
RV wall thickness [mm]	5.47 ± 1.20	5.12 ± 1.02	0.028
RV longitudinal dimension [mm]	25.35 ± 7.02	25.84 ± 6.37	0.342
RV basal dimension [mm]	28.74 ± 4.47	28.32 ± 4.24	0.266
RV mid cavity dimension [mm]	24.21 ± 4.85	23.75 ± 5.20	0.160
RV end-diastolic area [cm^2]	15.72 ± 5.20	15.62 ± 5.45	0.927
RV end-systolic area [cm^2]	10.94 ± 4.51	10.22 ± 4.25	0.007
RV fractional area change [%]	31.35 ± 10.32	35.40 ± 10.51	< 0.001
TAPSE [mm]	13.95 ± 2.80	15.79 ± 2.33	< 0.001
S' [cm/s]	8.84 ± 3.45	11.00 ± 3.43	< 0.001
E' [cm/s]*	7.95 ± 3.10	7.47 ± 3.17	0.197
A' [cm/s]*	9.68 ± 4.74	10.70 ± 4.57	0.050
Tricuspid regurgitation [grade]	2.02 ± 0.95	1.86 ± 0.91	0.013
RVSP [mm Hg]	31.07 ± 20.43	27.75 ± 17.35	< 0.001

RA — right atrial; RV — right ventricle; TAPSE — tricuspid annular plane systolic excursion; S' — peak systolic annular velocity; E' — peak early diastolic annular velocity; A' — peak late diastolic annular velocity; RVSP — right ventricle systolic pressure; *only in patients with sinus rhythm

Doppler imaging velocity and RV lateral wall basal and mid strain, although the authors did not observe significant changes in TAPSE and RV diameters. Moreover, Donal et al. concluded that RV septal pacing, the pacing site preferred in our electrophysiology lab, results in faster ventricular activation, reduced wall motion abnormalities, and myocardial perfusion defects [9]. Bleeker et al. [10] observed that after six months of follow-up, CRT decreased the grade of TR, reduced pulmonary artery pressure, and induced RV reverse remodelling. That observation is in concordance with the results of our study.

It has been previously shown in an acute study by Donal et al. [11], comparing four pacing modes (atrial, RV pacing, LV pacing and BiV), that peak systolic annular velocity (S') increases significantly with BiV pacing while there is no significant change in TAPSE or myocardial performance index. Our observation of the positive effect of CRT on TAPSE might be of prognostic value since TAPSE has previously been found to be an independent prognostic marker of clinical outcome in patients with HF [3, 4]. However, other trials did not demonstrate an impact of CRT on TAPSE [5, 8, 11, 12]. In the REVERSE study, CRT ON was not associated with a significant effect on TAPSE compared to CRT OFF (–0.8 ± ± 4.7 vs. 0.3 ± 5 mm, p = 0.06). However, the population of the REVERSE trial consisted of patients in NYHA classes I and II who had no, or only minor, symptoms of HF and that is not a typical group of patients undergoing CRT implantation [12]. It has been suggested that even in patients with preserved RV systolic function, reduced LVEF has an impact on TAPSE [13]. The other studies were based on a smaller number of patients.

The mechanism of how CRT improves RV systolic function remains unclear. Studies by Bleeker et al. [10] and Kanzaki et al. [14] showed a decrease of mitral regurgitation after CRT. Improvement of RV systolic function may be the result of pulmonary artery pressure and RV afterload reduction following a decrease of mitral regurgitation. Another possible explanation might be LV reverse remodelling observed after CRT. A reduction of LV diameters and an improvement of LV systolic function, vividly presented in our study, positively affect RV diastolic filling, which results in a better RV systolic function [15].

Patients responding well to HF pharmacotherapy present improvements in RV function and a decrease in pulmonary pressure, which follows decreased LV diastolic pressures [16]. In our study, three months of CRT treatment significantly decreased the grade of TR and RVSP, which potentially could affect the course of HF, but long term observations regarding this issue are missing. Additional factors which might aggravate TR are the RV lead apical position resulting in apical pacing, and a mechanical effect of rigid defibrillation leads in CRT-D devices. It has been shown in the study by Vaturi et al. [17] and in the DAVID trial [18], that apical pacing, avoided in our group, reduces also LV systolic performance and disables RV function through increased filling pressure. At our institution, the leads were placed in such a manner that the leads' coils did

not impair the tricuspid valve function. It has been suggested that factors leading to an increase in TR may decrease RV function and consequently worsen HF in patients with already elevated pulmonary pressure [19]. In our study group, after a three-month follow-up, TR and RVSP decreased. Similar effects have also been observed in other single-centre studies, where CRT induced LV reverse remodelling and normalised RV pressures and dimensions [10].

One of the parameters recommended by ASE/ESE guidelines reflecting the RV systolic function is RV FAC. Data on the impact of CRT on RV FAC comes from small single-centre studies. Janousek et al. [20] showed in a group of patients with systemic RV who underwent CRT that after more than 18 months of therapy, RV FAC increased significantly. That is consistent with our results. It is worth noting that RV FAC has been found to be a better marker of RV function than RV ejection fraction assessed in two-dimensional echocardiography [7]. The prognostic value of the presented results needs to be established in a long term follow-up and larger study groups.

Limitations of the study

Our study was performed in a single centre on a relatively small group of patients. The follow-up period was only three months. Our observation included patients with different clinical profiles in order to reflect real life clinical settings.

CONCLUSIONS

CRT improves RV systolic function evaluated with parameters assessed in a standard echocardiographic examination three months after therapy initiation. The improvement considers several echocardiographic parameters of different modalities reflecting RV systolic function.

Conflict of interest: *none declared*

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Wpływ terapii resynchronizującej na funkcję prawej komory ocenianą za pomocą standardowego badania echokardiograficznego

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Streszczenie

Wstęp i cel: Celem badania była ocena wpływu terapii resynchronizującej (CRT) na funkcję prawej komory w krótkim okresie po implantacji stymulatora resynchronizującego.

Metody: W badaniu uczestniczyło 57 chorych z niewydolnością serca (54 mężczyzn; 95% i 3 kobiety, 5%) w średnim wieku 66,4 ± 8,7 roku. Wszyscy badani byli w III lub IV klasie wg NYHA mimo stosowania optymalnej farmakoterapii, mieli frakcję wyrzutową lewej komory ≤ 35% i czas trwania zespołu QRS ≥ 120 ms w standardowym zapisie EKG. Wyjściowo i po 3 miesiącach od wszczepienia stymulatora resynchronizującego przeprowadzano badania kontrolne obejmujące wywiad dotyczący aktualnych objawów niewydolności serca, pomiary antropometryczne, badania laboratoryjne obejmujące ocenę stężenia NT-proBNP, EKG i badanie echokardiograficzne z dokładną oceną prawej komory.

Wyniki: Po upływie 3 miesięcy od zastosowania CRT średnia klasa NYHA w całej badanej grupie zmniejszyła się z 3,11 ± 0,28 do 2,25 ± 0,68 ($p < 0,001$), dystans pokonywany w teście 6-minutowego marszu zwiększył się z 298,04 ± 107,42 do 373,12 ± 127,15 m ($p < 0,001$). W całej badanej grupie CRT wpłynęła korzystnie na skurczową funkcję prawej komory. Skurczowa amplituda ruchu pierścienia trójdzielnego wzrosła z 13,95 ± 2,80 do 15,79 ± 2,33 mm ($p < 0,001$), podobnie jak składowa podłużna prędkości skurczowej części bocznej pierścienia zastawki trójdzielnej (S'), która wzrosła z 8,84 ± 3,45 do 11,00 ± 3,43 cm/s ($p < 0,001$). Stopień niedomykalności zastawki trójdzielnej zmniejszył się z 2,02 ± 0,95 do 1,86 ± 0,91 ($p = 0,013$). Skurczowe ciśnienie w prawej komorze zmniejszyło się z 31,07 ± 20,43 do 27,75 ± 17,35 mm Hg ($p < 0,001$). Zmiana pola powierzchni prawej komory oceniana w badaniu 2-wymiarowym wzrosła z 31,35 ± 10,30% do 35,40 ± 10,51% ($p < 0,001$).

Wnioski: Terapia resynchronizująca poprawia funkcję skurczową prawej komory ocenianą za pomocą standardowego badania echokardiograficznego po 3 miesiącach od rozpoczęcia terapii. Zaobserwowany korzystny wpływ stymulacji resynchronizujacej był spójny w zakresie wszystkich ocenianych parametrów echokardiograficznych świadczących o skurczowej funkcji prawej komory.

Słowa kluczowe: prawa komora, terapia resynchronizująca, echokardiografia, niewydolność serca

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