Echocardiographic evaluation of patients with severe heart failure and impairment of intraventricular conduction following cardiac resynchronisation therapy

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Abstract

Background: Echocardiographic examination is essential for clinical assessment of patients after cardiac resynchronisation therapy (CRT).
Aim: To assess the benefit of CRT in patients with end-stage heart failure at long-term follow-up.
Methods: 28 patients with end-stage heart failure, NYHA class ≥III (≥II in patients with indications for implantable cardioverter defibrillator and echocardiographic signs of ventricular mechanical systolic dyssynchrony), left ventricular ejection fraction (LVEF) <35%, QRS duration >120 ms and left bundle branch block morphology received a biventricular device. Standard colour Doppler echocardiography examination was performed at baseline, and then every 6 months, up to 2 years. Parameters of systolic and diastolic LV function, mitral insufficiency and right ventricular (RV) pressure were evaluated.
Results: Following CRT, a statistically significant improvement of LV dimensions ($p < 0.05$), and LVEF ($p < 0.001$) was recorded. CRT also resulted in a mitral regurgitation decrement ($p < 0.01$). Interventricular mechanical delay was shortened ($p = 0.0005$). After 2 years, non-significant worsening of LV dimensions was observed. At long-term follow-up CRT did not result in LV volume, left atrium, RV dimension or RV pressure reduction.
Conclusions: CRT is associated with reverse remodelling of the LV at mid-term follow-up.

Key words: cardiac resynchronisation therapy, heart failure

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Introduction

Echocardiography constitutes a crucial tool in the evaluation of patients with cardiac resynchronisation therapy (CRT). We report the outcomes of two-year echocardiographic follow-up of subjects with intraventricular conduction impairment and severe drug-refractory heart failure (HF).

Methods

Patients

The study involved 28 patients (CRT group) with ischaemic and non-ischaemic cardiomyopathy, depressed left ventricular (LV) function and impaired intraventricular conduction presenting with deterioration of HF despite optimal medical therapy (NYHA class III/IV) and patients with less advanced HF (NYHA class II or II/III) qualified for an implantable cardioverter-defibrillator (ICD) in whom echocardiography revealed signs of ventricular mechanical asynchrony. Reduction of LV ejection fraction (LVEF) ≤35% and increased LV end-diastolic volume ≥60 mm on echocardiography were regarded as evidence of LV function impairment. Impairment of intraventricular conduction was defined as the prolongation of QRS duration ≥120 ms (≥170 ms in patients with a previously implanted cardiac pacemaker). Ventricular systolic mechanical asynchrony was confirmed if interventricular delay (IVD) was ≥35 ms on echocardiography. The control group comprised 29 patients meeting the above-mentioned criteria in whom a CRT device was not implanted for various reasons (an attempt to implant a CRT device failed in 11 of them, while

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in the remaining cases the causes were as follows: lack of patient's consent, or no pacemakers or leads available). The shortest follow-up period in both groups was 6 months.

Implantation of CRT devices

In all patients the procedure was performed under fluoroscopy. The LV pacing lead was transvenously introduced via subclavian vein access into one of the coronary sinus (CS) branches. The lateral cardiac vein or the posterolateral vein was the preferred LV pacing site. The remaining CRT leads were placed in the right atrial appendage and the right ventricular apex (RVA) or at the septum near the RV outflow tract. The LV location was evaluated using chest x-ray in PA, RAO 30° and LAO 60° views.

Echocardiography

Echocardiography was performed prior to CRT implantation to confirm the indications and then at month 6, 12 and 24 after the procedure. All examinations were carried out using a Sonos 2000 device (Hewlett-Packard) equipped with a 2.0-3.5 MHz probe. Measurements were performed according to the recommendations of the American Society of Echocardiography [1]. Left ventricular volumes were determined by tracing the systolic and diastolic endocardial contour in four and dual chamber apical views. The value of LVEF was calculated with Simpson's method by averaging the results from four and dual chamber views. Mitral regurgitation was quantified based on the ratio of a regurgitant jet area with colour Doppler to left atrium (LA) area measured from apical view. Mitral regurgitation was classified as being <20%, 20-40% and >40%. Right ventricular systolic pressure was calculated as the sum of systolic pressure gradient through the tricuspid valve and estimated pressure in the right atrium. Interventricular delay was defined as pre-ejection period duration difference between aortic and pulmonary valves. Results were compared between the study and control groups. Haemodynamically optimal atrio--ventricular delay (AVD) was set following implantation of CRT in patients with sinus rhythm. The longest AV delay allowing complete pre-excitation of ventricles was determined using ECG, and subsequently this delay was shortened, if needed, based on the analysis of LV filling profile. AVD was found optimal if resulting in the longest LV filling time, without simultaneously cutting off the atrial wave, which means atrial filling termination (A velocity) occurring simultaneously with the beginning of LV systole [2].

Clinical assessment

Patient clinical status was assessed based on NYHA classification; also exercise capacity was determined using 6-minute walk test (6MWT).

Statistical analysis

The results are shown as means ± standard deviation or numbers and percentages. The significance of the differences between groups was determined using Kruskal-Wallis nonparametric analysis of variance (ANOVA) and Mann-Whitney test (in case of mitral insufficiency). Wilcoxon signed rank test was used to analyse changes within one group. A p value <0.05 was found statistically significant.

Results

Clinical characteristics of patients. At baseline, the CRT group comprised more patients in NYHA functional class III or IV compared to the control group. Otherwise there were no significant differences between groups (Table I).

Implantation of CRT device. Six subjects had a lead placed at the anterior wall, 12 subjects at the lateral wall, and 9 subjects at the posterior wall of the LV. In one patient lead location was apical.

NYHA class. Prior to enrolment in the study the percentage of patients with severe heart failure was higher in the CRT group. However, as soon as 3 months after the procedure significantly fewer CRT patients remained in NYHA class ≥III compared to the control group and this difference was maintained throughout the follow-up (Table II).

6-minute walk test. Three months after the procedure a significant difference was found with respect to 6MWT distance, and the prolongation of this distance in the CRT group, as compared with the control group, remained significant during the whole follow-up period (Table II).

Interventricular delay (IVD). At 6 and 12 months a significant reduction of IVD was shown in the CRT group (p=0.001). In the control group this parameter did not change significantly in either of the follow-up time-points. The difference between groups was detectable at 6 months and remained significant during the whole follow-up period (Table III).

End-systolic and end-diastolic diameters of the left ventricle (LVESD and LVEDD). In the CRT group, LV diameters were significantly reduced (p <0.05) after 6 months of biventricular pacing. These changes were not significant at 12 and 14 months. LV diameters tended to gradually increase in the control group, but the differences did not reach statistical significance. Having compared both groups over subsequent follow-up time-points, no significant differences were observed (Table III).

Left ventricular end-diastolic volume (LVEDV). In the CRT group, a gradual drop of LVEDV was observed (by 1, 2 and 2% at months 6, 12 and 24, respectively), while the variable was on the rise in the control group (by 1, 22 and 35%, respectively) (Figure 1). However, these changes did not reach statistical significance. Comparing both groups, a significant difference in the evaluated parameter was noticed at 12 months (Table III).

Left ventricular end-systolic volume (LVESV). It did not change significantly in the CRT group. There was a significant increase at 12 months in the control group

Table I. Baseline demographic and clinical characteristics of CRT patients and control group

Variable	CRT group (N=28)	Control group (N=29)	p
Age [years]	62±10 (40-76)	65±12 (47-63)	NS
Gender: males/females	23/5	22/7	NS
Arrhythmias:			
• paroxysmal atrial fibrillation	3 (10%)	8 (28%)	NS
• persistent atrial fibrillation	7 (25%)	3 (10%)	NS
• paroxysmal ventricular tachyarrhythmias	12 (43%)	9 (31%)	NS
Previously implanted pacemaker	10 (34%)	9 (31%)	NS
VVI	4 (16%)	2 (6%)	NS
DDD	6 (21%)	7 (24%)	NS
Previously implanted cardioverter defibrillator	1 (3%)	4 (13%)	NS
Underlying cardiac disease			
• ischaemic cardiomyopathy	19 (68%)	18 (62%)	NS
• non-ischaemic cardiomyopathy	9 (32%)	9 (31%)	NS
• muscle dystrophy	0 (0%)	2 (7%)	NS
Comorbidities			
• arterial hypertension	9 (32%)	12 (41%)	NS
• diabetes mellitus	8 (29%)	11 (38%)	NS
• chronic renal failure	5 (18%)	7 (24%)	NS
Conduction disorders			
• AV conduction disorders	8 (29%)	7 (24%)	NS
• left bundle branch block	14 (28%)	11 (48%)	NS
• right bundle branch block (RBB)	2 (7%)	1 (3%)	NS
• RBBB with left anterior hemiblock	0 (0%)	2 (7%)	NS
Pharmacological treatment			
• angiotensin converting enzyme inhibitors or angiotensin receptor blockers	26 (93%)	26 (90%)	NS
• beta-blockers	23 (82%)	27 (93%)	NS
• furosemide (mg/day)	98.8±73 (0-360)	78.6±87 (0-360)	NS
NYHA class ≥ III	24 (85%)	14 (48%)	0.007
QRS duration [ms]	180.7±34 (130-240)	180.6±33 (130-240)	NS
Left ventricular ejection fraction [%]	23±4 (14-35)	25±4 (17-35)	NS
Left ventricular end-diastolic diameter [cm]	7.2±0.7 (6.0-8.4)	6.8±7.0 (6.0-10.0)	NS
Interventricular delay [ms]	44±16 (35-80)	52±26 (35-120)	NS
Follow-up duration [months]	24±14 (58-8)	25±14 (80-7)	NS

Table II. Percentage of patients in NYHA class ≥ III and mean distance of 6-minute walk test in CRT and control groups at subsequent evaluations

Parameter	NYHA ≥ III [%]				6-minute walking distance			
Follow-up visit [months]	0	6	12	24	0	6	12	24
CRT group	82.1	14.3	4.6	14.3	299±137	393±169	404±133	473±129
Control group	48.3	62.0	63.6	77.8	326±144	265±162	278±154	281±190
p	0.0074	0.0002	<0.0001	0.0023	NS	<0.05	<0.05	<0.05

(p <0.05). Having compared both groups at month 12, the difference in the evaluated parameter was close to statistical significance (Table III).

Left ventricular ejection fraction. In the CRT group, LVEF increase was observed at 6 and 12 months of biventricular pacing. LVEF tended to gradually decline

Table III. Echocardiographic parameters at subsequent evaluations

	Baseline	6 months	12 months	24 months
Interventricular delay [ms]	n=57	n=57	n=43	n=30
CRT group	44.6±16.8	30.2±19.3	32.5±22.6	26.5±17.8
control group	51.9±25.9	54.4±29.3	52.0±29.6	56.3±32.2
p	NS	0.0005	0.0182	0.0192
Left ventricular end-systolic diameter	n=57	n=57	n=43	n=30
CRT group	6.0±0.9	5.7±0.8	5.9±1.0	6.0±0.8
control group	5.7±1.0	5.7±1.0	5.9±1.1	6.2±1.2
p	NS	NS	NS	NS
Left ventricular end-diastolic diameter	n=57	n=57	n=43	n=30
CRT group	7.2±0.7	6.8±0.8	7.0±0.7	7.4±0.8
control group	6.8±0.9	6.9±0.9	7.1±1.0	7.4±1.2
p	NS	NS	NS	NS
Left ventricular end-diastolic volume	n=57	n=57	n=43	n=30
CRT group	275.0±69	272.2±81	269.4±61	269.3±105
control group	263.5±99	267.1±98	322.8±101	356.3±168
p	NS	NS	0.0450	NS
Left ventricular end-systolic volume	n=57	n=57	n=43	n=30
CRT group	211.0±58	198.4±67	199.0±48	229.4±49
control group	197.7±86	202.7±86	251.9±111	290.9±153
p	NS	NS	0.0519	NS
Left ventricular ejection fraction [%]	n=57	n=57	n=43	n=30
CRT group	23.6±4.6	28.0±5.3	27.5±4.2	27.5±3.9
control group	24.6±4.1	24.3±5.1	23.7±4.7	18.6±5.1
p	0.4336	0.0073	0.0052	0.0016
Left atrium [cm]	n=57	n=57	n=43	n=30
CRT group	5.3±0.7	5.1±0.7	5.3±0.7	5.3±0.7
control group	5.1±0.7	5.2±0.7	5.1±0.6	5.5±0.7
p	NS	NS	NS	NS
Right ventricular diastolic diameter	n=57	n=57	n=43	n=30
CRT group	3.1±0.7	3.1±0.5	3.1±0.4	3.2±0.5
control group	3.2±0.7	3.2±0.7	3.2±0.7	3.4±0.9
p	NS	NS	NS	NS
Right ventricular systolic pressure	n=57	n=57	n=43	n=30
CRT group	37.6±12.2	36.1±11.8	35.9±16.4	54.5±52.0
control group	35.5±10.5	35.7±9.1	35.8±7.2	43.7±13.8
p	NS	NS	NS	NS

in the control group (differences NS). A difference between the two groups was seen at 6, 12 and 24 months (Table III).

Left atrium diameter (LA). In neither group did LA diameter change significantly. No differences were observed between the study groups either (Table III).

Mitral regurgitation (MR). There was a significant decrease in MR at 12 months in the CRT group. Statistical significance of differences was lost at 24 months (Figure 2). No changes were found in the control group (Figure 3). Comparing both groups, a significant difference was noted at 12 months (p=0.03). At 24 months the difference between groups was again statistically insignificant.

Right ventricular diastolic diameter (RVDD) and right ventricular systolic pressure (RVSP). Neither RVDD nor RVSP changed significantly in any of the groups. There were no differences observed between the study groups either (Table III).

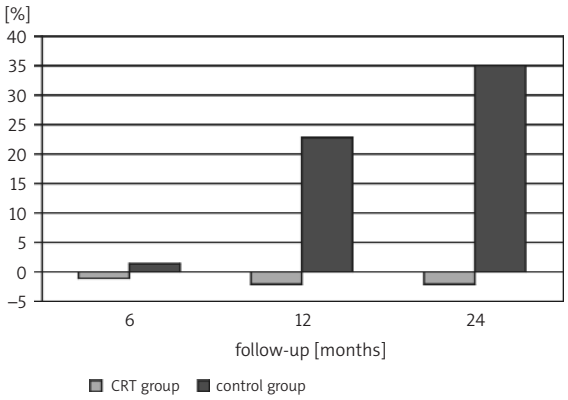

Figure 1. Left ventricular end-diastolic volume changes (%) in the CRT and control groups

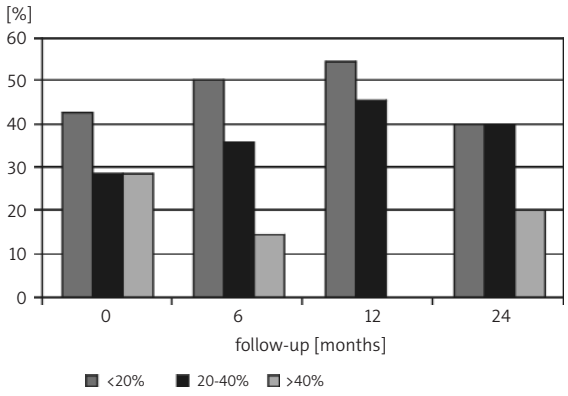

Figure 2. Mitral regurgitation changes (%) in the CRT group

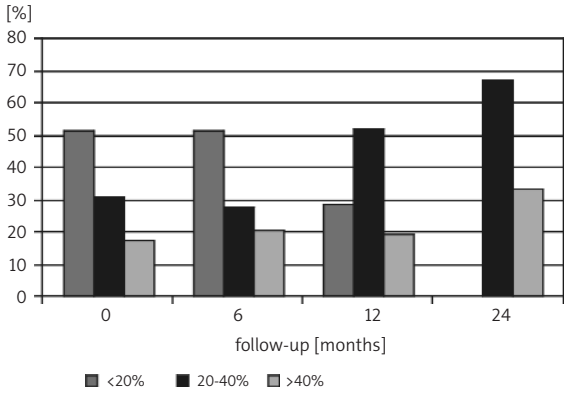

Figure 3. Mitral regurgitation changes (%) in the control group

Discussion

This study summarises two-year echocardiographic follow-up of patients with a CRT system implanted. No published data are available reporting on echocardiographic follow-up of CRT patients longer than 18 months [3]. In the reports of the two longest follow-ups of Stahlberg M et al. (3 years) and Molhoek S et al. (on average 23 months), echocardiographic examinations were not taken into account, the reporting being confined to parameters such as NYHA class, distance of 6-minute walk test and quality of life [4, 5].

One of the key elements of the pathophysiological circle leading to HF is heart remodelling. Reversal of this process would be highly desirable as it has been documented to be associated with worse prognosis of patients. Unfavourable myocardial remodelling may be partially prevented by the use of ACE inhibitors and beta-blockers. However, the latter have limited potential to reverse this process. Our study showed the influence of CRT on inhibition of progressive remodelling of the heart.

Left venticular end-systolic and end-diastolic diameters decreased in the CRT group at month 6 and 12 compared to the baseline. This change was statistically significant at month 6. At month 6 and 12 evaluations a slight decrease in LVESD and LVEDD was observed. These changes were accompanied by a statistically significant increase of LVEF. No beneficial changes of the analysed echocardiographic indices were found in the control group. Conversely, there was a tendency to a gradual increase of LV diameter and volume as well as to a decrease in LVEF. The results of the study remain consistent with the available published data [6-10].

Reversal of LV remodelling was first described by Chu-Pak Lau et al. [6]. As soon as at 3 months they observed a significant reduction of the LVESV and LVEDV. This favourable effect of CRT was also confirmed in the majority of large clinical trials. Analysis of results of 25 subjects participating in the PATH-CHF trial [7] showed significant reduction of LV diameters and volumes as well as improvement of LVEF at 6 months of biventricular pacing. In the MUSTIC trial [8] patients with sinus rhythm were found to have LV diameters decreased and increased LVEF.

Significant improvement of LV diameters and volumes was also observed in the MIRACLE clinical trial [9] over 3-month follow-up. These changes were also reported for patients not receiving beta-blockers. The twelve-week VIGOR-CHF trial showed a reduction of LV volume and size, although LVEF remained unchanged [10]. The CARE-HF trial [3] reported a significant reduction of LVEDV index as well as an increase of LVEF after 3 months of biventricular pacing. These benefits were maintained over an 18-month follow-up period.

We also assessed echocardiographic parameters after 24 months of biventricular pacing. After an initial improvement, a slight and insignificant increase of LVESD, LVEDD and LVESV was observed. The favourable changes of the remaining LV parameters (LVEDV, LVEF) were insignificant. Such results may be associated with the relatively small number of patients available for follow-up after 2 years (10 subjects). It is however possible that some stages of myocardial remodelling may be irreversible. Firstly, it should be highlighted that myocardial remodelling starts long before the clinical manifestation of HF. Patients eligible for CRT present with severe and symptomatic HF, which

is very advanced and – in some individuals – possibly irreversible. In the PATH-CHF trial [7] reverse remodelling was not observed in patients with large LVEDV at baseline (mean 351±52 ml). Secondly, systolic asynchrony is not the sole mechanism leading to progression of HF. A number of factors impact the myocardium over the years. They include, but are not limited to, increased preload (valvular insufficiencies), increased afterload (e.g. arterial hypertension), ischaemic heart disease, diabetes, etc. In our study group 68% of subjects were known to have ischaemic heart disease, 8% diabetes and 11% chronic renal failure. These conditions contribute to further progression of myocardial remodelling, also in patients treated with CRT. It would be interesting to obtain results of long-term evaluation of the biventricular pacing effect in a large patient population.

Another advantage of cardiac resynchronisation therapy indicated in the previous trials was reduction of MR, which became significant at 12 months of pacing. Such an improvement was not observed in the control group. Reduction of LA diameter was found in neither group. Similar results were reported by other investigators. Etienne et al. [11] reported significant reduction of mitral regurgitation jet area at 6 months associated with CRT. Breithardt et al. [12] confirmed reduction of functional mitral regurgitation jet area by almost 50% (from 25 to 13 mm^2) and decrease in duration of regurgitation immediately after initiation of biventricular pacing, which indicates improvement of effective LV systolic function. In another study [13], in patients on long-term CRT (median duration of 427 days) regurgitation jet area was evaluated after 72 hours of interruption of biventricular pacing, showing an increase of mitral regurgitant volume (from 7.8 ml to 16 ml). Reduction of MR severity was also found in large clinical trials. The MIRACLE trial [9] showed a significant reduction of MR jet area in the CRT group at 6 months of biventricular pacing (–2.7 cm^2). In the MUSTIC trial [8] patients with both sinus rhythm and chronic atrial fibrillation were shown to experience reduction of area of mitral regurgitant jet (by 45 and 50%, respectively). Area of MR jet was reduced by a mean of 4.2 cm^2 (p=0.003) in the CARE-HF trial population [3]. On the other hand, the PATH-CHF trial [7] failed to show a significant decrease in mitral regurgitation; however, most patients in this trial had minor MR at baseline (grade 1 or 2).

There are only two reports available [10, 14] evaluating CRT effect on LA size. Decrease of LA diameter was found in both cases as opposed to the results of our study. Both studies assessed volume in the two dimensional view while in our study LA diameter was measured from M-mode in the parasternal long axis view. The differences may be due to low sensitivity of LA diameter measurement from M-mode for the evaluation of LA enlargement. Evaluation of LA volume with longitudinal LA diameter in two-dimensional mode provides more accurate results.

Cardiac resynchronisation therapy did not result in significant reduction of RV size or RVSP. At baseline there was no significant elevation of RVSP with mean value of 37.6 mmHg (reference values <35 mmHg). Right ventricular size and RVSP showed no significant change, which may indirectly indicate that right ventricular function remains unaffected by CRT, in contrast to available reports on unfavourable results of RV apical pacing in HF patients [15, 16]. The VIGOR-CHF clinical trial showed a similar lack of effect on RV performance, where right atrial pressure and tricuspid regurgitation severity were not significantly altered. However, these parameters were not analysed in our study. Other studies have failed to evaluate CRT effect on echocardiographic RV parameters. It seems to be a very interesting area requiring further investigation.

As compared to the published data on reduction of LV size, the percentage of patients who experienced no CRT--related benefits was relatively high in our group. In the CRT group reduction of LVEDV by at least 10% did not occur in as many as 20 patients (71%). According to available reports, significant reduction of LV diameters cannot be confirmed in about 40% of subjects. It should also be noted that the extent of LVEDV reduction in the CRT group was relatively low (by 1, 2 and 2% at months 6, 12 and 24, respectively). Comparing with the PATH-CHF trial [7] at 6 months of biventricular pacing, LVEDV was reduced by about 10%, whereas in the MIRACLE trial it was reduced by about 7% [9]. It seems that several factors could have influenced the outcomes. One fourth of patients enrolled in the study group presented with chronic atrial fibrillation. CRT benefits in this group of patients are not definitely documented. In the MUSTIC AF trial [8] in patients with chronic atrial fibrillation no significant reduction of LV size was observed. In turn, Leon et al. [17] revealed favourable reduction of LV size in this patient population after implementation of CRT; however, the enrolled individuals underwent earlier ablation of the atrioventricular junction, preventing asynchronous intrinsic impulses.

In 10 (35%) patients CRT-ICD systems were implanted. All patients in the MIRACLE-ICD trial [18] had a CRT-ICD system implanted and subsequently comparison between patients with active (CRT-ICD) and inactive biventricular pacing (only ICD) was carried out. Although there was a trend towards reduction of LV diameters in the CRT-ICD group, it was statistically insignificant. The authors suggested that such an outcome might have resulted from the fact that patients requiring ICD were 'more ill', i.e. had higher risk of life-threatening ventricular arrhythmias. The InSync trial [19] in patients with NYHA class ≥II, LVEF ≤35%, and class I indications for implantation of ICD actually documented reduction of LV volume, but addition of biventricular pacing did not significantly improve the LV diameters, LVEF or MR severity in comparison with subjects treated only with ICD. In contrast to these trials stand the results of the CONTAC-CD study [18], also enrolling patients

with indications for ICD implantation (including patients with NYHA II class). In this trial LV size reduction was observed in subjects with active biventricular pacing at 6 months.

It should be emphasised that clinical benefits were also observed in patients without significant improvement on echocardiography. Similar outcomes were also reported in other studies [9, 18, 20]. It should be kept in mind that the apparently obvious relationship between worsening of HF and haemodynamic compromise (mainly in terms of LVEF) has not been confirmed. In HF patients LV function parameters at rest do not correlate with exercise capacity or severity of clinical symptoms [22].

Study limitations

The main limitation of this study is the lack of echocardiographic evaluation of intraventricular dyssynchrony.

Conclusions

1. Cardiac resynchronisation therapy in patients with moderate to severe heart failure and concomitant impairment of intraventricular conduction leads to reduction of LV diameter and improvement of LVEF as well as to reduction of mitral regurgitation severity over the mid-term follow-up (at 6 and 12 months).
2. Cardiac resynchronisation therapy affects neither RV size nor RVSP.

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Ocena echokardiograficzna chorych z zaawansowaną niewydolnością serca i zaburzeniami przewodzenia śródkomorowego po implantacji stymulatora dwukomorowego

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Streszczenie

Wstęp: Badanie echokardiograficzne jest ważnym elementem oceny chorego po implantacji stymulatora resynchronizującego (CRT).

Cel: Długoterminowa obserwacja echokardiograficzna chorych z zaburzeniami przewodzenia śródkomorowego i ciężką, oporną na leczenie farmakologiczne niewydolnością serca, po zastosowaniu CRT.

Metody: Badaniem objęto 28 chorych w wieku średnio 62,3±10,3 roku (40–76), z frakcją wyrzutową lewej komory (LVEF) ≤35%, LVEDD ≥60 mm oraz QRS ≥120 ms (≥170 ms u pacjentów z implantowanym wcześniej stymulatorem serca), u których pomimo stosowania optymalnego leczenia farmakologicznego występowały nasilone objawy niewydolności serca (HF) (III/IV klasa wg NYHA), oraz chorych z mniej zaawansowaną HF (II lub II/III klasa wg NYHA) kwalifikowanych do wszczepienia kardiowertera-defibrylatora serca, u których w badaniu echokardiograficznym stwierdzano cechy mechanicznej asynchronii skurczu komór – opóźnienie międzykomorowe skurczu (ang. *interventricular delay*, IVD) ≥35 ms. Grupę kontrolną stanowiło 29 pacjentów spełniających wyżej wymienione kryteria, u których z różnych powodów nie zastosowano CRT. Minimalny okres obserwacji pacjentów w obu grupach wynosił 6 mies. Badanie echokardiograficzne wykonywano przed wszczepieniem układu dwukomorowego (BIV), a następnie w 6., 12. i 24. miesiącu po zabiegu.

Wyniki: W ocenie echokardiograficznej po 6 mies. w grupie CRT obserwowano zmniejszenie wymiarów LV (LVESD 6,0±0,9 *vs* 5,7±0,8 cm, LVEDD 7,2±0,7 *vs* 6,8±0,8 cm, p <0,05), a także wzrost LVEF (23,6±4,6 *vs* 28,0±5,3%, p <0,001), a po 12 mies. stymulacji także istotne zmniejszenie stopnia niedomykalności mitralnej (p <0,01). Zmniejszenie objętości LV nie było istotne. Po 24 mies. obserwowano ponowne nieznamienne zwiększenie się wymiarów LV. Nie zaobserwowano zmniejszenia wielkości lewego przedsionka, prawej komory (RV) ani ciśnienia skurczowego w RV. W grupie kontrolnej obserwowano tendencję do stopniowego powiększania się wymiarów LV oraz spadek LVEF.

Wnioski: Stymulacja resynchronizująca u chorych z umiarkowaną i ciężką HF oraz towarzyszącymi zaburzeniami przewodzenia śródkomorowego prowadzi do zmniejszenia wymiarów LV oraz zwiększenia LVEF, a także zmniejszenia stopnia niedomykalności zastawki mitralnej w ocenie średnioterminowej (6 i 12 mies.). Stymulacja resynchronizująca nie wpływa na wielkość RV ani na wielkość ciśnienia skurczowego w RV.

Słowa kluczowe: stymulacja resynchronizująca, niewydolność serca

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