ORIGINAL ARTICLE

Influence of heart failure etiology on the effect of upgrading from right ventricular apical to biventricular or bifocal pacing in patients with permanent atrial fibrillation and advanced heart failure

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KEY WORDS

advanced heart failure, cardiac resynchronization therapy, ischemic cardiomyopathy, permanent atrial fibrillation

ABSTRACT

INTRODUCTION Chronic heart failure (HF) results from various disease processes. There are no data on the effect of the etiology of HF on the improvement after pacemaker upgrade.

OBJECTIVES The aim of the study was to assess changes in various parameters in patients with ischemic (IC) and nonischemic (NIC) cardiomyopathy after pacemaker upgrade in pacemaker-dependent patients with permanent atrial fibrillation, in the course of advanced HF during 12-month follow-up.

PATIENTS AND METHODS The study involved 34 patients who underwent an upgrade from right ventricular apical to biventricular or bifocal right ventricular pacing. In each patient, 12-lead electrocardiography, transthoracic echocardiography, 6-minute walking test, and the measurement of brain natriuretic peptide levels were performed. Ischemic etiology of HF was confirmed in 25 subjects. The subgroups of cardiomyopathy were compared in terms of the improvement in relative and absolute values of the parameters at 6 and 12 months.

RESULTS At baseline, the subgroups did not differ significantly in demographic data and the measured parameters. All patients completed the first period of follow-up showing clinical improvement after pacemaker upgrade. A significantly greater relative increase in the left ventricular ejection fraction was observed in the NIC subgroup at 6 months. The whole 12-month follow-up period was completed by a similar percentage of the IC and NIC patients (76% vs. 88.9%; $P = 0.73$). In the IC subgroup, a greater degree of mitral regurgitation was observed.

CONCLUSIONS Patients with IC or NIC who underwent an upgrade from right ventricular apical to biventricular or bifocal right ventricular pacing and completed a 12-month follow-up did not differ in clinical improvement. Significant differences were observed in echocardiographic parameters.

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INTRODUCTION Chronic heart failure (HF) with left bundle branch block is a result of various disease processes. Common causes of HF include ischemic heart disease, mainly cardiac muscle mass loss after myocardial infarction, inflammatory myocardial injury, or a progression of spontaneous cardiomyopathy.

The ischemic origin of HF distinguishes this type of cardiomyopathy (IC) from nonischemic cardiomyopathy (NIC) both etiologically and pathomorphologically due to greater homogeneity and progression of coronary atherosclerosis. According to the Heart Failure Survival Score, which evaluates patients referred for consideration of

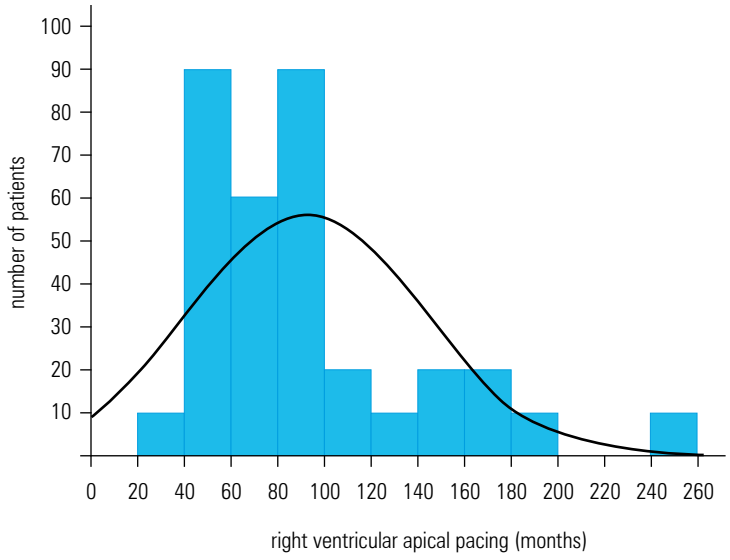


FIGURE 1 Duration of right ventricular apical pacing

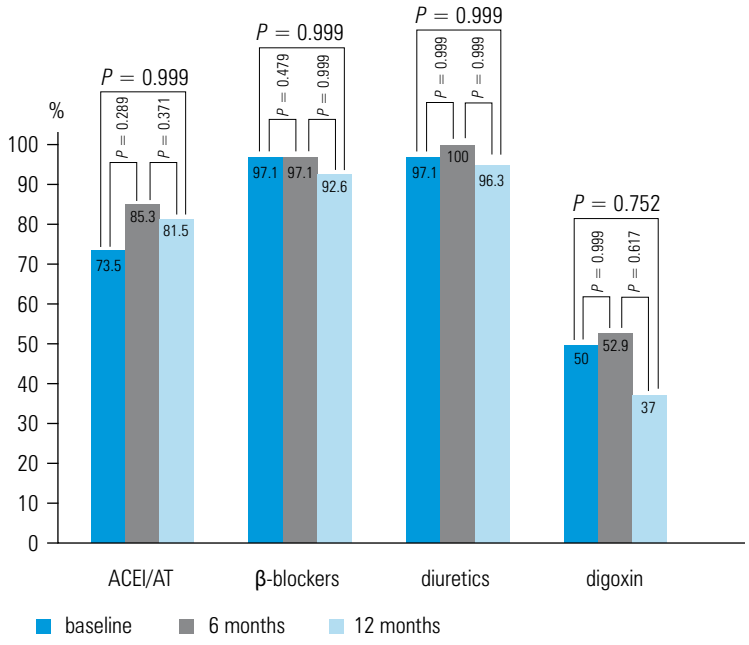


FIGURE 2 Percentage of patients receiving different groups of drugs; the significance of changes in medications was assessed using the McNemar's test
Abbreviations: ACEI – angiotensin-coverting enzyme inhibitors, AT – angiotensin T1 receptor blocker

cardiac transplantation, ischemic etiology is one of the independent risk factors apart from QRS width ≥120 ms, low mean arterial blood pressure, low oxygen consumption during cardiopulmonary exercise testing, high heart rate, low sodium level, and low left ventricular ejection fraction (LVEF).[1]

Small single-center and large population-based studies provide various data ranging from greater improvement after cardiac resynchronization therapy (CRT) in patients with NIC,[2-4] through no differences,[5,6] to relatively more positive changes in patients with more advanced cardiomyopathy caused by ischemic disease with worse baseline prognosis in this type of cardiomyopathy.[7] The differences in the study outcomes are probably caused by the differences in the criteria for detecting cardiomyopathy types, duration of follow-up after CRT, and criteria for improvement.

All these studies were conducted in patients scheduled for CRT as the first electrotherapy procedure. Additionally, randomized studies of CRT to date have been almost exclusively restricted to patients in sinus rhythm. However, about 20% of the patients receiving CRT have permanent atrial fibrillation (AF). Patients suffering from AF constitute a particular subgroup within the population of patients with HF.[8]

There are no data on the effect of HF etiology on the improvement after pacemaker upgrade in patients in whom HF developed in the course of apical right ventricular pacing and permanent AF.

We assessed changes in various parameters in patients with IC and NIC, pacemaker-dependent, with HF and permanent AF after upgrading from right ventricular apical pacing to biventricular pacing (BVP) or bifocal pacing (BFP) during 12-month follow-up.

PATIENTS AND METHODS

The study population consisted of 34 patients (8 women, 26 men), mean age 70.3 ±8.5 years, with chronic HF (New York Heart Association [NYHA] class III – 28 patients and class IV – 6 patients) and AF, and with right ventricular apical pacing (92.2 ±48.5 months; FIGURE 1) in whom upgrading to BFP (10 patients) or to BVP (24 patients) was performed. Patients had more than 95% ventricular pacing (spontaneous atrioventricular or postablation block – 12 patients).

The inclusion criteria were as follows: 1) advanced systolic left ventricular failure, NYHA class III and IV, despite optimal pharmacotherapy of at least 3 months duration prior to qualification, 2) permanent AF, 3) spontaneous or postablative atrioventricular block leading to over 95% of circadian rhythm in heart stimulation, 4) right ventricular apical pacing.

All patients provided written informed consent for pacing system upgrade. The study was approved by the local ethics committee.

Initially, all patients were scheduled for left ventricular lead implantation, that is, for an upgrade from right ventricular apical pacing to BVP. In 10 BFP patients, due to the failure to cannulate the coronary sinus or the absence of vein allowing for efficient fixation of the left ventricular lead, and due to lack of consent for microthoracotomy, we decided to stimulate the upper septal part of the right ventricular outlet tract.

All patients scheduled for the intervention received optimal individually-tailored pharmacotherapy. The percentage distribution of drugs used in the study population is presented in FIGURE 2.

The inclusion criteria, follow-up procedure, and additional examinations have been described elsewhere.[9]

In each patient who met the inclusion criteria, 12-lead electrocardiography, echocardiography, 6-minute walking test (6MWT), and the

TABLE 1 Baseline characteristics of the study group

	IC (n = 25)	NIC (n = 9)	*P*
men/women	19/6	7/2	–
percentage of women, %	24	22.2	0.91[a]
age, y	71.9 ±7.6	65.7 ±9.7	0.09[b]
duration of RVAP, mo	93.6 ±52.1	88.1 ±38.7	0.98[b]
duration of AF, mo	78.7 ±61.9	94.2 ±32.6	0.13[b]
BFP, n (%)	6 (24)	4 (44.4)	0.25[a]
percentage of BVP, n (%)	19 (76)	5 (55.6)	0.25[a]
NYHA class	3.2 ±0.4	3.1 ±0.3	0.70[b]
LVEF, %	31 ±7	30 ±8	0.69[b]
LVEDD, mm	67 ±8	65 ±7	0.48[b]
LVESD, mm	56 ±10	54 ±10	0.61[b]
MR, degree	2.6 ±0.7	2.3 ±0.5	0.33[b]
6MWT, min	285 ±121	265 ±155	0.88[b]
BNP, pg/ml	921 ±738	657 ±597	0.21[b]
percentage of RVAP, %	97 ±4	97 ±4	0.48[b]
QRS duration during RVAP, ms	199 ±22	195 ±17	0.69[b]

Data presented as mean ± standard deviation.

a χ^2 test for categorical (qualitative) variables
b Mann-Whitney *U* test for independent samples

Abbreviations: AF – atrial fibrillation, BFP – bifocal pacing, BNP – brain natriuretic peptide BVP – biventricular pacing, IC – ischemic cardiomyopathy, LVEDD – left ventricular end-diastolic diameter, LVEF – left ventricular ejection fraction, LVESD – left ventricular end-systolic diameter, MR – mitral regurgitation, NIC – nonischemic cardiomyopathy, NYHA – New York Heart Association, RVAP – right ventricular apical pacing, 6MWT – 6-minute walking test

measurement of brain natriuretic peptide (BNP) were performed.

The set of examinations was the same during the 12-month follow-up, in both 6-month stages.

The study population was further divided into those with IC and those with NIC. Ischemic etiology of advanced HF was confirmed in 25 patients, while nonischemic etiology in 9.

Criteria for the diagnosis of IC were as follows: previous myocardial infarction, previous myocardial revascularization, confirmation of coronary artery obstruction by coronary angiography, multislice computed tomography (MSCT), or fixed defect in perfusion scintigraphy, detection of segmental wall motion abnormalities by transthoracic echocardiography, and a history of anginal pain. Patients with coronary artery stenosis, anginal pain, and advanced left ventricular systolic failure were referred for invasive therapy and were excluded from the analysis.

Nonischemic etiology was confirmed if none of the IC criteria were met.

The IC group consisted of 25 patients of whom 20 had a history of myocardial infarction. These patients underwent a panel of diagnostic tests including coronary angiography (n = 12), MSCT (n = 5), or perfusion scintigraphy (n = 3). Four patients apart from coronary angiography had also MSCT, while 2 patients additionally underwent perfusion scintigraphy.

In the remaining 5 patients, IC was diagnosed on the basis of medical records, physical examination, and additional tests (perfusion scintigraphy in 1 patient, coronary angiography in 1 patient, MSCT in 1 patient, transthoracic echocardiography in 2 patients). The NIC group included 9 patients.

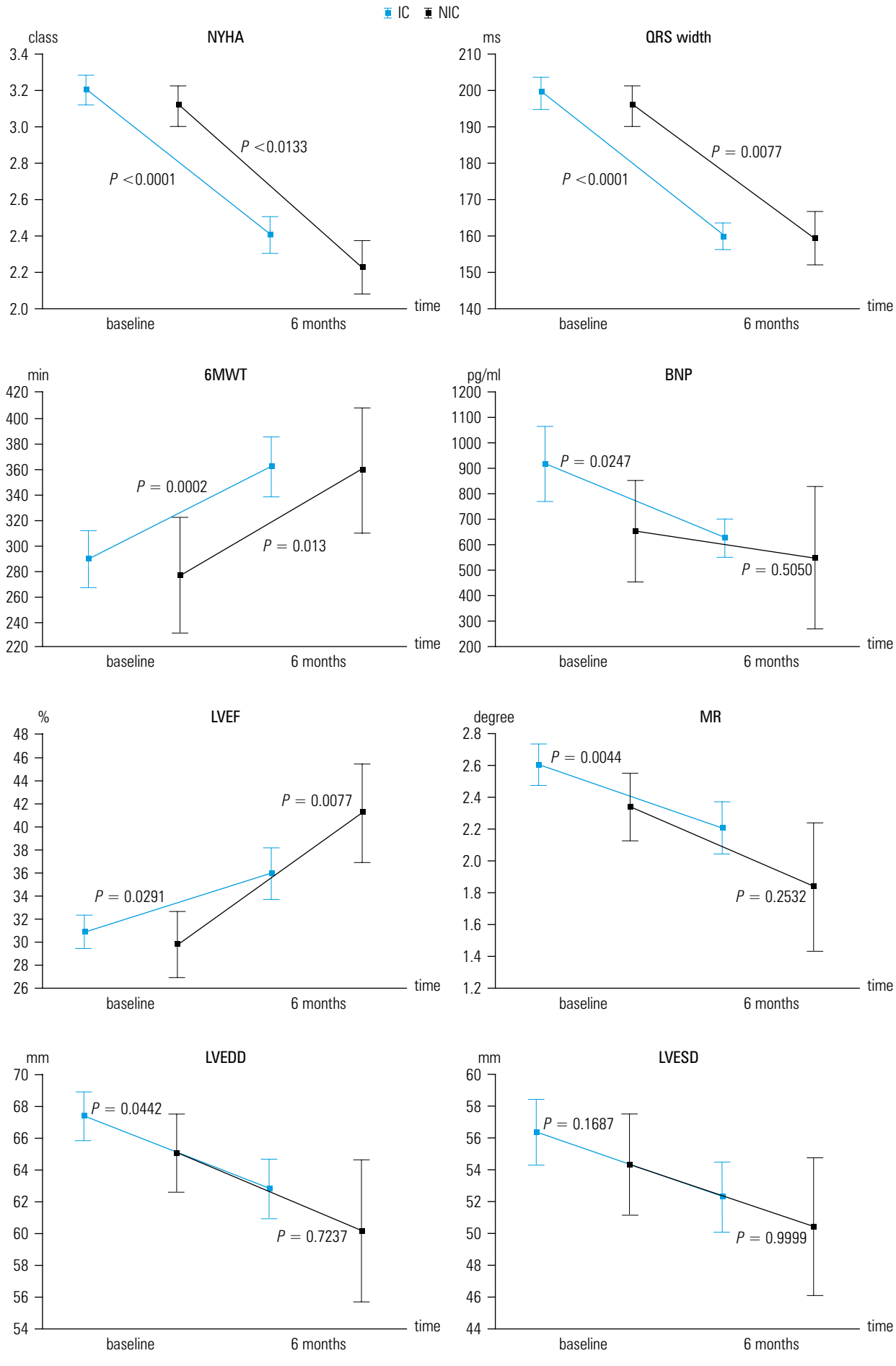
The absence of visible lesions in the epicardial coronary arteries was confirmed by coronary angiography (n = 6) and MSCT (n = 3).

The 2 subgroups were compared with respect to the degree of improvement after pacemaker upgrade measured in absolute and relative terms at 6 and 12 months. A relative change in the parameter (ΔP) was calculated according to the following formula:

$$\Delta P = \frac{P_1 - P_0}{P_0}$$

where P_0 denotes the parameter measured during right ventricular apical pacing (baseline value), and P_1 the parameter measured at 6 or 12 months, i.e., during BFP or BVP.

A statistical analysis was performed using the STATISTICA package version 9.1 (StatSoft) and included descriptive statistics (minimum, maximum, mean, standard deviation) and the comparison of mean measurable parameters for independent samples using the Mann-Whitney *U* test, and sign test for dependent samples or the χ^2 test for categorical (qualitative) variables.


IC
NIC
class
NYHA
3.4
3.2
3.0
2.8
2.6
2.4
2.2
2.0
P <0.0001
P <0.0133
time
baseline
6 months
ms
QRS width
210
200
190
180
170
160
150
140
P <0.0001
P = 0.0077
time
baseline
6 months
min
6MWT
420
400
380
360
340
320
300
280
260
240
220
P = 0.0002
P = 0.013
time
baseline
6 months
pg/ml
BNP
1200
1100
1000
900
800
700
600
500
400
300
200
P = 0.0247
P = 0.5050
time
baseline
6 months
%
LVEF
48
46
44
42
40
38
36
34
32
30
28
26
P = 0.0291
P = 0.0077
time
baseline
6 months
degree
MR
2.8
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2.2
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1.8
1.6
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P = 0.0044
P = 0.2532
time
baseline
6 months
mm
LVEDD
70
68
66
64
62
60
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P = 0.0442
P = 0.7237
time
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LVESD
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54
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50
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46
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P = 0.1687
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time
baseline
6 months

TABLE 2 Comparison of the examined parameters between patients with ischemic and nonischemic cardiomyopathy at 6 months

	IC (n = 25)	NIC (n = 9)	P^a
men/women	19/6	7/2	–
NYHA	2.4 ±0.5	2.2 ±0.4	0.35
LVEF, %	36 ±11	41 ±13	0.32
LVEDD, mm	63 ±9	60 ±13	0.98
LVESD, mm	52 ±11	50 ±13	0.86
MR, degree	2.2 ±0.8	1.8 ±1.0	0.36
6MWT, min	373 ±108	391 ±119	0.43
BNP, pg/ml	652 ±370	553 ±838	0.08
QRS duration during BVP/BFP, ms	160 ±18	159 ±22	0.97
ΔQRS	–0.25 ±0.13	–0.24 ±0.12	0.89
ΔLVEF	0.10 ±0.23	0.26 ±0.12	0.029
ΔLVEDD	–0.09 ±0.15	–0.13 ±0.29	0.81
ΔLVESD	–0.11 ±0.26	–0.14 ±0.37	0.80
ΔMR	–0.29 ±0.41	–0.5 ±0.55	0.40
Δ6MW	0.21 ±0.17	0.27 ±0.16	0.49
ΔBNP	–0.50 ±0.76	–0.97 ±1.54	0.69

Data presented as mean ± standard deviation.

a Mann-Whitney *U* test for independent samples

Abbreviations: see TABLE 1

The McNemar's test was used to assess the significance of changes in medication use.

RESULTS The 2 subgroups (IC and NIC) did not differ significantly at baseline (TABLE 1). All patients completed the first 6 months of follow-up showing clinical improvement after pacemaker upgrade both when analyzed as the whole group or the 2 subgroups.

The IC group improved with respect to the NYHA class, LVEF, diastolic left ventricular dimension, mitral regurgitation degree, 6MWT, BNP levels, and QRS width (FIGURE 3).

The NIC group showed a significant improvement in the NYHA class, LVEF, 6MWT, and QRS width (FIGURE 3).

A comparison of the absolute values and the sum of relative parameter change in the 2 subgroups indicated a greater relative increase in LVEF at 6 months in the NIC group (TABLE 2).

We assessed the effect of HF etiology on survival, clinical, and echocardiographic improvement at 12 months. The whole 12-month follow-up period was completed by a similar percent of IC and NIC patients (76% vs. 88.9%; $P = 0.734$). The study termination in the IC group was caused by sudden cardiac death (n = 1), death due to HF (n = 2), neurological complications (n = 2), and orthopedic complications (n = 1). In the NIC group, 1 patient had sudden cardiac death in the course of muscular dystrophy 8 months after pacemaker upgrade.

Endpoint-free survival did not differ significantly in the mode of pacing (BVP in the IC group was 74% vs. 50% in the NIC group; $P = 0.233$). Changes in medication use at 6 and 12 months were nonsignificant (FIGURE 2).

The 2 groups were compared at 12 months taking into account only those free of endpoints (TABLE 3).

IC patients showed a significant improvement in the following parameters: NYHA class, LVEF, left ventricular dimension, mitral regurgitation, 6MWT, and QRS width (FIGURE 4).

In the NIC group, NYHA class, QRS width, and LVEF were improved (FIGURE 4).

At 12 months, IC patients showed a significantly greater degree of mitral regurgitation compared with NIC subjects despite the fact that there were no significant differences between the 2 subgroups before pacemaker upgrade. The IC group was larger than the NIC group and benefited from pacemaker upgrade, but had a greater hemodynamic left ventricular injury at the end of follow-up.

FIGURE 3 Changes in the NYHA class, QRS duration, 6-minute walking test, brain natriuretic peptide level, left ventricular ejection fraction, mitral regurgitation, left ventricular end diastolic diameter, left ventricular end systolic diameter in the ischemic and nonischemic subgroups over a 6-month follow-up. The significance of changes was assessed using the sign test for dependent samples. In the box plot, the range (standard error) is represented by whiskers. The mean is represented by point in the middle of the whiskers.
Abbreviations: see TABLE 1

TABLE 3 Comparison of the examined parameters between patients with ischemic and nonischemic cardiomyopathy at 12 months

	IC (n = 19)	NIC (n = 8)	*P* [a]
NYHA	2.3 ±0.5	2.3 ±0.5	0.94
LVEF, %	43 ±11	43 ±15	0.98
LVEDD, mm	62 ±11	58 ±15	0.69
LVESD, mm	49 ±12	48 ±14	0.81
MR, degree	2.2 ±0.9	1.3 ±0.5	0.04
6MWT, min	326 ±102	373 ±136	0.47
BNP, pg/ml	594 ±669	644 ±832	0.79
QRS duration during BVP/BFP, ms	157 ±18	155 ±20	0.76
ΔQRS	–0.27 ±0.13	–0.25 ±0.12	0.71
ΔLVEF	0.22 ±0.23	0.26 ±0.11	0.75
ΔLVEDD	–0.12 ±0.16	–0.18 ±0.30	0.87
ΔLVESD	–0.20 ±0.30	–0.22 ±0.36	0.87
ΔMR	–0.42 ±0.59	–0.70 ±0.45	0.20
Δ6MW	0.07 ±0.38	0.19 ±0.27	0.40
ΔBNP	–2.72 ±6.52	–0.38 ±1.17	0.22

Data presented as mean ± standard deviation.

a Mann-Whitney *U* test for independent samples

Abbreviations: see TABLE 1

DISCUSSION Straburzynska-Migaj et al.[10] demonstrated differences in the significance of QRS broadening induced by the left bundle branch block depending on the type of cardiomyopathy. In IC, unlike in NIC, the broadening of QRS complex was not a marker of significant myocardial injury. Peichl et al.[11] studied electrical propagation in the left bundle branch block and showed the variation of the propagation vector in patients with IC and myocardial infarction. These findings indicate that the significance of electrical dyssynchrony in the left bundle branch block depends on the type of cardiomyopathy. There was also a study on the effect of postinfarct scar on propagation delay in the heart muscle during pacing.[12]

Varying responses to CRT, depending on the type of cardiomyopathy, were also observed on echocardiography showing the regression of ventricular remodeling only during late follow-up. Short-term studies did not demonstrate any differences.[6] The MIRACLE study showed even later reversal of changes in certain echocardiographic parameters, which improved within the first 6 months after CRT in the IC group.[4] This was not equivalent to differences in clinical outcomes. As demonstrated in the prolonged 18-month follow-up in the MIRACLE study, the clinical benefit (quality of life, NYHA class, 6MWT) were similar in the IC and NIC groups.[4]

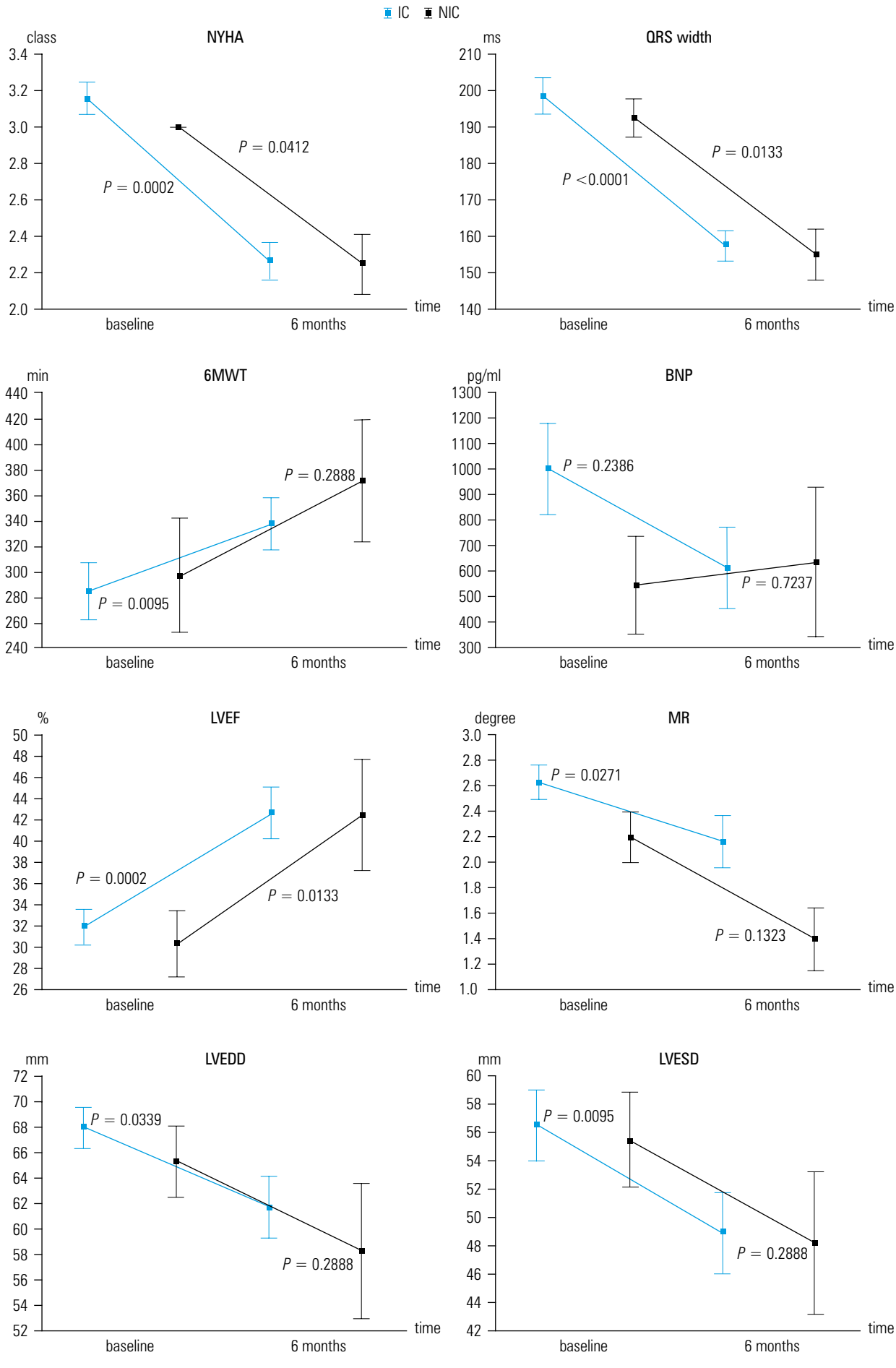
The analysis of CARE-HF suggests that the long-term effects of CRT on symptoms, morbidity, and mortality are similar in patients with IC and NIC. These benefits were observed even though IC patients had worse overall prognosis.[7]

Moreover, the 3-year MADIT-CRT study in patients with moderate HF showed differences in the prognostic value of risk factors relating to worsening HF or death between cardiomyopathy types with a 45% increase in the total risk associated with cessation of CRT in the IC group.[13]

In the present study, we evaluated a different population of patients with cardiac remodeling in the course not only of IC and NIC but also long-term right ventricular apical pacing with permanent AF. Upgrade procedure was individually determined by the course of cardiac veins, but the study subgroups did not differ significantly in the mode of pacing system.

In the NIC group, pacemaker upgrade induced more favorable changes in ventricular remodeling: greater improvement in LVEF at 6 months, and greater reduction of mitral regurgitation at 12 months. The present study corroborates the findings of other investigators that cardiomyopathy groups differ in the process of remodeling after CRT.

FIGURE 4 6-minute walking test, brain natriuretic peptide level, left ventricular ejection fraction, mitral regurgitation, left ventricular end diastolic diameter, left ventricular end systolic diameter in the ischemic and nonischemic subgroups over a 12-month follow-up. The significance of changes was assessed using the sign test for dependent samples. In the box plot, the range (standard error) is represented by whiskers. The mean is represented by point in the middle of the whiskers.
Abbreviations: see TABLE 1


IC
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P = 0.0002
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P <0.0001
P = 0.0133
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P = 0.0095
P = 0.2888
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P = 0.2386
P = 0.7237
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P = 0.0002
P = 0.0133
time
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P = 0.0271
P = 0.1323
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P = 0.0339
P = 0.2888
time
baseline
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44
42
P = 0.0095
P = 0.2888
time
baseline
6 months

Probably the more advanced HF in the IC group in the present study, similar to that observed in the CARE-HF trial, was the reason for the shortening of the follow-up period in as many as 6 patients in this group, including 3 deaths. In the NIC group, 1 patient died. It may be concluded that cardiac remodeling and the course of destruction followed by the improvement after CRT is different in IC compared with NIC. They are more complex and dependent on further ischemic episodes and progression of coronary and peripheral vascular atherosclerosis.

Late effect of CRT on the reduction of mitral regurgitation, more pronounced in NIC, preceded by greater improvement in LVEF reflects the impact of left ventricular geometry, which undergoes major changes in NIC in the absence of postinfarct scar. The present finding is an additional argument in favor of the more complex mechanism of mitral regurgitation in IC.

One of the limitations of this study is a small sample of patients, which was further subdivided into groups according to pacing types (BVP and BFP). This is a result of conducting a single center study and technical possibilities of left ventricular lead placement in patients with permanent AF.

Conclusions The study subgroups did not differ in clinical improvement during the follow-up period. Significant differences were observed in echocardiographic parameters at 6 and 12 months. The ischemic subgroup revealed a smaller degree of LVEF improvement at 6 months and minor reduction in mitral regurgitation later on. A significantly smaller reduction of mitral regurgitation may be associated with ischemic etiology.

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Wpływ etiologii niewydolności serca na efekt rozbudowy systemu stymulującego wierzchołka prawej komory do stymulacji dwukomorowej lub dwupunktowej u chorych z utrwalonym migotaniem przedsionków i zaawansowaną niewydolnością serca

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SŁOWA KLUCZOWE

kardiomiopatia niedokrwienna, stymulacja resynchronizująca, utrwalone migotanie przedsionków, zaawansowana niewydolność serca

STRESZCZENIE

WPROWADZENIE Przewlekła niewydolność serca jest skutkiem różnych procesów chorobowych. Ciągle brakuje badań na temat wpływu etiologii niewydolności serca na poprawę po zmianie systemu stymulacji.

CELE Celem badania była ocena zmian różnych parametrów u chorych z kardiomiopatią niedokrwienną (*ischemic cardiomyopathy* – IC) i nie-niedokrwienną (*nonischemic cardiomyopathy* – NIC) zależnych od rytmu stymulatora w przebiegu zaawansowanej niewydolności serca z utrwalonym migotaniem przedsionków podczas 12-miesięcznej obserwacji po zabiegu rozbudowy układu stymulującego.

PACJENCI I METODY Do badania zakwalifikowano 34 pacjentów, u których przeprowadzono rozbudowę systemu stymulującego wierzchołka prawej komory do stymulacji dwukomorowej lub dwupunktowej prawej komory. U każdego pacjenta wykonano 12-odprowadzeniowy zapis EKG, badanie echokardiograficzne, test 6-minutowego marszu oraz oznaczano stężenie mózgowego peptydu natriuretycznego w surowicy. Etiologię niedokrwienną niewydolności serca potwierdzono u 25 pacjentów. Chorych z grupy IC i NIC porównano pod względem uzyskanej poprawy w wartościach względnych i bezwzględnych mierzonych parametrów po 6 i 12 miesiącach obserwacji.

WYNIKI Wyjściowo badane podgrupy nie różniły się istotnie pod względem danych demograficznych oraz analizowanych parametrów. Pierwszy okres obserwacji ukończyli wszyscy chorzy, odnosząc korzyści kliniczne ze zmiany systemu stymulacji. Istotnie większe względne zwiększenie frakcji wyrzutowej lewej komory w czasie pierwszych 6 miesięcy nastąpił w podgrupie NIC. Pełen 12-miesięczny okres obserwacji ukończył podobny odsetek chorych z IC i NIC (76% *vs* 88,9%; p = 0,73). W podgrupie IC wykazano istotnie większy stopień niedomykalności mitralnej.

WNIOSKI Chorzy z IC i NIC po rozbudowie systemu stymulującego wierzchołka prawej komory do stymulacji dwukomorowej lub dwupunktowej prawej komory, którzy ukończyli 12-miesięczny okres obserwacji, nie różnili się pod względem uzyskanej poprawy klinicznej. Wykazano za to znamienne różnice w zakresie parametrów echokardiograficznych.

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