Impact of lead position on tricuspid regurgitation, ventricular function, and heart failure exacerbation and mortality after cardiac implantable electronic device implantation. Preliminary results from the PACE-RVTR Registry

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ABSTRACT

Background: The most frequent mechanism of lead-related tricuspid regurgitation (LRTR), which occurs in 7.2% to 44.7% of patients implanted with a cardiac implantable electronic device (CIED), is leaflet impingement or the restriction of its movement by a ventricular lead. It is unclear if the position of the lead tip — in the right ventricular apex (RVA) or other location (non-RVA) — has any influence on the development of LRTR. The study aimed to determine the impact of the CIED lead tip position on the development or progression of tricuspid regurgitation (TR) and its potential impact on heart failure exacerbation and mortality.

Methods: One hundred and two consecutive patients who received CIEDs between March 2020 and October 2021 were included in the prospective registry (PACE-RVTR). Patients were assigned to two groups depending on the lead position — the RVA group and the non-RVA group. All patients underwent echocardiographic evaluation before implantation and one year later.

Results: In terms of baseline clinical characteristics, the two groups did not differ. Before CIED implantation, patients in the non-RVA group had better left ventricular systolic function ($P = 0.004$). Pacemakers were implanted more often in the non-RVA group ($P = 0.001$) while implantable cardioverter-defibrillators in the RVA group ($P = 0.008$). Progression to severe or massive TR was more common in the non-RVA group ($P = 0.005$).

Conclusion: Severe and massive TR occurred more often in patients with the non-RVA position of the lead. The right ventricular lead position did not impact heart failure progression or all-cause mortality at two-year follow-up.

Key words: cardiac implantable electronic device, heart failure, right ventricle, tricuspid regurgitation, valve disease

WHAT'S NEW?

Severe and massive tricuspid regurgitation occurred more often in patients with the non-right ventricular apex position of the lead. The position of the right ventricular lead did not affect the progression of heart failure and all-cause mortality at one-year follow-up. Tricuspid regurgitation progression by one grade was unaffected by the type of the implanted device.

INTRODUCTION

The development or progression of tricuspid regurgitation (TR) after implantation of cardiac implantable electronic devices (CIEDs) is a growing concern [1–4]. This complication occurs in 7.2% to 44.7% of patients who received CIEDs [1, 2, 5–24]. It may result from ventricular remodeling in the natural course of heart failure (HF) or from a direct interaction between the lead and the tricuspid leaflets. The most frequent mechanism is leaflet impingement or leaflet movement restriction by a ventricular lead [11, 15, 21, 25–28]. Many authors reported that the posterior and septal leaflets are most often affected [10, 11, 15, 17, 21, 23, 26]. The most favorable position of the lead is the center of the valve orifice or one of the commissures [15, 17, 21, 23, 26, 29, 30]. It seems that placement of the lead in the right ventricular apex (RVA) more often causes TR than other locations [27]. The reason for this may be the placement of the lead closer to the posterior leaflet and its impingement [27]. Targeting a non-RVA location for the lead usually results in a central position of the tricuspid orifice [5, 27, 31, 32]. On the other hand, Cheng et al. [26] reported that significant progression of TR after CIED implantation occurred more often when the lead tip was placed in the interventricular septum (IVS). Polewczyk et al. [28] reported that in patients with lead-related tricuspid regurgitation (LRTR), the non-apical location of the lead was more frequent. Some authors claim that the position of the lead is irrelevant to TR development [17, 33, 34]. Nevertheless, coexisting TR in patients with CIEDs is associated with increased mortality [2, 7, 8, 22, 28, 35] and right ventricular failure more often than in patients without TR [6, 8, 11, 12, 22, 23]. Thus, our study aimed to determine the impact of the CIED lead tip position on TR development and progression as well as on the function of the right and left ventricles and decompensated HF-free and survival.

METHODS

Design of the study

One hundred and two consecutive patients who received a CIED — pacemaker (PM), implantable cardioverter-defibrillator (ICD), cardiac resynchronization therapy defibrillator (CRT-D), or pacemaker (CRT-P) between March 2020 and October 2021 were involved in the single-center PACE-RVTR registry. Patients were assigned to two groups depending on the lead position — the RVA group and the non-RVA group, including the upper lower parts of the IVS, the right ventricular outflow tract (RVOT), and His bundle. The position of the lead was determined on the basis of the description of the implantation procedure and chest radiography in the posteroanterior view, performed routinely after surgery (Figure 1). All patients underwent echocardiographic evaluation directly before and one year after CIED implantation (15.2 [12.0–16.0] months). Clinical data, including mortality and hospital admissions, were retrieved from the electronic medical records. HF-free survival was defined as hospitalization for HF exacerbation or an increase in diuretic doses up to the last check-up of

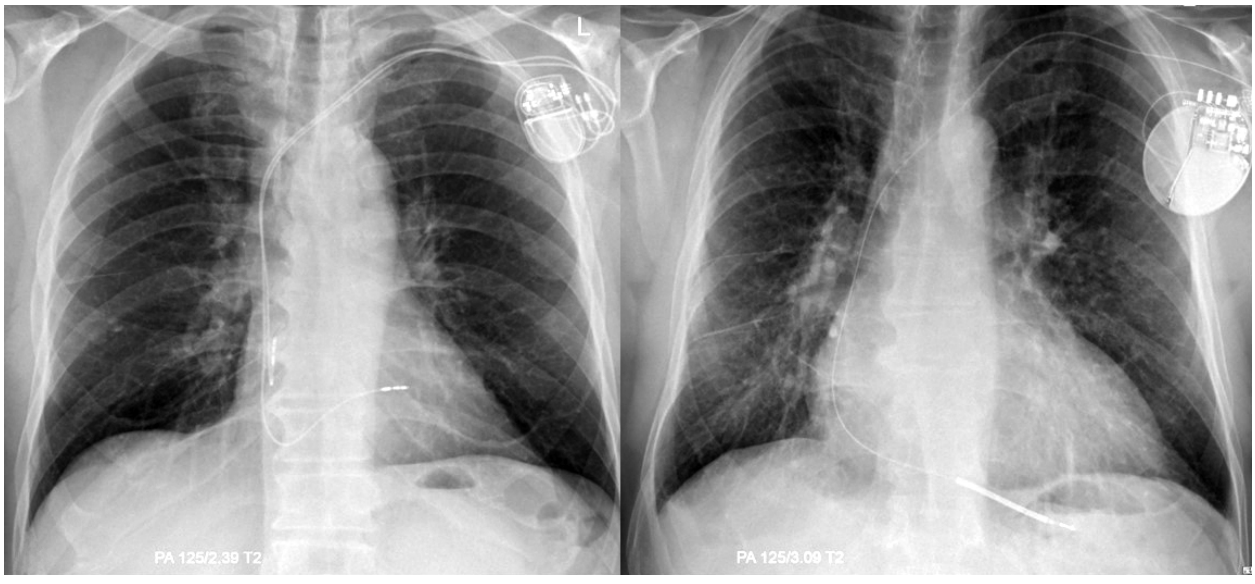

Figure 1. Position of the lead. **A.** Non-right ventricular apex. **B.** Right ventricular apex

the medical records in July 2023 (25.5 [17.0–34.0] months). LRTR was defined as an increase in TR severity by at least one grade. The percentage of ventricular pacing was checked one year after implantation.

Echocardiographic examination

Two-dimensional transthoracic echocardiography was performed before CIED implantation-and one year later by Vivid 7 GE Healthcare (Chicago, IL, US). TR grade (trivial/none 0, mild 1, moderate 2, severe 3, massive 4), other valvular insufficiencies, dimensions, and function of the right ventricle (RV) and the left ventricle (LV) were evaluated in accordance with the guidelines of the European Association of Cardiovascular Imaging [36, 37]. In the case of the RV, the evaluated parameters included RV and tricuspid valve (TV) diameters in the four-chamber view, RV area, fractional area change, tricuspid annular plane systolic excursion (TAPSE), TAPSE/tricuspid regurgitation peak gradient, and right ventricular systolic pressure; in the case of the left ventricle, these were diastolic and systolic volume and ejection fraction (EF) measured by the Simpson method. The analysis of dyssynchrony was performed according to a review by Galderisi et al. [38], and the main focus was on interventricular dyssynchrony and measured interventricular mechanical delay. According to that article, interventricular mechanical delay >40 ms was considered dyssynchrony.

Data analysis

The Kolmogorov-Smirnov test was used for the evaluation of data distribution. The numerical variables were presented as mean value and standard deviation or median and percentile distribution, depending on the result of the Kolmogorov–Smirnov test. For normally distributed independent and dependent variables, Student's t-test and paired Student's test were used, respectively. The Mann–Whitney U test was used for comparing the non-parametric independent variables, and the Wilcoxon test for dependent variables. Differences in categorical parameters were checked using Yates's χ^2 and Fisher's exact tests or McNemar's test in the case of dependent variables. P <0.05 was adopted as statistically significant. HF-free survival and overall survival were analyzed using the log-rank test and the Kaplan-Meier estimator. Calculations were performed using Statistica 10 software (TIBCO Software Inc., Palo Alto, CA, US).

RESULTS

Baseline characteristics

The average period from CIED implantation to echocardiographic examination was 15 (6) months. The RVA group included 24 patients (17 men) and the non-RVA group — 78 patients (40 men). In the non-RVA group, 65.7% of patients had the lead in the upper part of the IVS, 5.9% in the lower part, 1.9% in the RVOT, and 2.9% in the His bundle. Median age was 67.9 (60.0–77.0) years old and was similar in both groups (P = 0.39). The groups did not differ in the prevalence of atrial fibrillation (P = 1.0), coronary heart disease (P = 0.24), diabetes mellitus (P = 0.61), or chronic obstructive pulmonary disease (P = 1.0) (Table 1).

Before CIED implantation, patients in the non-RVA group showed better LV systolic function — 52.5% (33.0–57.0%) vs. 32.5% (23.5–49.5%); P = 0.004; they also a lower New York Heart Association (NYHA) classification grade (P = 0.03). They exhibited lower LV end-diastolic volume (113.0 [81.5–157.0] ml vs. 175.5 [149.0–219.0] ml; P = 0.001) and systolic volume (45.5 [33.0–95.0] ml vs. 129.0 [92.0–154.0] ml; P = 0.001); the same relation persisted after CIED implantation. The groups did not differ in terms of the right ventricular dimension and function or TR and other valvular diseases (Table 2).

Pacemakers were implanted more often in the non-RVA group and ICDs in the RVA group (P = 0.003). CRT devices were implanted in both groups with the same frequency. The groups did not differ in terms of the pacing mode (P = 0.11) and ventricular pacing percentage (group 1: 37.5% [1.0–99.0%] vs. group 2: 19.1 [1.0–91.0%]; P = 0.81) (Table 3).

Tricuspid valve and right ventricle at one-year follow-up

Tricuspid regurgitation progression in both groups was similar (by one grade P = 0.33; by two or more grades P = 0.35) (Table 4). Comparison of particular TR degrees before and after CIED implantation, respectively, is as follows (Table 5):

- The non-RVA group: none/trivial TR (61.5% vs. 24.4%; P = 0.001), mild TR (28.3% vs. 44.9%; P = 0.04), moderate TR (8.9% vs. 14.1%; P = 0.45), severe and massive TR (1.3% vs. 14.1%; P = 0.005);
- The RVA group: none/trivial TR (45.8% vs. 37.5%; P = 0.77), mild TR (41.7% vs. 33.3%; P = 0.76), moderate TR (12.5% vs. 25.0%; P = 0.46), severe and massive TR (0.0% vs. 4.2%; P = 1.00).

In the non-RVA group, TR progression by at least one grade was related to the position of the lead in the upper part of the IVS. Moreover, interventricular dyssynchrony did not affect TR progression in all patients (P = 0.55) (Table 6).

Tricuspid regression progression by one grade was independent of the type of the implanted device (patients with PM: non-RVA — 34.6% vs. RVA — 66.7%; P = 0.19 and with ICD/CRT-D/CRT-P: non-RVA 23.1% vs. RVA 27.8%; P = 0.74). Moreover, in patients in the non-RVA group with ICDs and CRT-Ds, there was a tendency for TR progression by 2 or more grades in comparison to patients with PMs (P = 0.06) (Table 4).

Fractional area change was higher in the non-RVA group than in the RVA group (43.1% [mean SD 11.1%] vs. 37.4 [mean SD 10.6%]; P = 0.03); however, other parameters of the right ventricular function and dimensions were comparable in both groups (Table 5).

Table 1. Patient characteristics

	All (n = 102)	RVA (n = 24)	Non-RVA (n = 78)	*P*-value
Men, n (%)	57 (55.9)	17 (70.8)	40 (51.3)	0.10
Age, years, median (Q1–Q3)	67.9 (60.0–77.0)	66.5 (58.0–77.0)	68.4 (61.0–76.0)	0.39
Weigh, kg, median (Q1–Q3)	83.7 (73.0–91.5)	84.0 (71.5–92.0)	83.6 (74.0–91.5)	0.68
Height, m, median (Q1–Q3)	1.70 (1.64–1.76)	1.73 (1.67–1.78)	1.69 (1.64–1.76)	0.25
Coronary artery disease, n (%)	52 (50.9)	15 (62.5)	37 (47.4)	0.24
Diabetes mellitus, n (%)	29 (28.4)	8 (33.3)	21 (26.9)	0.61
Pulmonary disease, n (%)	7 (6.9)	1 (4.2)	5 (6.4)	1.00
Atrial fibrillation, n (%)	38 (3.2)	9 (37.5)	29 (37.2)	1.00
NYHA class, n (%)				
I	60 (58.8)	8 (33.3)	52 (66.7)	0.03
II	34 (33.3)	14 (58.3)	20 (25.6)	
III	5 (4.9)	1 (4.2)	4 (5.1)	
IV	3 (2.9)	1 (4.2)	2 (2.6)	
Bilirubin, µmol/l, median (Q1-Q3)	12.8 (7.2–14.6)	11.3 (7.1–12.4)	13.3 (7.2–17.5)	0.33
INR, median (Q1–Q3)	1.6 (0.9–1.2)	1.2 (0.9–1.1)	1.7 (0.9–1.2)	0.49
Creatinine, µmol/l, median (Q1–Q3)	89.9 (75.0–103.0)	100.9 (81.5–114.5)	86.5 (69.0–101.0)	0.008
Time since CIED implantation, months, median (Q1–Q3)	15.2 (12.0–16.0)	15.7 (12.0–15.0)	15.1 (12.0–16.0)	0.97

Abbreviations: CIED, cardiac implantable electronic device; INR, international normalized ratio; NYHA, New York Heart Association; RVA, right ventricular apex

Table 2. Results of echocardiographic examination before cardiac implantable electronic device implantation

	All (n = 102)	RVA (n = 24)	Non-RVA (n = 78)	*P*-value
RV dimension in four-chamber view, mm, median (Q1–Q3)	37.0 (35.0–40.0)	38.0 (35.0–40.0)	37.0 (35.0–41.0)	0.86
Area of RA in diastole, cm^2, median (Q1–Q3)	17.4 (15.0–21.7)	16.9 (14.3–21.0)	17.7 (15.4–21.9)	0.33
Area of RA in systole, cm^2, median (Q1–Q3)	12.0 (10.4–16.3)	12.0 (11.0–14.9)	12.2 (10.3–16.5)	0.87
TV diameter, mm, median (Q1–Q3)	32.0 (29.0–38.0)	31.0 (29.0–35.0)	33.0 (29.0–38.0)	0.67
FAC, %, mean (SD)	38.4 (10.6)	38.1 (11.0)	38.5 (10.5)	0.89
TAPSE, mm, median (Q1–Q3)	20.0 (17.0–23.0)	18.0 (16.0–21.0)	20.0 (17.0–25.0)	0.15
RVSP, mm Hg, mean (SD)	33.8 (15.8)	33.5 (19.3)	33.9 (14.9)	0.94
TAPSE/TRPG, mm/mm Hg, median (Q1–Q3)	0.5 (0.4–0.9)	0.5 (0.3–0.9)	0.5 (0.4–0.9)	0.84
LV EDV, ml, median (Q1–Q3)	127.5 (86.0–169.0)	175.5 (149.0–219.0)	113.0 (81.5–157.0)	0.001
LV ESV, ml, median (Q1–Q3)	58.0 (36.0–120.0)	129.0 (92.0–154.0)	45.5 (33.0–95.0)	0.001
LVEF, %, median (Q1–Q3)	50.0 (30.0–55.0)	32.5 (23.5–49.5)	52.5 (33.0–57.0)	0.004
TR, n (%)				
None/trace	59 (57.8)	11 (45.8)	48 (61.5)	0.50
Mild	32 (31.4)	10 (41.7)	22 (28.2)	
Medium	10 (9.8)	3 (12.5)	7 (8.9)	
Severe	1 (0.9)	0 (0.0)	1 (1.3)	
Massive	0 (0.0)	0 (0.0)	0 (0.0)	
Aortic stenosis, n (%)				
None	88 (86.3)	23 (96.0)	65 (83)	0.13
Mild	10 (9.8)	0 (0.0)	10 (12.8)	
Medium	3 (2.9)	0 (0.0)	3 (3.8)	
Severe	1 (0.9)	1 (4.2)	0 (0.0)	
Aortic regurgitation, n (%)				
None	88 (86.3)	18 (75.0)	70 (90.0)	0.1
Mild	11 (10.8)	4 (16.7)	7 (8.9)	
Medium	3 (2.9)	2 (8.3)	1 (1.3)	
Severe	0 (0.0)	0 (0.0)	0 (0.0)	
Mitral stenosis, n (%)				
Mild	0 (0.0)	0 (0.0)	0 (0.0)	1.0
Medium	0 (0.0)	0 (0.0)	0 (0.0)	
Severe	0 (0.0)	0 (0.0)	0 (0.0)	
Mitral regurgitation, n (%)				
None	58 (56.8)	11 (54.2)	47 (60.3)	0.43
Mild	30 (29.4)	7 (29.2)	23 (29.5)	
Medium	9 (8.8)	4 (16.7)	5 (6.4)	
Severe	5 (4.9)	2 (8.3)	3 (3.8)	

Abbreviations: EDV, end-diastolic volume; ESV, end-systolic volume; FAC, fractional area change; LVEF, left ventricular ejection fraction; RA, right atrium; RV, right ventricle; SD, standard deviation; TAPSE, tricuspid annular plane systolic excursion; TRPG, tricuspid regurgitation peak gradient; TV, tricuspid valve; other — see Table 1

Table 3. Results of cardiac implantable electronic device implantation (CIED) controls and echocardiographic examinations after one year of follow-up

	All (n = 102)	RVA (n = 24)	Non-RVA (n = 78)	*P*-value
CIED type and parameters				
Type of device, n (%)				
PM	58 (56.9)	6 (25.0)	52 (66.7)	0.003
ICD	28 (27.5)	12 (50.0)	16 (20.5)	
CRT-P/CRT-D	16 (15.6)	6 (25.0)	10 (12.8)	
Pacing mode, n (%)				
AAI	0 (0.0)	0 (0.0)	0 (0.0)	0.11
VVI	27 (26.5)	8 (33.3)	19 (24.4)	
DDD	60 (58.8)	10 (41.7)	50 (64.1)	
BiV	15 (14.7)	6 (25.0)	9 (11.5)	
Percentage of ventricular pacing, %, median (Q1–Q3)	20.0 (1.0–93.0)	37.5 (1.0–99.0)	19.1 (1.0–91.0)	0.81
Echocardiographic parameters				
TV diameter, mm, median (Q1–Q3)	33.0 (30.0–38.0)	33.5 (29.0–38.0)	33.0 (36.0–42.0)	0.98
FAC, %, mean (SD)	41.7 (11.2)	37.4 (10.6)	43.1 (.11.1)	0.03
TAPSE, mm, mean (SD)	19.5 (4.7)	17.9 (4.2)	20.0 (4.8)	0.06
RVSP, mm Hg, mean (SD)	30.2 (14.2)	29.3 (17.5)	30.4 (13.4)	0.79
TAPSE/TRPG, mm/mm Hg, median (Q1–Q3)	0.62 (0.46–0.94)	0.53 (0.45–1.2)	0.65 (0.47–0.93)	0.57
LV EDV, ml, median (Q1–Q3)	114.8 (86.0–166.0)	149.5 (119.0–236.0)	108.2 (85.3–194.0)	0.003
LV ESV, ml, median (Q1–Q3)	59.0 (38.0–103.0)	93.0 (49.5–146.0)	55.5 (32.5–80.5)	0.003
LVEF, %, median (Q1–Q3)	49.0 (31.0–58.0)	30.0 (26.5–51.5)	53.0 (38.0–59.0)	0.003
TR, n (%)				
None/trace	28 (27.4)	9 (37.5)	19 (24.4)	0.33
Mild	43 (42.2)	8 (33.3)	35 (44.9)	
Medium	17 (16.7)	6 (25.0)	11 (14.1)	
Severe	10 (9.8)	1 (4.2)	9 (11.5)	
Massive	2 (1.9)	0 (0.0)	2 (2.6)	
Aortic stenosis, n (%)				
None	92 (90.2)	23 (96.8)	69 (88.5)	0.051
Mild	7 (6.9)	1 (4.2)	6 (7.7)	
Medium	3 (2.9)	0 (0.0)	3 (3.8)	
Severe	0 (0.0)	0 (0.0)	0 (0.0)	
Aortic regurgitation, n (%)				
None	88 (86.2)	16 (66.7)	72 (92.3)	0.1
Mild	11 (10.8)	5 (20.8)	6 (7.7)	
Medium	3 (2.9)	3 (12.5)	0 (0.0)	
Severe	0 (0.0)	0 (0.0)	0 (0.0)	
Mitral stenosis, n (%)				
None	97 (95)	22 (91.7)	75 (96.2)	0.19
Mild	4 (3.9)	1 (4.2)	3 (3.8)	
Medium	1 (0.9)	1 (4.2)	0 (0.0)	
Severe	0 (0.0)	0 (0.0)	0 (0.0)	
Mitral regurgitation, n (%)				
None	54 (52. 9)	9 (37.5)	45 (57.7)	0.09
Mild	30 (29.4)	10 (41.7)	20 (25.6)	
Medium	16 (15.7)	5 (20.8)	11 (14.1)	
Severe	2 (1.9)	0 (0.0)	2 (2.6)	
Interventricular dyssynchrony, n (%) IVMD >40 ms	13 (12.7)	2 (8.3)	11 (14.1)	0.719

Abbreviations: AAI, single atrial stimulation; BiV, biventricular stimulation; CRT-D, cardiac resynchronization therapy defibrillator; CRT-P, cardiac resynchronization therapy pacemaker; DDD, dual chamber stimulation; ICD, implantable cardioverter defibrillator; IVMD, interventricular mechanical delay; LV, left ventricle; PM, pacemaker; RVSP, right ventricular systolic pressure; TR, tricuspid regurgitation; VVI, single ventricle stimulation; other — see Tables 1 and 2

Table 4. Level of progression of tricuspid regurgitation (TR) after cardiac implantable electric device (CIED) implantation and the position of the lead

	All (n = 102)	RVA (n = 24)	Non-RVA (n = 78)	*P*-value
TR progression				
No progression, n (%)	42 (41.2)	13 (54.2)	29 (37.2)	0.16
TR progression by 1 grade n (%)	35 (34.3)	6 (25.0)	29 (37.2)	0.33
None/trace to mild	25 (24.5)	3 (12.5)	22 (28.2)	
Mild to moderate	5 (4.9)	2 (8.3)	3 (3.8)	
Moderate to severe	4 (3.9)	1 (4.2)	3 (3.8)	
Severe to massive	1 (0.9)	0 (0.0)	1 (1.3)	
TR progression by ≥2 grade, n (%)	16 (15.7)	2 (8.3)	14 (17.9)	0.35
None/trace to medium	9 (8.8)	2 (8.3)	7 (8.9)	
None/trace to severe	2 (1.9)	0 (0.0)	2 (2.6)	
Mild to severe	5 (4.9)	0 (0.0)	5 (6.4)	
Moderate to massive	0 (0.0)	0 (0.0)	0 (0.0)	
Regression, n (%)	10 (9.8)	3 (125)	7 (8.9)	0.69

	All (n = 102)			RVA (n = 24)			Non-RVA (n = 78)			*P*-value (Group 1 vs. Group 2)	
	PM (n = 58)	ICD/ CRT-D/ CRT-P (n = 44)	*P*-value	PM (n = 6)	ICD/ CRT-D/ CRT-P (n = 18)	*P*-value	PM (n = 52)	ICD/ CRT-D/ CRT-P (n = 26)	*P*-value	PM	ICD/ CRT-D/ CRT-P
TR progression due to CIED type											
No progression, n (%)	22 (37.9)	19 (43.2)	0.68	4 (66.7)	9 (50.0)	0.65	18 (34.6)	10 (38.5)	0.80	0.187	0.54
Progression by 1 grade, n (%)	24 (41.4)	11 (25.0)	0.09	1 (16.7)	5 (27.8)	1.00	23 (44.2)	6 (23.1)	0.09	0.384	0.74
Progression by ≥2 grades, n (%)	6 (10.3)	10 (22.7)	0.10	0 (0.0)	2 (11.1)	1.00	6 (11.5)	8 (30.8)	0.06	1.000	0.16
Regression, n (%)	6 (10.3)	4 (9.1)	1.00	1 (16.7)	2 (11.1)	1.00	5 (9.6)	2 (7.7)	1.00	0.497	1.00

Abbreviations: see Table 3

Table 5. Comparison of changes in echocardiographic parameters before and after implantation of cardiac implantable electric devices

Parameter	All (n = 102)			RVA (n = 24)			Non-RVA (n = 78)		
	Before implantation	After implantation	*P*-value	Before implantation	After implantation	*P*-value	Before implantation	After implantation	*P*-value
TR grade, n (%)									
None	59 (57.8)	28 (27.4)	0.001	11 (45.8)	9 (37.5)	0.77	48 (61.5)	19 (24.4)	0.001
Mild	32 (31.4)	43 (42.2)	0.15	10 (41.7)	8 (33.3)	0.76	22 (28.2)	35 (44.9)	0.045
Medium	10 (9.8)	17 (16.7)	0.21	3 (12.5)	6 (25.0)	0.46	7 (8.9)	11 (14.1)	0.45
≥Severe	1 (0.9)	12 (11.7)	0.002	0 (0.0)	1 (4.2)	1.00	1 (1.3)	11 (14.1)	0.005
RA in diastole, cm^2, median (Q1–Q3)	17.4 (15.0–21.7)	19.4 (16.3–24.0)	0.12	16.9 (14.3–21.0)	18.5 (15.4–21.3)	0.74	17.7 (15.4–21.9)	19.8 (16.6–24.2)	0.11
RA in systole, cm^2, median (Q1–Q3)	12.0 (10.4–16.3)	13.0 (11.3–16.9)	0.33	12.0 (11.0–14.9)	12.2 (10.1–15.5)	0.79	12.2 (10.3–16.5)	13.2 (11.5–18.1)	0.37
TV diameter, mm, median (Q1–Q3)	32.0 (29.0–38.0)	33.0 (30.0–38.0)	0.39	31.0 (29.0-35.0)	33.5 (29.0–38.0)	0.11	33.0 (29.0–38.0)	33.0 (36.0–42.0)	0.82
RV in 4 chambers, mm, median (Q1–Q3)	37.0 (35.0–40.0)	38.0 (36.0–42.0)	0.15	38.0 (35.0–40.0)	37.0 (34.0–42.0)	0.79	37.0 (35.0–41.0)	38.0 (36.0–42.0)	0.07
RV in diastole, cm^2, mean (SD)	18.9 (5.4)	20.5 (5.9)	0.004	18.3 (4.3)	21.0 (6.1)	0.06	19.2 (5.8)	20.3 (5.8)	0.03
RVSP, mm Hg, mean (SD)	33.8 (15.8)	30.2 (14.2)	0.06	33.5 (19.3)	29.3 (17.5)	0.39	33.9 (14.9)	30.4 (13.4)	0.08
FAC RV, %, mean (SD)	38.4 (10.6)	41.7 (11.2)	0.28	38.1 (11.0)	37.4 (10.6)	0.88	38.5 (10.5)	43.1 (11.1)	0.17
TAPSE, mm, median (Q1–Q3)	20.0 (17.0–23.0)	19.5 (4.7)	0.32	18.0 (16.0–21.0)	17.9 (4.2)	0.29	20.0 (17.0–25.0)	20.0 (4.8)	0.57
TAPSE/TRPG, mm/mm Hg, median (Q1–Q3)	0.5 (0.4–0.9)	0.62 (0.46–0.94)	0.12	0.5 (0.3–0.9)	0.53 (0.45–1.2)	1.00	0.5 (0.4–0.9)	0.65 (047–0.93)	0.10
LV EDV, ml, median (Q1–Q3)	127.5 (86.0–169.0)	114.8 (86.0–166.0)	0.38	175.5 (149.0–219.0)	149.5 (119.0–236.0)	0.72	113.0 (81.5–157.0)	108.2 (85.3–194.0)	0.51
LV ESV, ml, median (Q1–Q3)	58.0 (36.0–120.0)	59.0 (38.0–103.0)	0.36	129.0 (92.0–154.0)	93.0 (49.5–146.0)	0.49	45.5 (33.0–95.0)	55.5 (32.5–80.5)	0.69
LVEF, %, median (Q1–Q3)	50.0 (30.0–55.0)	49.0 (31.0–58.0)	0.003	32.5 (23.5–49.5)	30.0 (26.5–51.5)	0.24	52.5 (33.0–57.0)	53.0 (38.0–59.0)	0.006

Abbreviations: see Table 2

Table 6. Progression of tricuspid regurgitation in relation to the position of the lead within the right ventricle and interventricular dyssynchrony

	All (n = 102)	No progression or decrease in TR (n = 51)	Progression of TR by at least 1 degree (n = 51)	*P*-value
Upper part of IVS, n (%)	67 (65.7)	28 (54.9)	39 (76.5)	0.04
Lower part of IVS, n (%)	6 (5.9)	5 (9.8)	1 (1.9)	0.20
RVOT, n (%)	2 (1.9)	1 (1.9)	1 (1.9)	1.00
His bundle, n (%)	3 (2.9)	1 (1.9)	2 (3.9)	1.00
Apex, n (%)	24 (23.5)	16 (31.4)	8 (15.7)	0.10
Dyssynchrony, n (%)	13 (12.7)	5 (9.8)	8 (15.7)	0.55

Abbreviations: IVS, interventricular septum; RVOT, right ventricular outflow tract; TR, tricuspid regurgitation

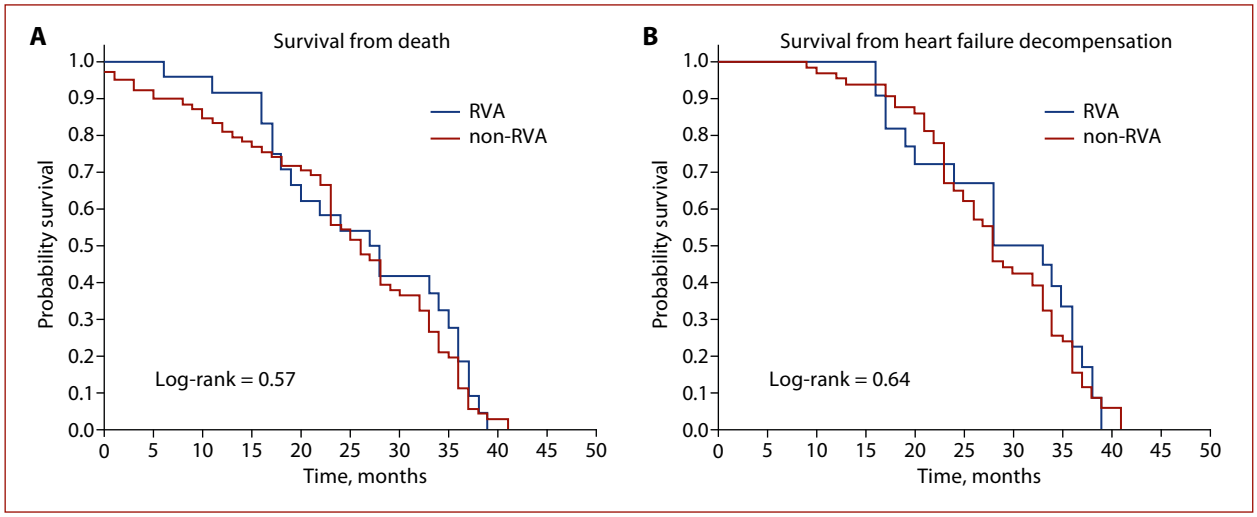

Figure 2. Overall survival and heart failure decompensation

Abbreviations: see Table 1

In the non-RVA group, the RV area was larger than before implantation (20.3 [mean SD 5.8] cm^2 vs. 19.2 [mean SD 5.8] cm^2 before implantation; $P = 0.03$), and EF increased to 53.0 (38.0–59.0)% from 52.5 (33.0–57.0)%; $P = 0.006$ (Table 5).

Mortality and heart failure exacerbation at two-year follow-up

The two groups did not differ in terms of the HF decompensation rate (RVA group 25.0% vs. non-RVA group 25.6%; $P = 0.64$) and deaths (RVA group 4.2% vs. non-RVA group 5.1%; $P = 0.58$) (Figure 2).

DISCUSSION

This study aimed to determine the impact of the CIED lead tip position on TR development and progression as well as on the RV and LV function and decompensated HF-free and overall survival.

The non-RVA group was more numerous, as it is believed that non-apical pacing is more physiological and ensures better function of the right and left ventricles; therefore, this position is preferable [39, 40]. At the beginning of the study, the group included healthier patients with higher EF and lower NYHA grades who could tolerate CIED implantation better and usually could bear longer attempts to place the lead in a position other than the RV apex. TR progression was more pronounced in the non-RVA group, with a significantly higher number of severe and massive TR. As TR progression was mainly related to the position of the lead in the upper part of the IVS, this may have resulted from damaging the TV apparatus when attempting to obtain the target position of the lead in the IVS, as the chordae tendineae of the TV are densely distributed in the RV and some of them are directly connected to the IVS [41]. This finding is consistent with the observations of Cheng et al. [26] and Polewczyk et al. [28]. On the other hand, in the study of Yu et al. [27], including only patients with pacemakers, targeting the lead to a non-RVA position resulted in placing it in the middle of the TV with the lowest chance of the leaflet impingement, while RVA placement was associated with TR progression. According to Saito et al. [33], the RV pacing site is not associated with TR worsening and did not directly affect RV function at a 2-year follow-up. Rothschild, Schleifer, and Poorzand drew a similar conclusion [17, 34, 42], and Anvardeen et al. [43] suggested that only tricuspid leaflet interference by the endocardial lead is a predictor of TR development or progression, which in turn, in studies by other authors, is more often found in the case of the RVA lead position [27].

The question arises which option is safer for the patient — the non-RVA position with more physiological pacing

and location in the middle of the TV (provided that the TV apparatus is not damaged during the attempts to achieve that position), or RVA placement with higher risk of the lead restricting movements of the posterior leaflet.

As non-RVA pacing prevents the RV and LV negative remodeling [40, 44], which is consistent with the results of our study, and, consequently, secondary TR, non-RVA lead placement seems to be a better solution if surgery is performed by an experienced electrophysiologist who can place the lead in the desired location without unnecessary manipulation and risk of entanglement and damage to the chordae tendinae. Three-D echocardiographic examination performed during the procedure and directly after lead implantation may help prevent severe TR development because it enables lead replacement if its position is not optimal for TV functioning.

The importance of pacing the heart as physiologically as possible led to the development of the idea of His bundle pacing. Zaidi et al. reported that patients with His bundle pacing had a lower risk of developing LRTR; they also had decreased severity of existing TR and improved LVEF [45].

According to Xin et al. [46], who conducted an almost 10-year follow-up of RVA pacing in patients with normal LV function, long-term RVA pacing significantly increased ventricular dyssynchrony and TR degree. In our study, which had a shorter follow-up period, we did not observe any difference in the occurrence of dyssynchrony between both groups or any impact on the progression of TR.

The impact of the lead position on heart function, decompensated HF, or mortality was not demonstrated in this study. The most significant limitation of our study is a relatively short follow-up period, while RV remodeling and TR development may occur after a longer period, as presented in a meta-analysis from 2022 [2]. In our study, none of the patients with severe or more advanced tricuspid regurgitation experienced deterioration of right ventricular function, which, in the light of the current ESC guidelines for the management of valvular heart disease [47], provided an argument for the Heart Team to forego both tricuspid valve correction and lead replacement. In fact, further observation is needed to determine whether early replacement of the right ventricular lead would be a better solution than waiting for the occurrence of the right ventricular enlargement or dysfunction, especially since it is safer to remove the lead soon after implantation before it adheres to the structures of the tricuspid valve apparatus and the right ventricle. The study participants are still followed up with periodical assessments of their symptoms and changes in parameters of the right ventricular function. If their condition worsens, they will be re-qualified for intervention. Rdzanek et al. suggested in their study [48] that the presence of PM leads, when they collide with the valve leaflets, decreases the chances of a successful percutaneous tricuspid edge-to-edge procedure. However, they did not consider the presence of CIED leads as an echocardiographic exclusion criterion and indicated that the commissural position is preferable for edge-to-edge repair [48]. An often-studied aspect is the impact of CIED type on TR progression. Some authors suggest that ICDs predispose to TR because defibrillator leads are thicker and more rigid than pacing leads, which makes it more difficult to maneuver them into the target position, and damaging the tricuspid valve apparatus is more likely [6, 18, 22, 26, 49, 50]. In our study, in patients in the non-RVA groups with ICD/CRT-D devices, there was a tendency for TR progression by two or more grades ($P = 0.06$) (Table 4).

Limitations of the study

The most significant limitations of the study are the small number of patients and the relatively short follow-up period. Moreover, the difference between both groups in terms of LVEF, LV end-diastolic volume, LV ESV, creatine level, and NYHA class is the major drawback of our study. We did not perform systematic imaging of the tricuspid valve using a three-dimensional probe, which precluded the determination of the RV lead position within the tricuspid orifice.

CONCLUSION

Severe and massive TR occurred in patients with the non-RVA position of the lead. The position of the lead did not impact HF exacerbation or mortality at two years of follow-up.

Article information

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