Evaluation of the impact of tricuspid regurgitation on the right ventricle and atrium of the heart caused by pacemaker leads

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ABSTRACT

Introduction: The number of permanent device implantations to treat conduction disorders has dramatically increased over the past decade. The aim of the study was to investigate the development of lead-induced tricuspid regurgitation (TR) after permanent endocardial lead implantation.

Methods: This prospective analytical observational study included 30 adult patients (≥18 years) that qualified for a cardiac pacemaker. Before implantation, demographic and anthropometric data were recorded. A 2D echocardiogram was performed to evaluate TR prior to implantation and at 6-week and 9 - 16-month follow-ups. TAPSE and tissue doppler imaging (RV S') were used to evaluate right ventricular function and basal and mid-right ventricular (RV) diameter and right atrial (RA) area.

Results: The TR grade significantly worsened in 46% of patients from baseline to the 9 - 16-month post-implantation. However, the TR was not clinically significant. RV function, RV dimension and RA area remained within normal reference ranges. There was a negligible correlation between TR at baseline vs. the 9 - 16-month follow-up for TAPSE, RA area, basal and mid-RV diameter.

Conclusion: After long-term follow-up, TR grade worsened after lead implantation, but not to clinically significant (moderate or severe) levels.

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INTRODUCTION

Over the last decade, the number of implantable cardiac devices has increased rapidly. The increased use of pacemaker implantation can be attributed to an ageing population.[1] Therefore, the impact of endocardial implantations on the tricuspid valve is becoming increasingly important. Although tricuspid regurgitation is a common valvular lesion, it typically results from either a physiological functional or structural abnormality.[2] During the 1980s, Gibson was the first to describe the increase in lead-induced TR coincident with the use of implantable devices.[3]

Lead-induced tricuspid regurgitation (TR) is a growing concern worldwide due to the rapid increase in the use of cardiac conduction devices, for example, permanent pacemakers (PPM), implantable cardiac defibrillators (ICD) and biventricular pacemakers (BIV)].[4-6] Lead-induced tricuspid regurgitation is defined as the echocardiographic / clinical situation where tricuspid regurgitation occurs or is aggravated by implantation of a pacemaker / defibrillator lead that transverses the tricuspid valve.[7] PPM or ICD leads can damage the tricuspid valve (TV) and may result in severe symptomatic TR with clinical sequelae, including fatigue and exercise intolerance due to low cardiac output.[8] Moderate and severe TR is associated with a poor prognosis.[6] Some studies found that permanent endocardial lead implantation can lead to TR,[9-11] while others reported that lead-induced TR does not worsen after cardiac device implantation, but may develop or worsen later on during the chronic phase of TR.[1,12-14]

The aim of the study was to investigate whether pacemaker leads implanted in the right side of the heart resulted in lead-induced tricuspid regurgitation, and whether right ventricular size, right atrial size and right ventricular function were affected.

METHODS

This prospective observational study was conducted at a private cardiology practice in the Western Cape, South Africa. Thirty adult patients (≥18 years) that qualified for cardiac pacemaker implantation were included in the study. The indication for

pacemaker implantation was based on the American College of Cardiology / American Heart Association Classifications.[15] Patients with pre-existing TR (moderate to severe), Ebstein anomaly, infective endocarditis, carcinoid syndrome, endomyocardial biopsy, chest trauma, rheumatic fever and congenital heart defects were excluded.

Demographic (age, gender, race, and ethnicity) and anthropometric data (weight, height, and body mass index [BMI]) were recorded before clinical implantation. The indication for implantation, type of leads used, pacemaker type, and programme mode was recorded during implantation.

A 2D echocardiogram was performed to evaluate TR prior to implantation (baseline) and at 6-week and 9 - 16-month follow-up using vena contracta. Echocardiography was performed according to the British Society of Echocardiography guidelines.[16] Right ventricular function was evaluated using tricuspid annular plane systolic excursion (TAPSE) and tissue doppler imaging (right ventricular peak systolic velocity, (RV S'). RV'S were only recorded at 6-week follow-up. Two patients were lost to follow-up at 9 - 16 months.

ETHICS AND STATISTICS

Ethical approval was obtained from the Health Sciences Research Ethics Committee (HSREC) of the University of the Free State, South Africa (ETOVS nr: HSD2016/1441). All statistical analysis was done using Stata statistical software (Version 13.1; Stata Corp, College Station). Mean with standard deviation (SD) (data normally distributed) or median with interquartile range (IQR) (data not normally distributed) was used to summarise continuous variables. Frequencies and percentages were used to summarise categorical variables. A p-value of less than 0.05 was regarded as statistical significant.

RESULTS

The median age and BMI of the patients were 72 years and 28.9kg/m^2, respectively. Most patients were classified as overweight (n=11, 36.67%), obese grade I (n=11, 36.67%) and obese grade II (n=2, 6.67%). An equal amount of male and female patients were included (n=15) with no statistically significant differences in age, height and BMI. As expected, the mean weight significantly differed between males and females (p=0.037) (Table I).

Sick sinus syndrome (SSS) was the most frequent indication for pacemaker implantation, which included sick sinus syndrome with atrial fibrillation and syncope (n=22; 73%) followed by ventricular tachycardia (n=2; 6.67%) and 3rd degree heart block (n=2; 6.67%). Medtronic pacemakers were predominantly implanted (53.3%), followed by Biotronik (46.7%). The most frequently used leads were 5076-52/5076-58 from Medtronic (30%) and Solia S53 / Solia S60 from Biotronik (30%). All the pacemaker leads were (screw-in) active fixation leads. Ventricular pacing was performed in 93% and atrial pacing in 97% of the study population.

TABLE I: Demographic and anthropometric data.

Variable	n (%)	Median	Q1	Q2	p-value
Age (years)	**30**	**71**	**65.75**	**82**	
Age (Female)	15 (50%)	78	66	83	0.403
Age (Male)	15 (50%)	70	65	79	
Height (cm)	**30**	**170**	**162.75**	**181**	
Female	15 (50%)	168	161	173	0.013
Male	15 (50%)	178	168	184	
Weight (kg)	**30**	**83**	**72.25**	**97**	
Weight (female)	15 (50%)	79	64	84	0.037*
Weight (male)	15 (50%)	92	74	97	
BMI (kg/m^2)	**30**	**28.9**	**25.3**	**32.4**	
BMI (female)	15 (50%)	28.7	23.9	32.5	0.962
BMI (male)	15 (50%)	29.1	25.9	32.3	

*SD: standard deviation, %: percentage, cm: centimetres, kg: kilogram, BMI: body mass index, kg/m^2: kilogram force per square metre. *statistical significance <0.05.*

TRICUSPID REGURGITATION

Fourteen patients (47%) presented with no TR, 15 (50%) with trace TR and 1 (3%) with mild TR at baseline. A significant difference in TR grade was calculated between baseline and 6-week follow-up (n=30; p=0.018) and baseline and 9 - 16-month follow-up (n=28; p=0.002) (Table II).

RIGHT VENTRICULAR FUNCTION

The mean RV ventricular peak systolic velocity (m/s) was normal (≥9cm/s) at baseline and at 6-week follow-up in all patients, indicating normal RV long-axis systolic function.[17] The RV peak systolic function at baseline and 6-week follow-up was comparable (p=0.728) (Table III).

Mean TAPSE was normal at baseline, 6-week and 9 - 16-month follow-up (≥16mm). The mean TAPSE at baseline and 6-week follow-up values were comparable (23.53mm vs. 23.33mm) and did not differ significantly (p=0.527). However, the mean TAPSE differed significantly from baseline to 9 - 16-month follow-up (mean 23.53 vs. 22.68; p=0.023) (Table III).

RIGHT VENTRICLE LINEAR DIMENSION

Basal (RVD1) and mid-cavity right ventricle linear dimension measurement (RVD2)

The mean RVD1 at baseline, 6-week and 9 - 16-month follow-up was within the normal reference range (females ≤43mm; males ≤47mm).[16] The RVD1 increased significantly from baseline to 6-week follow-up (p=0.002) and from baseline to 9 - 16-month follow-up (p=<0.001). The RVD1 in female and male patients increased significantly from baseline to 6-week follow-up (p=0.018 and 0.030). However, the RVD1 significantly increased from baseline to 9 - 16-month follow-up in male patients (p=0.002) (Table IV).

The mean RVD2 at baseline, 6-week and 9 - 16-month follow-up for the female and male patients were within normal limits (females ≤35mm; males ≤ 42mm).[16] The mean RVD2 increased significantly from baseline to 6-week and 9 - 16-month follow-up in both groups (p<0.05).

RIGHT ATRIUM AREA

The mean right atrial area for female and male patients was within the normal range (females ≤19cm^2; males ≤22cm^2)[16] (Table V). The RA area increased significantly from baseline to 6-week follow-up (p=0.004) and from baseline to 9 - 16-month follow-up (p=0.002). The male patients showed a significant increase in the RA area from baseline to 6-week follow-up (p=0.026) and from baseline to 9 - 16-month follow-up (p=0.012), but the female patients not (p>0.05).

Correlation of TR with RV peak systolic velocity, TAPSE, right atrium area, RVD1 and RVD2

TR progressed from none or trace (baseline) to mild (9 - 16-month follow-up) in 7 (23%) patients (Table VI). The increase in TR severity was correlated with TAPSE, right atrium area, RVD1 and RVD2 at the 9 - 16-month follow-up.

There was a negligible correlation (0.00 to 0.30; 0.00 to -0.30) between TR baseline vs. the 9 - 16-month follow-up for TAPSE, RA area, RVD1 and RVD2 (<0.3; <-0.3) (Table VII).

DISCUSSION

The study aimed to investigate the development of lead-induced TR after permanent pacemaker implantation. The primary indication for pacemaker implantation was SSS and most patients were either overweight or obese. TR progressed from baseline to 6-week and 9 - 16-months in 13 patients, but no patients demonstrated moderate to severe TR after follow-up.

The study population was of advanced age (71 years), with females slightly older than males at presentation. The BMI of 80% of patients was classified as either overweight or obese I and II. Obesity is a modifiable risk factor for the development of cardiac disease and is a rapidly growing problem seen in modern-day societies.[18] Excessive amounts of adipose tissue contribute to haemodynamic and metabolic changes. The total blood volume and cardiac output increase with a higher body mass index and are associated with altered cardiac morphology and function, including the development of right ventricular (RV) dilation and dysfunction.[18]

Niazi, et al. (2020)[19] studied 153 patients receiving permanent pacemaker implantations and 15.8% of patients that presented with TR had a BMI >30kg/m^2. In this study, 13 patients (43%) had a BMI >30kg/m^2, of which 3 patients (4.3%) had increased TR after a 6-week follow-up and 6 patients (21.4%%) had increased TR after a 9 - 16-month follow-up. However, according to Attanasio, et al. (2017),[20] CIED implantation can be safely achieved in obese patients with a BMI >30kg/m^2.

CLINICAL INDICATION FOR PACEMAKER IMPLANTATION

SSS, including those with atrial fibrillation and syncope, was the most frequent indication for pacemaker implantation. In a study

TABLE II: Tricuspid regurgitation (TR) grade at baseline, 6-week and 9 - 16-month follow-up.

TR	n (%)	None	Trace	Mild	p-value
Baseline	30 (100%)	14 (46.7%)	15 (50%)	1 (3.3%)	
6-week follow-up	30 (100%)	9 (30%)	17 (56.6%)	4 (13.3%)	0.018*
9 - 16-month follow-up	28 (93%)	6 (21.4%)	14 (50%)	8 (28.6%)	0.002*

*mild TR: <0.3cm, trace TR: mild TR not met, but subjectively present. *statistical significance <0.05.*

TABLE III: RV peak systolic velocity and TAPSE at baseline, 6-week and 9 - 16-month follow-up.

Parameter	Mean ± SD	Range (Min-max)	p-value
RV peak systolic velocity (m/s)			
Baseline (n=30)	0.12 ± 0.02	0.1 - 0.19	0.728
6-weeks follow-up (n=30)	0.12 ± 0.02	0.1 - 0.17	
TAPSE (mm)			
Baseline (n=30)	23.53 ± 2.45	19 - 32	-
6-week follow-up (n=30)	23.33 ± 2.71	18 - 32	0.527
9 - 16 months follow-up (n=28)	22.68 ± 3.04	17 - 32	0.023*

*SD: standard deviation, min: minimum, max: maximum, RV: right ventricle, TAPSE: tricuspid annular plane systolic excursion. *statistical significance <0.05.*

TABLE IV: RVD1 and RVD2 per study group and for male and female patients.

Parameter	n	Mean ± SD	Range (min-max)	p-value
RVD1 (mm) (study group)				
Baseline	30	34.97 ± 3.71	27 - 44	-
6-week follow-up	30	36.5 ± 3.21	30 - 44	0.002*
9 - 16-month follow-up	28	37 ± 3.40	30 - 44	<0.001*
Female				
Baseline	15	35.20 ± 4.25	30 - 44	-
6-week follow-up	15	36.47 ± 3.64	30 - 44	0.018*
9 - 16-month follow-up	14	36.14 ± 3.84	30 - 44	0.088
Male				
Baseline	15	34.73 ± 3.22	27 - 40	
6-week follow-up	15	36.53 ± 2.85	32 - 42	0.030*
9 - 16-month follow-up	14	37.85 ± 2.77	33 - 43	0.002*
RVD2 (mm) (study group)				
Baseline	30	30.1 ± 4.20	22 - 37	-
6-week follow-up	30	32.23 ± 2.86	26 - 38	<0.001*
9 - 16-month follow-up	28	32.39 ± 2.75	26 - 37	<0.001*
Female				
Baseline	15	30.20 ± 4.70	22 - 37	-
6-week follow-up	15	32.47 ± 3.00	28 - 38	0.021*
9 - 16-month follow-up	14	32.29 ± 2.55	29 - 37	0.011*
Male				
Baseline	15	30.00 ± 3.82	24 - 37	-
6-week follow-up	15	32.00 ± 2.80	26 - 37	0.006*
9 - 16-month follow-up	14	32.50 ± 3.03	26 - 37	0.003*

*SD: standard deviation, min: minimum, max: maximum, RVD1: Basal right ventricular dimension, RVD2: Mid-cavity right ventricular dimension. *statistical significance <0.05.*

TABLE V: RA area per study group and for male and female patients.

Parameter	n	Mean ± SD	Range (min-max)	p-value
RA area (cm²)				
Baseline	30	15.43 ± 2.42	10.4 - 20	-
6-week follow-up	30	16.65 ± 2.79	12 - 25	0.004*
9 - 16-month follow-up	28	16.86 ± 2.81	12 - 24	0.002*
Female				
Baseline	15	14.97 ± 2.05	12 - 20	-
6-week follow-up	15	16.21 ± 3.04	12.9 - 25	0.072
9 - 16-month follow-up	14	16.28 ± 3.35	12.4 - 24	0.685
Male				
Baseline	15	15.9 ± 2.73	10.4 - 19.7	-
6-week follow-up	15	17.09 ± 2.55	12 - 20.8	0.026*
9 - 16-month follow-up	14	17.44 ± 2.11	12 - 20.1	0.012*

*SD: standard deviation, min: minimum, max: maximum, RA: right atrium. *statistical significance <0.05.*

TABLE VI: Patients demonstrating worsening TR from baseline to 9 - 16-month follow-up and corresponding TAPSE, RA area, RVD1 and RVD2.

Patient number	TR baseline	TR 9 - 16-month follow-up	TAPSE baseline	TAPSE 9 - 16- month follow-up	RA area base-line	RA area 9 - 16-month follow-up	RVD1 baseline	RVD1 9 - 16-month follow-up	RVD2 baseline	RVD2 9 - 16-month follow-up
4	Trace	Mild	23.0	23.0	16.1	15.8	37.0	37.0	34.0	33.0
5	Trace	Mild	23.0	23.0	20.0	20.4	44.0	44.0	37.0	37.0
7	Trace	Mild	24.0	24.0	14.1	17.6	34.0	43.0	24.0	31.0
9	Trace	Mild	25.0	25.0	19.2	18.5	40.0	40.0	37.0	37.0
22	Trace	Mild	23.0	23.0	16.0	16.0	36.0	36.0	31.0	31.0
23	Trace	Mild	20.0	19.0	12.0	12.0	27.0	33.0	26.0	29.0
24	None	Mild	24.0	17.0	15.5	24.0	32.0	35.0	29.0	33.0
Mean			**23.14**	**22.00**	**16.13**	**17.76**	**35.71**	**38.29**	**31.14**	**33.00**

TR: tricuspid regurgitation, TAPSE: tricuspid annular plane systolic excursion, RA: right atrium, RVD1: Basal right ventricular dimension, RVD2: Mid-cavity right ventricular dimension.

TABLE VII: Summary of correlation coefficients for 7 patients demonstrating worsening TR from baseline to 9 - 16-month follow-up for TAPSE, RA area, RVD1 and RVD2.

	TAPSE	RA Area	RVD1	RVD2
TR baseline versus 9 - 16-month follow-up	-0.2566	0.2558	0.2741	0.2306

Correlation coefficients of zero and near to zero indicate no correlation between the 2 variables.

conducted by Dalia, et al. (2020), sinus node dysfunction (SND) and high-grade atrioventricular (AV) block were the most common indications for permanent pacemaker implantation.[21]

LEAD-INDUCED TRICUSPID REGURGITATION

Most patients' TR grade at baseline was either none or trace (97%). Mild TR was reported in 13.3% of patients after 6-week follow-up and in 28.6% of patients after 9 - 16-month follow-up. Although the TR grade progressed significantly from baseline to 6-week and 9 - 16-month follow-up, none of the patients' TR grading progressed to clinically relevant moderate or severe TR. These results concur with other studies that also reported worsening of TR after pacemaker implantation.[5,11,12] In 2020, Nadar and co-workers reported a progression in TR at 12-

month follow-up after patients received a pacing lead across the tricuspid valve (TV).[6] The patients also demonstrated an increase in the incidence of right heart failure. The most likely explanation for the progression of TR is the mechanical effect of the lead as it crosses the TV, leading to mal-coaptation and interference with valve function. Fibrosis and adhesions also contribute to valve dysfunction, which can occur as early as 5 days after implantation because of the body's reaction to a foreign object.[6]

Discussions on lead-induced TR development and progression after pacemaker implantation remain controversial. The current body of evidence regarding symptomatic TR after lead implantation seems to be based mainly on case reports and observational studies.[2] Some reports confirm the development of lead-induced TR after pacemaker implantation,[6,9,11,19,22,23] while other do not.[1,12-14]

Anvardeen, et al. (2019) documented a 30% increase in TR after 1-year follow-up and reported that endocardial lead interference of the tricuspid leaflet was a predictor for new or progressive TR.[24] They also indicated that the lead position, nature of the lead, patient factors such as age and gender, atrial fibrillation, and RV dyssynchrony, measured by the percentage of RV pacing, were not associated with TR development.

None of the patients in this study developed moderate or severe TR after 9 - 16-month follow-up.

Right ventricular peak systolic velocity

The RV peak systolic velocity did not differ significantly between baseline and 6-week follow-up (p=0.728). All the measurements were within the normal reference limit and concluded that RV function (RV S') was not negatively influenced by pacemaker-lead implantation. These findings are in keeping with other Silva, et al. (2007),[25] Agarwal, et al. (2009),[5] Núñez-Gil, et al. (2011)[26] and Chen et al. (2013).[27]

In 2011, Núñez-Gil and co-workers included 85 patients in a study using standard pacemaker indications. After pacemaker implantation, echocardiography was used to evaluate RV function. RV apical pacing did not affect RV systolic function, despite induction of electromechanical dyssynchrony.[26]

Tricuspid annular plane systolic excursion (TAPSE)

TAPSE was used to evaluate RV systolic function. The results confirmed that RV function was not influenced by pacemaker lead implantation after a 6-week and 9 -16 -month follow-up. All TAPSE measurements (baseline, 6-week and 9 - 16-month follow-ups) were within the normal reference range of ≥16mm[17] and no RV systolic dysfunction was documented. The mean baseline and 6-week follow-up TAPSE values were comparable and did not significantly differ (p=0.527). However, when comparing the mean baseline TAPSE with that of the 9 - 16-month follow-up, a significant decrease in TAPSE was noted (within normal reference range, p=0.023). These results concur with results reported in literature.[9,28,29]

In 2012, Porapakkham, et al. reported that RV dysfunction is not commonly seen after pacemaker implantation.[30] They used 2D echocardiography to analyse RV function (TAPSE and S' velocity) with a mean follow-up of 6.4 years. They documented that only 4% of patients had RV dysfunction (normal TAPSE ≥16mm and S' velocity ≥9cm/s). The site of pacing, pacing mode and percentage of ventricular pacing did not influence right ventricular function.[30]

However, in 2020, Nadar, et al. reported a decline in TAPSE from baseline to late follow-up. They reported that the presence of a pacemaker lead across the TV led to the development of new TR or the worsening of pre-existing TR and was associated with an increase in RV size, deterioration of RV function, and an increase in PA pressure. TAPSE (mm) decreased from 1.87 ± 0.44 to 1.68 ± 0.42 over a period of 12 - 24 months.[9]

RV dimensions and right atrial size

The RVD1 measurement increased significantly from baseline to 6-week follow up in the female patients and from baseline to 6-week and 9 - 16-month follow-up in the male patients. Both males and females demonstrated a significant increase in RVD2 size from baseline to 6-week and from baseline to 9 - 16-month follow-up. Only the male patients showed a significant increase in the RA area from baseline to 6-week follow-up and from baseline to 9 - 16-month follow-up. However, it is still important to note that all mean RVD1 and RVD2 measurements were within the normal reference limits (sex-specific ranges used).

Sinkar, et al. (2021) documented no increase in RV parameters (e.g. RV length, basal-diameter and mid-diameter) and RA size after a 6-month follow-up after the insertion after PM implantation. According to the authors, a follow-up period of 6 months may be too short to reveal changes in RV and RA dimensions.[29]

In 2015, Arabi, et al. prospectively assessed the effect of trans-tricuspid placement of PPM, ICD and CRT leads in 41 patients.[31] The RV diameter showed a progressive increase after cardiac device implantation after a 12-month follow-up when compared to baseline measurements. Both the RVD1 and RVD2 also increased from baseline to the 9 - 16-month follow-up reported a significant increase in the RA minimum diameter from baseline to the 12-month follow-up (40.4 ± 8.7cm vs. 43.1 ± 7.6cm, $p<0.05$). The RA diameter also increased in this particular study from baseline to the 9 - 16-month follow-up (15.4 ± 2.4cm^2 vs. 16.9 ± 2.8cm^2) and showed a significant difference ($p<0.05$). None of the patients in the Arabi, et al. (2015) study showed deterioration in the development of clinical right-sided heart failure after cardiac device implantation. According to Arabi, et al. (2015) the follow-up period of 12 months was too short to observe significant changes in the echocardiographic parameters, which concur with the results of this specific study.

Nemoto, et al. (2015) raised an important point that mild TR comprises early tricuspid annular dilation and right / left atrial enlargement.[32] Atrial volume and tricuspid annular dilation are early and sensitive indicators of tricuspid regurgitation significance. RV enlargement occurs in the later stages with lead-induced TR. However, each of these effects occur in conjunction with TR severity. None of the patients that presented with TR warranted clinical treatment after lead-induced implantation in our study. At most, after 9 - 16 months, patients presented with only mild TR. The fact that the TR was not moderate to severe after 9 - 16-month implantation could explain why the RV and RA did not increase to abnormal clinical values.

The increase in TR severity was compared with TAPSE, right atrium area, RVD1 and RVD2 at the 9 - 16-month follow-up. There was a negligible correlation (0.00 to 0.30; 0.00 to -0.30) between TR baseline vs. the 9 - 16-month follow-up for TAPSE, RA area, RVD1 and RVD2 (<0.3; <-0.3).

STUDY LIMITATIONS

A small sample size limits the study. A follow-up time of 9 - 16 months may not be adequate to evaluate the impact of pacemaker lead-induced tricuspid regurgitation. An adequately powered prospective study with a longer follow-up period will contribute much to the knowledge base of this topic.

CONCLUSION

Lead-induced TR is a growing concern worldwide, as can be seen in the rapid increase in the usage of implantable devices to treat cardiac conduction disorders. The TR grade deteriorated in almost half of the patients from baseline to long-term follow-up. None of the patients developed clinically significant moderate or severe TR after pacemaker implantation. After long-term follow-up, RV function, RV dimensions, and RA area remained within the normal reference limits. This study provided baseline information within the South African context on the development of lead-induced TR.

Conflict of interest: none declared.

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