Tricuspid Insufficiency Does Not Increase Early After Permanent Implantation of Pacemaker Leads

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ABSTRACT ***Background:*** **Interference between pacemaker (PM) lead and tricuspid apparatus may cause tricuspid regurgitation (TR). However, data regarding TR in patients with implanted PM are controversial. Our aim is to find out the degree of TR in a group of patients before and following PM implantation in a prospective manner.** ***Methods:*** **The study group consisted of the patients referred for implantation of permanent PM or implantable cardioverter defibrillator (ICD). All patients underwent two-dimensional and Doppler echocardiographic evaluation before and after device implantation. The severity of TR was qualitatively classified into four groups as normal or trivial, mild, moderate, or severe. All studies were reviewed for accuracy by a second independent interpreter.** ***Results:*** **Sixty-one patients (mean age 53 $\pm$ 8 years, 44 male) referred for PM (n $=$ 55) or ICD (n $=$ 6) implantation consisted of the study population. Echocardiographic degree of TR was mild in 21 (70%), moderate in 7 (23%) and severe in 2 (7%) patients before PM implantation. Following device implantation, mild TR was noted in 23 (76%), moderate in 10 (33%), and severe in 2 (6%) cases. After the procedure, the TR severity was increased from normal/trivial to mild in 5 (16%) cases and from mild to moderate in 3 (10%). There was no worsening of the severity of TR in patients with moderate regurgitation following device implantation. The severity of TR did not change at a mean follow-up of 6 $\pm$ 3 months.** ***Conclusions:*** **New or worsening TR is relatively rare after PM implantation. It is not associated with an acute worsening or clinical deterioration. But echocardiographic follow-up is recommended to monitor other complications in chronic phase.** doi: 10.1111/j.1540-8191.2006.00251.x *(J Card Surg 2006;21:391-394)*

Interference between pacemaker (PM) lead and tricuspid apparatus may cause tricuspid regurgitation (TR). However, data regarding TR in patients with implanted PM are controversial. Functional or anatomical interference between PM lead and tricuspid apparatus, as well as perforation or laceration of valves, have all been accused as the causative mechanism.[1-7] Both increased and decreased incidence of TR has been reported mostly in retrospective studies. However, the patients with implanted PM leads have comorbidities, making the retrospective assessment less clear. In this regard, we aimed to assess prospectively, the degree of TR before and after right ventricular (RV) lead placement utilizing two-dimensional and Doppler echocardiography in a group of patients referred for implantation of PM or implantable cardioverter defibrillator (ICD).

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MATERIAL AND METHODS

Study population

The study population consisted of patients, who were referred to the departments of cardiology and cardiovascular surgery for implantation of either permanent PM or ICD. Patients with previous chronic leads or temporary leads during initial evaluation were excluded from the study.

Echocardiographic evaluation

M-mode, two-dimensional, and Doppler echocardiographic studies were performed using a commercially available real-time scanner (Vingmed system V model, GE, Horten, Norway) equipped with a 2.5-MHz transducer; all examinations and measurements were made according to the recommendations of the American Society of Echocardiography before implantation.[8] Continuous-wave Doppler measurements were made from apical four-chamber views, in order to obtain maximum tricuspid flow velocities. A complete echocardiographic study was performed following device implantation. Echocardiographic measurements

TABLE 1
Clinical Characteristics of the Study Population (n = 61)

Demographic Parameters	Values
Mean age (years)	53 ± 8
Male/Female	44/17
Timing of echo study before device implant (days)	3 ± 3
Timing of echo study after device implant (days)	1 ± 1
PM/ICD	55/6
CAD	47
DCMP	2
Left ventricular EF (%)	45 ± 12

PM = pacemaker; CAD = coronary artery disease; DCMP = dilated cardiomyopathy; EF = ejection fraction; ICD = implantable cardioverter defibrillator.

were repeated after a 6-month follow-up period; settings common to all patients were selected during the follow-up period according to the results of the first echocardiographic study. The severity of TR and qualitative classification into four groups as normal or trivial, mild, moderate, and severe were described elsewhere.[9] All measurements were reviewed by another author. In cases in which there was disagreement, two investigators jointly examined the recordings and repeated the measurements.

The same echocardiographic evaluation also has been performed in a subgroup of randomly selected patients (n = 20) off pacing or during RV pacing.

RESULTS

Study population consisted of 61 patients (mean age 53 ± 8 years, 44 male) referred for PM (n = 55) or ICD (n = 6) implantation. The clinical characteristics of the patients were summarized in Table 1. TR was detected in 30 (49%) patients before implantation. Forty patients received a dual chamber device, while 21 patients got their single chamber (ventricular) devices implanted. The ventricular leads were placed or screwed to the RV apex in all patients. Echocardiographic degree of TR was mild in 21 (70%), moderate in 7 (23%) and severe in 2 (7%) patients before PM/ICD implantation. Following device implantation, mild TR was noted in 23 (76%), moderate TR in 10 (33%) and severe TR in 2 (6%) cases. After the procedure, the TR severity was increased from normal/trivial to mild in 5 (16%) cases and from mild to moderate in 3 (10%). There was no worsening of the severity of TR in patients with moderate regurgitation following device implantation. The severity of TR did not change at mean follow-up of 6 ± 3 months. None of our cases demonstrated any decrease in terms of TR severity after PM or ICD implantation (Fig. 1). Graphical presentation of TR severity before and after device implantation is depicted in Figure 2.

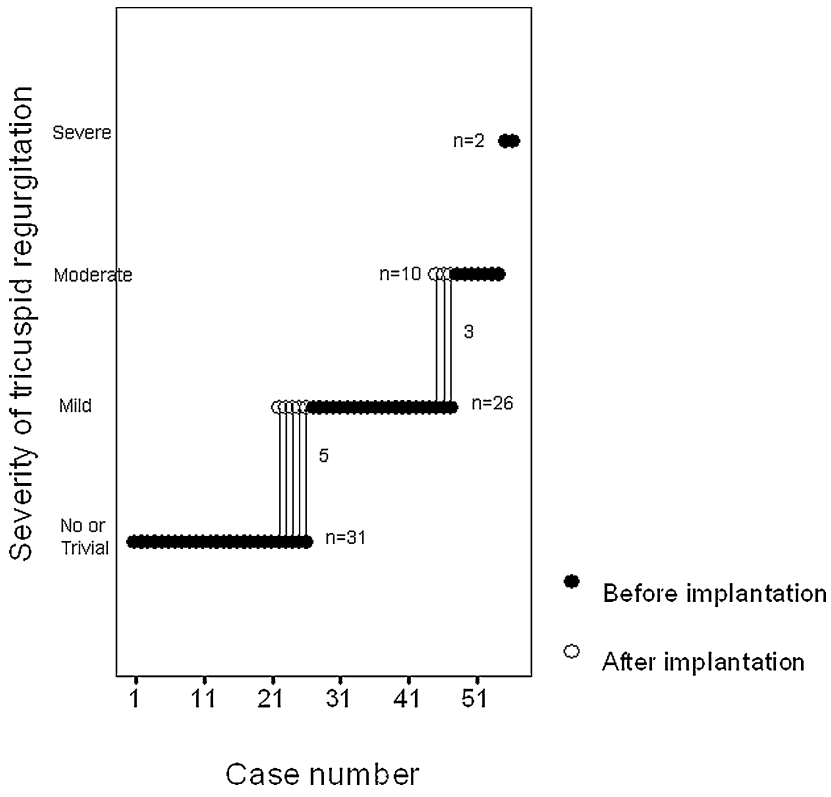

Figure 1. *Distribution of the patients' status of TR before and after device implantation. Solid circles represent preimplant patients. Empty circles and lines indicate postimplant patients with increased degree of TR. Five patients in no or trivial TR group and 3 patients in mild TR group had an increased TR following device implantation.*

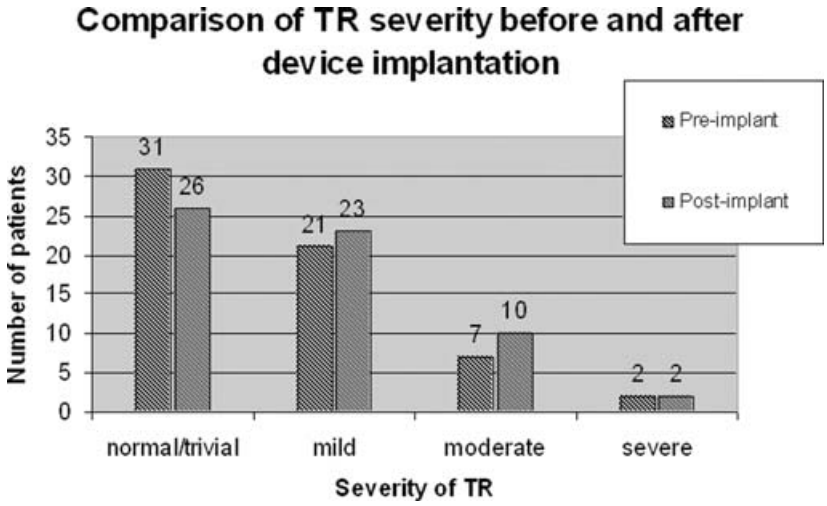

Figure 2. *Comparison of TR severity before and after PM/ICD implantation.*

Echocardiographic evaluation among patients (n = 20) who were not depend on pacing did not reveal any increase or decrease in the severity of TR when compared during RV pacing or not.

DISCUSSION

Our results indicate that new or worsening TR was relatively rare soon after PM implantation. If there is an increase in the severity of TR, it is not usually associated with an acute worsening or clinical deterioration. Although this conclusion also has been confirmed in previous studies, some investigators have noted severe tricuspid valve malfunction following RV lead placement.[1-7,10] Fortunately, these patients with severe TR likely represent extreme cases.

At baseline, the incidence of TR in our study patients was 49%. The prevalence of TR in people with a normal heart varies from 0% to 53% depending on the echocardiographic techniques and varying definitions of significant TR.[9,11,12] Our study is a report from a single center, where echocardiographic technique has been constant, and the definitions are standardized.

An interesting functional explanation for the presence of TR is an abnormal sequence of activation of the RV, when paced from the apex, with delayed activation of the papillary muscles. Besides, it has been shown that RV pacing in a dog model increases mitral valve incompetence in addition to tricuspid valve.[13] This has been tested in a subgroup of patients by echocardiographic assessment with or without pacing after PM implantation in our study. Although our result may be secondary to relatively small number of study cases, RV pacing did not cause any significant change in the severity of TR consistent with previous reports.[14-17]

While the findings of this study suggest better outcomes, important limitations need to be highlighted. First, this was a single center experience in a relatively small number of patients, and the degree of TR is partly dependent on the experience of the operator and institution. Secondly, the shadowing of the PM lead might cause an underestimation of the severity of the TR. Additionally, the male dominance of the study might prevent us from generalizing our conclusion to women population.

CONCLUSION

We conclude that insertion of a permanent electrode to the RV apex does not acutely worsen TR nor deteriorate the clinical status. Although it has a benign nature, we recommend a precise evaluation to rule out rare complications of the procedure in the presence of a new TR following implantation.

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