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The effects of right ventricular apical pacing with transvenous pacemaker and implantable cardioverter defibrillator on mitral and tricuspid regurgitation ☆

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Abstract

Background: The incidence of new or worsening tricuspid regurgitation (TR) or mitral regurgitation (MR) after permanent pacemaker (PPM) or implantable cardioverter defibrillator (ICD) lead placement has not been well investigated. We studied the effect of transvenous leads implantation and right ventricular (RV) pacing on tricuspid and mitral valve regurgitations.

Methods: We reviewed the charts of all patients undergoing PPM or ICD lead placement in our electrophysiology laboratory from December 2001 to December 2006.

Results: A total of 206 patients (120 with PPM and 86 with ICD) had baseline echocardiography within 6 months before, and a follow up study at least 6 months after lead insertion. The mean age was 74 ± 14 years; 56% were men. The follow-up period was 29 ± 19 months. TR worsened by at least one grade after lead insertion in 44.7% patients ($P < 0.001$). Pre- and post-implant changes in TR severity did not differ with respect to lead type (ICD vs. PPM) or degree of RV pacing dependence. As for MR; patients with high frequency of RV pacing (>40%) had a higher incidence of worsening MR when compared to those with low frequency of RV pacing (44% vs. 19%; $P < 0.001$).

Conclusion: PPM or ICD lead implantation worsens TR; that effect is probably induced by mechanical interferences with the TV closure and was consistent regardless the lead type or degree of RV Pacing. MR was noted to increase in patients with high frequency of RV pacing frequency; this is probably caused by the mechanical dyssynchrony induced by RV pacing.

Keywords: Tricuspid regurgitation; Mitral regurgitation; Right ventricular pacing; Permanent pacemaker; Implantable cardioverter defibrillator

Introduction

Since the introduction of an endocardial transvenous lead for permanent cardiac pacing in 1959 the technology in the field has evolved rapidly and leads to an increase in endocardial lead implantation for permanent pacemaker (PPM) and implantable cardioverter defibrillator (ICD) [1]. Right ventricular (RV) apical pacing induces an iatrogenic intraventricular delay of electrical conduction, which reflects in an interventricular and intraventricular desynchronization [2]. Endovascular cardiac pacing is a known cause of tricuspid regurgitation (TR), but the underlying mechanism has not been fully understood. Multiple studies were done trying to understand the impact of right ventricle pacing on tricuspid valve. Explanations were different in these studies as while some presumed that it is mainly a mechanical effect resulting from the presence of the pacemaker electrode at the right ventricle (RV), which would interfere with the motion of the tricuspid leaflets [3,4]; others tried to tie that solely to the dyssynchrony induced by RV pacing [5,6]. However a similar effect on the mitral valve has been reported [7]; and it is unclear if it is universal in all patient who undergo RV pacing.

We studied the effect of transvenous lead implantation and RV pacing on tricuspid and mitral valve regurgitations (MR) trying to figure out the potential mechanism RV pacing may induce.

☆ There is no conflict of interest for any of the co-authors.

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Methods

Patient selection

All charts of patients who underwent placement of a PPM or ICD lead placement at our electrophysiology laboratory between 12/2001 and 12/2006 were reviewed. Patients were included in the study if a baseline echocardiography within 6 months before and a follow up study at least 6 months after lead insertion was available. Patients with bi-ventricular pacemaker devices, lead replacement or QRS > 120 ms were excluded. The patients were divided depending on the degree of RV pacing dependence documented on repeated visits into group 1, which included patients with high degree of RV pacing, (RV pacing >40% of the time), and group 2, which included patients with low frequency of RV pacing (RV pacing <40% of the time).

Pacemaker and ICD settings

All right ventricular leads were implanted apically. Patients with single lead were all programmed to be in VVI-R mode (ventricular pacing, ventricular sensing, inhibition response and rate-adaptive). Patients with dual lead were set up to be in DDD-R mode (dual-chamber sensed and paced, rate-modulated) with algorithm to prevent pacemaker-mediated tachycardia (PMT). Back-up rate varied from 50 to 60 beats per minute (BPM) for patients with known or are expected to develop bradycardia. The difference was related to operator and patients' factors. Back-up rate in primary prevention ICD patients without associated bradycardia (20 patients) was 40 BPM.

Echocardiography evaluation

A complete echocardiography evaluation was followed for both ICD and PPM groups including the measurement of the left ventricular (LV) function, and the evaluation of atrioventricular (AV) valves. Grading the severity of both MR and TR was done according to the recommendations of the American Society of Echocardiography [8]. The regurgitation was graded as: absent, mild, moderate, or severe.

The intensity of MR was assessed using either of the following measurements: the Doppler vena contracta width (mild if vena contracta width <3 mm, moderate 3–7 and severe if > 7 mm); color jet area (mild if jet area <4 cm^2, moderate 4–10 cm^2 and severe if jet area >10 cm^2); regurgitant volume (mild if regurgitant volume <30 mL, moderate if 30–60 mL, or severe if >60 mL); and/or regurgitant fraction (mild if regurgitant fraction <30%, moderate if 30–50%, or severe if >50 %) [8]. In case of inconsistency between these measurements, the Doppler vena contracta width was used for final decision. Quantification of TR was done using Doppler vena contracta width (mild if vena contracta width <3 mm, Moderate 3–7 and severe if >7 mm) and color jet area (mild if vena contracta width <5 cm^2, moderate 5–10 cm^2 and severe if >10 cm^2) [8]. The RV systolic pressure (RVSP) or pulmonary artery systolic pressure (PSAP) was measured using the TR jet. The most important factor in determining the systolic pressure is the method of right atrial (RA) pressure measurement. The RA pressure was measured considering the inferior vena cava size and respiratory variation. RA pressure was considered 5 mm if the inferior vena cava is normal sized. For dilated inferior vena cava with normal respiratory variation, an assumed constant of 10 mm was employed. For a dilated inferior vena cava without respiratory variation, an assumed constant of 15 mm was used [8].

Left ventricle end systolic dimension (LVESD) and left ventricle end diastolic dimension (LVEDD) are measured at the level of the LV minor axis, approximately at the mitral valve leaflet tips from the parasternal long-axis acoustic window [9]. Volumetric biplane Simpsons' method was used for evaluation of left ventricle ejection fraction (LVEF) [9].

Statistical analysis

Chi-square analysis was used, considering TR and MR as categorical variables. Single and multiple variable analysis was performed, using Student's t-test for continuous variables and χ^2 test for categorical variables to compare the changes in TR and MR before and after implantation of RV lead in group 1 versus group 2. Same analysis was done to compare the changes in TR and MR in ICD vs. PPM groups. All tests were two tailed with $P = 0.05$ considered significant. We used SPSS for Windows version 14.0 (SPSS Inc., Chicago, IL, USA).

Results

Baseline characteristics

A total of 206 patients were enrolled in this study. Demographic, pacing and echocardiographic characteristics of the studied patients are depicted in Table 1. The mean age of the patients was 74 ± 14 years, and the follow-up duration was 29 ± 19 months.

The effect of RV pacing on TR

TR worsened by at least one grade after lead insertion in 44.7% of patients. Prior to implant (Fig. 1), 34.5%, 45.1%, 16.0%, and 4.4% of patients had absent or trace, mild, moderate, or severe TR. Post-implant, the respective frequencies were 13.6%, 52.9%, 23.8%, and 9.7% (chi-square 54.6, $P < 0.001$). In regard to the type of the RV lead, the TR worsened at least one grade in 42.5% ($n = 51$) in the PPM group and in 46.5% ($n = 40$) in the ICD group. There was no statistical difference between the 2 groups ($P = 0.56$). RV pacing dependence had no effect on the worsening of TR severity (39% high frequency of RV pacing vs. 45% low frequency of RV pacing; $P = 0.52$). Also the type of RV pacing had no effect on the worsening of TR severity (43.2% VVI vs. 45.6% DDD; $P = 0.5$).

The effect of RV pacing on MR

Prior to implant 44.5%, 33%, 15.0%, and 8% of patients had absent or trace, mild, moderate, or severe MR. Post-implant, the respective frequencies were 36.6%, 23.6%, 31.1% and 8.5%. There were trends of worsening of MR, but were not statistically significant ($P = 0.06$; Fig. 2). This is related to heterogeneous response to RV