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Impact of Transvenous Ventricular Pacing Leads on Tricuspid Regurgitation in Pediatric and Congenital Heart Disease Patients

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Abstract

Introduction—Transvenous ventricular pacing leads across the tricuspid valve may cause or exacerbate tricuspid regurgitation (TR). The literature in adults is inconclusive and no studies have investigated the association between pacing leads and TR in children or congenital heart disease patients.

Methods and Results—A retrospective chart review was conducted at a large children's hospital, yielding 123 patients with initial placement of a transvenous lead across their tricuspid valve that had adequate echocardiographic data for review. The median age was 16 years (range 2–52) at time of lead placement. The pre-procedure echo was compared both to the first echo after lead placement and the most recent echo. Median time was 242 days from implant to first echo, and 827 days to most recent echo. There was no difference in TR between the pre-procedure echo and first follow-up echo (p=NS). However, TR was more likely to progress mildly between the pre-procedure echo and the most recent echo ($p<0.02$) with a mean increase from 1.54 to 1.69 on a 0 to 4 ordinal scale. There were 76 pts (62%) with CHD. Mean pre-procedure TR was 1.82 in right-sided valvular CHD (e.g., tetralogy of Fallot, repaired AV canal) vs. 1.43 without right-sided CHD ($P<0.01$).

Conclusions—In patients with transvenous ventricular leads across the tricuspid valve, echocardiography demonstrates a small, but statistically significant change in TR. The detected change is minimal, suggesting that there is little impact of transvenous leads on TR, even in growing children or patients with right-sided structural heart disease.

Keywords

Pediatric; transvenous; pacemaker; implantable cardioverter-defibrillator (ICD); tricuspid regurgitation; cardiac growth; echocardiogram

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Introduction

The first description of endocardial transvenous lead placement was written in 1959 (1). The procedure became widespread shortly thereafter and since then, the prevalence of intracardiac leads has grown enormously. Although the majority of leads are still placed in adults, children represent an important subgroup of patients who receive transvenous systems and consensus indications have been developed for lead placement in pediatrics (2). As the annual number of pediatric pacemaker and implantable cardioverter-defibrillator leads increases, the long-term effects of these leads in pediatrics merit further investigation. With the exception of rare anatomy with ventricular transposition or leads placed in the coronary sinus, all intracardiac ventricular leads cross the tricuspid valve (TV). However, despite the increasing number of patients who live with leads across their valve, the literature in adults is inconclusive on whether these intracardiac leads cause clinically relevant tricuspid regurgitation (TR).

Case reports in adults have noted severe TV regurgitation with an intracardiac lead across the valve (3,4). A retrospective study of 374 adults after transvenous pacemaker lead placement across the TV showed increased prevalence of significant TR when compared with age and sex-matched controls (5). However, one prospective study suggested that transvenous pacemaker leads do not exacerbate TR (6).

There is a risk of tricuspid regurgitation in children by mechanisms similar to those postulated in adults. These mechanisms include lead adherence to the valve tissue, laceration of valve tissue and changes in ventricular activation timing causing poor closure of the tricuspid valve (4–6). In addition, normal growth in children changes the heart's size. Adhesions of the lead to the valve apparatus may cause or exacerbate tricuspid regurgitation in patients who have had transvenous pacemaker leads placed before their heart is fully grown. Finally, children have a much higher prevalence of congenital heart disease than adults. The combination of right-sided congenital heart disease and lead-induced tricuspid regurgitation has not been published in the literature.

Transthoracic echocardiography has been the primary modality for quantifying tricuspid regurgitation in children for over 20 years (7). It has been previously described in adults in the setting of transvenous pacemaker leads (4–6). The purpose of this study was to determine whether transvenous pacemaker leads are associated with increased echocardiographic findings of tricuspid regurgitation.

Methods

Study Design

The study was a retrospective chart review at a tertiary children's hospital. We reviewed all patients at our hospital that had a transvenous lead placed across their tricuspid valve from January 1, 2000 until December 31, 2006. From this group, patients qualified for inclusion if they had at least one transthoracic echocardiographic study ("echo") before the placement of their transvenous lead and at least one echo afterwards. Although evaluating TR is a standard component of our echo protocol, patients that did not have tricuspid regurgitation

evaluated by echo on at least one pre-procedure echo and at least one post-procedure echo were excluded.

Data Collection

Permission for data review was obtained from our Institutional Review Board. The paper and electronic medical records, internal departmental charts, and departmental databases were cross-referenced for each patient. Information on clinical details, lead placement, echocardiograms, and electrocardiograms were tabulated for review.

All echocardiograms at our institution have a final report on file that grades the severity of tricuspid regurgitation as an ordinal variable: 0 – Absent, 1 – Trivial, 2 – Mild, 3 – Moderate, 4 – Severe. The date of lead placement was identified for each patient and the pre-procedure echo that was closest in time to that echo was identified. The first post-procedure echo was also identified. Finally, the most recent echo in our database was identified. A TR value was obtained from each of these echos based on the scale noted above. A subset of 10% of all echos evaluated in our study was independently reviewed by a blinded pediatric echocardiographer to ensure internal consistency.

All electrocardiograms (ECGs) at our institution have a final report on file. From those final reports, the ECGs can be categorized as demonstrating native AV conduction, dual-chamber pacing, ventricular-pacing, or an intrinsic ventricular rhythm. For each echocardiogram included in our study, we evaluated the ECG obtained at or near the time of echo. Each ECG has been read by a pediatric cardiologist and a 10% subset was independently reviewed by a blinded pediatric electrophysiologist to ensure internal consistency.

Height and weight were gathered from the clinical data on the echocardiogram report. If height and weight were not available, the clinical chart was examined for a recorded height and weight near that time. Rarely, a height was not recorded at a clinic or echo visit. In these cases, heights were tabulated from other visits and data was extrapolated using standard growth charts available from the Centers for Disease Control and Prevention (8). Body surface area (BSA) was calculated using the Mosteller formula (9).

Statistical analysis

Because regurgitation is an ordinal variable, changes were evaluated over time using the nonparametric Wilcoxon signed-rank test. A paired t-test was used to compare height, weight and BSA. For comparisons of TR between multiple groups, the Kruskal-Wallis test was applied.

Results

Patient demographics

At our institution, 1106 patients had pacemakers or ICDs placed between 2000 – 2006. Of those, 123 patients underwent initial transvenous placement of a pacemaker or ICD lead across their tricuspid valve without previous leads in place across the TV and met our echo inclusion criteria. The median age was 16 years at time of lead placement with a range from 2 years to 52 years old. ICD leads were placed in 68 of these patients (55%) and pacemaker

leads were placed in 55 patients. Only 2 of these pacemaker leads were part of a biventricular lead system (1.6% of all procedures). The median time of follow-up was 242 days from the implantation procedure to the first echo and 827 days from the implantation procedure to the most recent echo. Table 1 shows median values for height, weight and BSA at the time of lead implantation and at the time of the last echo.

Echocardiography results

There was no difference in TR between the pre-procedure echo and first follow-up echo (p=NS). However, TR was more likely to progress between the pre-procedure echo and the most recent echo, with a mean increase from 1.54 to 1.69 on the 0 to 4 ordinal scale ($p<0.02$). There is no difference in mean duration of follow-up for those whose TR gets worse versus those whose TR stays the same or gets better (p=NS). There is no difference in TR when ICD or pacemaker leads are considered independently. No patients with moderate or severe TR at their final echo had significant right ventricular hypertension.

The TR increased by two ordinal levels in 4 patients (3%). In all 4 patients, it increased from absent TR to mild TR. The TR increased by one ordinal level in 27 patients (22%). Of these 27, the TR changed from absent to trivial in 1 patient, from trivial to mild in 18 patients and from mild to moderate in 8 patients. There were no patients whose TR increased to severe.

The TR was unchanged in 77 patients (63%). Of this group, 1 patient had no TR, 29 patients had trivial TR and 47 patients had mild TR.

In 15 patients (12%), the TR improved. 3 patients (2.4% of all patients) improved by two ordinal levels, all from mild TR to absent TR. 12 patients improved by one ordinal level. Of these, 10 improved from mild to trivial TR, one improved from moderate to mild TR, and one improved from severe to moderate TR.

Potential impact of age on tricuspid regurgitation

Age did not predict an increase in regurgitation. Patients who were under the age of 18 showed no significant difference between their pre-procedure echo, where the TR had a mean of 1.47, versus their last echo where the mean was 1.63 (p=NS). Patients age 18 or older also showed no significant difference with a pre-procedure mean TR of 1.64 and a mean TR on the last echo of 1.76 (p=NS).

Potential impact of congenital heart disease on tricuspid regurgitation

There were 76 patients (62%) with congenital heart disease (CHD). Of those, 35 patients (46%) had right-sided disease, consisting of 27 patients with Tetralogy of Fallot, 6 patients with an atrioventricular canal, and one patient with Ebstein's anomaly of the tricuspid valve. This group also contained one patient with {S,L,L} transposition of the great vessels, status post a Senning procedure and RV to PA conduit. With this repair, the patient had a systemic mitral valve and a ventricular lead across the tricuspid valve. Before the procedure, patients with right-sided CHD had a mean pre-procedure TR of 1.82, compared to 1.43 in those patients who did not have right-sided CHD ($p<0.01$). In patients with right-sided CHD, the mean TR did not change significantly from the pre-procedure echo to the last echo (1.82

versus 1.94, p=NS); however, in patients who did not have right-sided CHD, the TR increased from 1.43 to 1.59 (p=0.04) from the pre-procedure echo to the last echo. There was no significant change in TR from the pre-procedure echo to the first follow-up echo in any subgroup.

Impact of atrioventricular conduction and synchrony on tricuspid regurgitation

At the time of the pre-procedure echocardiogram, 92 patients (75%) were conducting across the intrinsic AV node, 21 patients (17%) were being dual-chamber paced and 10 patients (8%) were either ventricular-paced or their baseline rhythm was an intrinsic ventricular rhythm. All patients who were paced at the pre-procedure ECG were paced with epicardial systems. At the time of the last echocardiogram, 79 patients (64%) were conducting across their own AV node, 35 patients (28%) were being dual-chamber paced and 9 patients (7%) were either ventricular-paced or their baseline rhythm was an intrinsic ventricular rhythm. A Kruskal-Wallis test was not significant for differences between patients with intrinsic conduction, dual-chamber pacing or ventricular rhythms when comparing the pre-procedure TR to the first post-procedure TR (p=NS) or when comparing the pre-procedure TR to the most recent echo TR (p=NS).

Discussion

In patients with transvenous ventricular leads across the tricuspid valve, early echocardiography, with a median follow-up of less than a year, demonstrates no change in TR after lead placement. However, after extended follow-up with a median of more than two years, the increase in TR is statistically significant. On the ordinal scale of 0 to 4, the pre-procedure TR had a mean of 1.54, which is between the trivial and mild scores. At our last follow-up, the mean score had increased to 1.69, which is still in that same range of trivial to mild. This is not likely to be clinically significant. There were no extreme changes from absent or trivial TR to moderate or severe TR in any of our patients.

One reason we hypothesized why TR may increase in children is because children grow after lead placement (10, 11). In the present cohort, overall change in height was 9.5 cm from lead implantation to the last echo; however, in patients less than 18 years of age at the time of lead placement, the median change in height was 10.9 cm. In patients who were 18 or older at the time of lead placement, the median change was only 3cm. This suggests that most of the change in height in our population occurred in those patients who were under 18 at the time their lead was placed. The changes in weight and BSA similarly reflect more growth before age 18. However, despite evidence for growth, the subset of patients less than 18 years old were not more likely to have an increase in the TR jet. This suggests that linear growth does not play a substantial role in the development of TR in young patients who have intracardiac leads across the tricuspid valve.

Patients with right-sided congenital heart disease have more TR prior to receiving a transvenous lead that those who have no right-sided congenital heart disease, although the mean for both was within the trivial-to-mild range. Patients without right-sided disease did develop slightly more tricuspid regurgitation after lead placement; however, in this group the difference was slight, changing from a mean of from 1.43 to a mean of 1.59, both well

within the trivial-to-mild range. Even after lead placement for a prolonged period of time, the patients without right-sided disease had less mean TR than the patients with right-sided disease did at any point.

It has been hypothesized that ventricular pacing and the subsequent changes in ventricular activation timing cause poor closure of the tricuspid valve. In our study, we compared three groups to each other: those patients who had intrinsic AV node conduction, those who were dual-chamber paced, and those who had ventricular pacing or an intrinsic ventricular rhythm. Among all of these groups, there were no differences in the TR when the pre-procedure echos, the first post-procedure echos and the last echos were compared. Discoordinated ventricular activation due to pacing is not significantly changing the degree of TR during the follow-up interval in this group.

Overall, pediatric and CHD patients who have transvenous pacemaker or ICD leads placed across their tricuspid valve have a statistical increase in tricuspid regurgitation; however, there does not seem to be a clinically significant change in tricuspid regurgitation during the follow-up period. These findings suggest that those patients with transvenous leads across the tricuspid valve who develop a more significant increase in tricuspid regurgitation should be evaluated carefully for other pathologies that might exacerbate tricuspid regurgitation.

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Table 1

Median height, weight and body surface area (standard deviation in parenthesis).

Age (in years)		<18	≥18	All ages
Height (cm)	At time of procedure	154 (27.3)	168 (11.6)	164 (23.6)
	At last echo	165.5 (24.8)	168.5 (11.6)	167 (20.6)
	p-value*	<0.01	0.04	<0.01
Weight (kg)	At time of procedure	48.2 (23.7)	66 (15.9)	59.1 (23.1)
	At last echo	58 (25.1)	67.8 (17.2)	63.5 (23.1)
	p-value*	<0.01	<0.01	<0.01
Body surface area (m^2)	At time of procedure	1.48 (0.47)	1.76 (0.22)	1.66 (0.43)
	At last echo	1.63 (0.46)	1.78 (0.24)	1.72 (0.40)
	p-value*	<0.01	0.01	<0.01

*Two-tailed paired t-test used for comparisons