# Comparative Chronic Valve and Venous Effects of Lumenless versus Stylet-Delivered Pacing Leads in Patients with and Without Congenital Heart

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***Background:*** *Standard, 5–7-Fr diameter pacing leads (PLs) can adversely affect atrioventricular valve (AVV) and venous (superior vena cava [SVC], innominate [INN]) integrities. Although chronic pacing/sensing performances have been reported on the steroid-eluting, lumenless, 4.1-Fr PL (Model 3830, Medtronic Inc., Minneapolis, MN, USA), comparative valve and venous effects are largely unknown.*

***Methods:*** *Patients (n = 134) were divided into two PL groups: Group 1 (n = 65, Model 3830) and Group 2 (n = 69, various 5–7 Fr models) and followed up to 9 years postimplant. Patient demographics, clinical findings, valve function, and venous dimensions were reviewed. Statistical significance was defined as P < 0.05.*

***Results:*** *Patient implant age (mean 16.4 years vs 17.3 years), presence of congenital heart defect (CHD), and preexisting valve issues were comparable between groups. New or worsening valve insufficiency occurred in 12% of Group 1 patients (mean follow-up 4.3 ± 2.8 years) and 27% of Group 2 patients (mean follow-up 6.2 ± 3.5 years; P < 0.05). Significant SVC or INN narrowing was found in 11 % of Group 1 and 24% of Group 2 patients (P = 0.0004). All Group 1 patients <12 years of age showed normal while 50% of those from Group 2 exhibited stunted SVC or INN growth (P < 0.05).*

***Conclusion:*** *The lumenless, 4.1-Fr diameter PL offers improved clinical benefits, better AVV integrity, and venous development compared with larger 5–7-Fr diameter PL and should be considered especially in younger patients with/without CHD. (PACE 2015; 38:1343–1350)*

***electrophysiology, clinical, pacing, pediatrics, echocardiography***

## Introduction

The design of transvenous pacing leads (PLs) has significantly evolved over the past decade and has facilitated usage in children and patients with congenital heart defects (CHDs). The advanced technology also allows transvenous pacing in younger patients with smaller body surface areas. However, to date, there have been limited data on valvular and vascular effects of transvenous ventricular PLs in the young. During transvenous lead implant, as the ventricular PL is inserted through the atrioventricular valve (AVV; tricuspid valve in normal hearts and mitral valve in patients with transposition of the great arteries), there are potential risks of causing valve damage resulting in fibrotic reactions leading to stenosis or insufficiency in the long term. Studies among adult patients have shown that there is an increased prevalence of tricuspid valve insufficiency (TI) or worsening of any preexisting TI among patients who receive transvalvular leads, ranging between 25% and 29% of older patients with pacemakers.[1–5] Webster et al. reported a 25% incidence of TI worsening following pacemaker implant in pediatric and CHD patients.[6]

Other adverse effects of transvenous PLs include vascular complications, such as thrombosis and stenosis, with reported incidences from 35% to 64%.[7,8] Although most venous complications are subclinical, pacemaker-induced superior vena cava (SVC) syndrome has also been reported.[9–11] The pathophysiology of PL-related venous complications has been related to mechanical stress from the pacemaker lead and lead-vascular interactions which predispose the vessel wall to inflammation, neointimal proliferation, and progressive fibrotic reactions.[12] These concerns especially apply to growing children whose venous structure is still developing.

In 2005, the lumenless 4.1-Fr diameter, catheter-delivered PL, Model 3830 (Medtronic Inc., Minneapolis, MN, USA) was approved by the United States Food and Drug Administration

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for use in all patients. Compared to conventional hollow-core, stylet-delivered PLs with diameters between 5 Fr and 7 Fr, the 4.1-Fr diameter lead has a solid core. Previous reports have shown no differences in comparative lead sensing or pacing performances.[13] Several studies have reported excellent short-term as well as chronic lead performance (pacing/sensing) outcomes of the lumenless 4.1-Fr diameter M3830 leads in pediatric and CHD populations.[13–18] Unfortunately, there is limited comparative information on AVV integrity or vascular (SVC/innominate [INN]) dimensions between these lead designs and none in the young or those with CHD.[18,19] The aim of our study was twofold: to compare any AVV effects, either stenosis or insufficiency, following the implantation of the lumenless 4.1-Fr diameter leads compared with conventional 5–7-Fr diameter leads among children and young adults with and without CHD, as well as to evaluate any chronic adverse vascular effects on the SVC/INN vein luminal diameters and growth.

## Materials

Prior to the introduction of the lumenless lead in 2005, transvenous PLs implanted in our institution typically consisted of established stylet-delivered, hollow-core designs from various manufacturers, ranging from 5 Fr to 7 Fr diameters. Since 2002, and for the inclusion of this study, all of these implanted leads were bipolar, steroid-eluting, and active-fixation helical designs from multiple manufacturers (Medtronic Inc.; St. Jude Medical, St. Paul, MN, USA; Guidant/Boston Science, St. Paul, MN, USA). The lumenless 4.1-Fr diameter lead (Medtronic M3830) is also a bipolar, steroid-eluting helical design with intracardiac delivery via a steerable peel-away sheath (Select Secure® system). The solid core of the M3830 permits the smaller 4.1 Fr diameter. Since August 2005, in most cases, the Medtronic M3830 lead was implanted at our institution. All leads (4.1 Fr and 5–7 Fr diameters) were 59 cm or 69 cm in length and of a polyurethane/silicone insulation design. Readily visible differences in lead diameters are illustrated in Figure 1.

## Methods

This is a retrospective chart review of all patients who received transvenous pacemakers in our institution during the current era of steroid-eluting PLs, from 2002 to 2014. The study was approved by Detroit Medical Center and Wayne State University School of Medicine Human Investigation Committee. For the purpose of determining the lead effects on AVV function, patients with only transvenous atrial leads were excluded. To determine any vascular effects on SVC/INN flow and dimensions, all transvenous leads (atrial and ventricle) were included. Patients were divided into two groups: Group 1, all patients who received the 4.1 Fr (M3830), and Group 2, patients with standard 5–7-Fr diameter leads. Further grouping of the 5–7-Fr diameter leads into subsets was not performed due to relatively small numbers in each subset as indicated below. Patient demographic, cardiac anatomy, indication for pacemaker implantation, echocardiographic/Doppler findings of preexisting AVV stenosis or insufficiency information were collected at baseline and compared between the two groups.

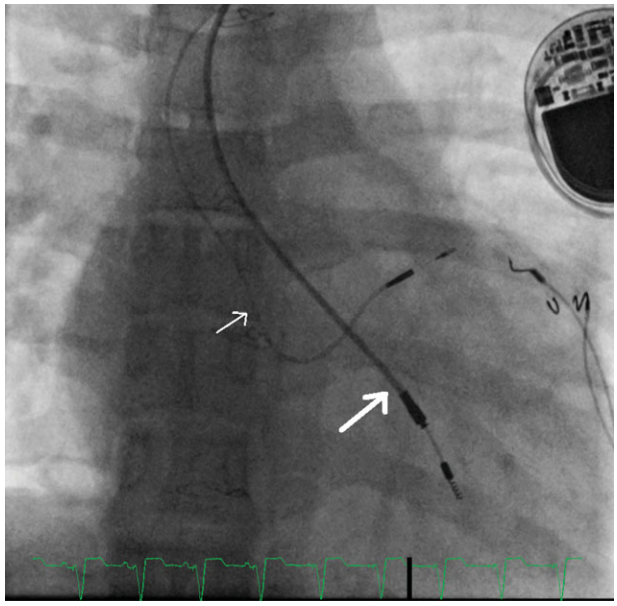

**Figure 1.** *Exemplary fluoroscopic image in the antero-posterior projection illustrating diameter differences between 4.1-Fr and 6-Fr pacing leads. The patient is post repair of d-TGA with the intraatrial baffle (Mustard) surgery. The 4.1-Fr diameter lead (small arrow) is implanted in the venous (left) atrium while the 6-Fr diameter lead (large arrow) in the venous (left) ventricle. Note two previous epicardial leads (one fractured helix) on right side of the image. d-TGA = dextro transposition of great arteries.*

### AVV Effects

Data on AVV stenosis or insufficiency were collected by review of transthoracic echocardiogram with Doppler studies. A subset of 20% of all studies was independently reviewed by two pediatric echocardiographers, blinded to PL design, to ensure intraobserver consistency. AVV insufficiency (AVVI) was assessed on the basis of color and spectral Doppler utilizing the standard

apical four-chamber view, in accordance with the recommendations of the American Society of Echocardiography.[20] AVVI was classified as none/trace, mild, moderate, and severe. Post-AVVI was defined as worsening of AVV competency postimplant when compared with preimplant values. The evaluation of AVV stenosis was performed using transvalvular velocity on the continuous wave Doppler.[21] The degree of valve stenosis was graded on a scale of none/trivial, mild, moderate, and severe. The worsening of AVV function was defined as an increase in the degree of valve dysfunction severity of at least one grade. Tricuspid valve function was evaluated in patients with normal atrioventricular (AV) concordance and mitral valve function for patients with AV discordance, for example, patients with the Mustard intraatrial baffle surgical repair for dextro (d-) or those with congenital corrected (cc-) transposition of the great arteries [TGA]). Echocardiogram/Doppler studies performed within 1 month prior to pacemaker implantation were reviewed and analyzed in all patients as a baseline. Postimplant echocardiograms performed at 1 month and at least annually thereafter were reviewed and analyzed for up to 9 years.

### Systemic Venous Effects and Dimensions

The luminal diameters of the INN vein and SVC were measured online from venograms performed during cardiac catheterization studies at implant (Syngo Dynamics—Siemens Healthcare, Forchheim, Germany). Since a previously published study has shown that vascular diameters in growing children are more closely correlated with height rather than weight or body surface area, the venous diameters were measured in four regions, distal INN, mid-INN, INN-SVC junction, and mid-SVC, and compared to the expected value in relation to height according to the published formula: distal INN (2.07 + 0.081 height), mid-INN (1.73 + 0.086 height), INN-SVC (2.4 + 0.072 height), and mid SVC (3.2 + 0.093 height).[22] For patients over the age of 12 years, age-related changes in venous diameters were not observed and assumed to approximate adult values.[22] Conventionally, significant narrowing of the SVC or INN was defined as the postimplant vessel luminal diameter at the site of obstruction more than 60% of the more distal vessels.[23] Additional assessment of the SVC/INN flow patterns were obtained during pre- and all postlead implant echocardiogram/Doppler studies.

In evaluation of lead-related systemic venous effects, patients who received any transvenous lead (atrial, ventricular, or both) were subdivided depending on lead number and type. In this manner, vascular effects among patients with either one or two transvenous 4.1-Fr PLs were compared with effects from patients with either one or two standard 5–7-Fr leads as well as from patients with both the lumenless and standard leads.

### Ventricular Lead Implantation

Indications for pacemaker implant followed standard guidelines for pediatric and congenital heart patients. Prior to the pacemaker implantation, all patients underwent a standard left and right heart catheterization study to evaluate hemodynamic status and venograms for vascular patency, especially among those with repaired CHD. Subclavian veins or axillary veins were entered percutaneously. PLs were positioned to obtain optimal pacing threshold, sensing, and contractility.[24] Specific attempts at "His/para-His" pacing were not performed due to concerns for elevated pacing thresholds.[25,26] Follow-up data up to 9 years after implant included information on AVV function during follow-up echocardiogram/Doppler studies and SVC venogram during any subsequent cardiac catheterizations.

### Statistical Analysis

Statistical analysis was performed using SAS version 9.1 (SAS Institute Inc., Cary, NC, USA). Since AVV stenosis or insufficiency is a categorical variable, changes in the degree of valve stenosis or insufficiency were evaluated over time using the nonparametric Wilcoxon signed-rank test. A paired *t*-test was used to compare the demographic data. Agreement between the two echocardiographers' reading was calculated using the kappa ($\kappa$) statistic for categorical variables. Variables with a lower 95% confidence limit of $\kappa > 0.4$ were considered to have acceptable agreement. Finally, paired and nonpaired *t*-test was used to compare significance narrowing of systemic veins. Statistical significance was defined as $P < 0.05$.

## Results

During the time frame of this study, 255 PLs were implanted in 182 patients, of whom 134 patients received ventricular leads. Patient ages ranged between 5 years and 45 years (mean 16.8 ± 8.3 years), with 75 (56%) males. There were 65 patients who received the 4.1-Fr M3830 lead (Group 1) and 69 patients with standard 5–7-Fr diameter leads (Group 2). Of these 69 patients with leads from Group 2, a breakdown of specific lead diameters showed that diameters ranged from 5 Fr to 5.9 Fr (n = 22), 6 Fr to 6.9 Fr (n = 30), and 7 Fr to 7.4 Fr (n = 17). Relatively small numbers per subset of Group 2 precluded separate comparisons with the 4.1-Fr diameter lead group. Patient demographic data in both groups were

**Table I.**

Transvenous Ventricular Leads Demographics

| | Group 1 | Group 2 | P Value |
|---|---|---|---|
| n = 134 | 65 | 69 | NS |
| Age at implant (years) † | 16.4 ± 7.8 | 17.3 ± 8.7 | NS |
| Weight at implant (kg) † | 59.6 ± 25.7 | 57.2 ± 22.5 | NS |
| Male: female | 39:26 | 36:33 | NS |
| Nonstructural CHD (n = 44) | 21 | 23 | NS |
| Structural CHD (n = 90) | 49 | 41 | NS |
| Septal defects | 8 | 7 | NS |
| TOF/PA-VSD | 6 | 8 | NS |
| d-TGA s/p arterial switch | 5 | 4 | NS |
| d-TGA s/p Mustard palliation | 17 | 13 | 0.04 |
| cc-TGA | 6 | 5 | NS |
| Others‡ | 7 | 4 | NS |

†Mean ± standard deviation.
‡Other forms of CHD include congenital aortic stenosis, subaortic stenosis, truncus arteriosus, Ebstein's anomaly of tricuspid valve.
cc = congenitally corrected; cc-TGA = congenitally corrected transposition of great arteries; CHD = congenital heart defects; d-TGA = dextro transposition of great arteries; PA-VSD = pulmonary atresia, ventricular septal defect; TGA = transposition of great arteries; TOF = tetralogy of Fallot.

comparable and are shown in Table I. For review of vascular dimensions, all patients (those with atrial plus ventricular leads) were studied. However, only those patients who received ventricular leads were included for valvular issues. Among the 134 patients, 90 (67%) had undergone repair of various anatomical CHD such as d-TGA with an atrial baffle (Mustard procedure) AV or V septal defects, tetralogy of Fallot with or without pulmonary valve replacement, and hypertrophic cardiomyopathy with myomectomy. Patients without surgically repaired CHD included those with congenital complete AV block, congenital long QT syndrome, and hypertrophic cardiomyopathy. Presence of congenital heart conditions and AVV morphologies were comparable between groups. There were no acute complications observed during lead implantation in either group. One lead in Group 1 (4.1 Fr) and four leads in Group 2 (Standard 5–7 Fr) required replacement secondary to lead fracture or displacement. These incidents were detected upon follow-up and occurred beyond 6 months and within 2–3 years postimplant. There were no other complications noted. Since the larger diameter leads were utilized prior to 2005 in our institution, longer follow-up data, as expected, were available compared to the lumenless 4.1-Fr lead that was utilized only after August 2005. Therefore, comparative 9-year (August 2005–2014) follow-up was available in all 69 patients from Group 2 but only 15 patients from Group 1. At least 3-year follow-up postimplant data were available on all patients.

### AVV Effects

To diminish observer variability, a subset of echocardiographic images (20%) were blindly and independently reviewed by two echocardiographers demonstrating nearly perfect agreement ($\kappa$ = 0.85 and 0.89) with the initial echocardiographic/Doppler interpretation of AVV effects. In comparison between groups, nine (14%) of patients in Group 1 and 11 (16 %) of Group 2 patients exhibited some preexisting AVVI (P = NS). During the comparative 9-year postlead implant follow-up interval, review of valve integrities showed that eight (12%) patients in Group 1 exhibited worsening of AVVI but without symptoms while 19 (27%) patients in Group 2 ($P < 0.05$) had clinically symptomatic worsening of AVVI (Fig. 2). Of these latter patients, three underwent surgical tricuspid valvuloplasty due to hemodynamically significant right atrial dilatation and refractory atrial arrhythmia. There were no differences in AVV assessment between the preprocedure and first follow-up echocardiogram/Doppler study at the 1-month interval (P = NS). Onset of AVVI was noted within 27.4 ± 15.8 months at follow-up: five patients with preexisting AVVI from Group 1 developed worsening of AVVI on one ordinal scale (mild to moderate or moderate to severe), compared to all patients from Group 2 ($P < 0.05$). While there were no patients from Group 1, three patients from Group 2 exhibited extreme AVVI more than one ordinal scale. New onset AVVI, all detected within the first 3 years postimplant, occurred in four patients from Group 1 and eight patients from Group 2. The change in AVVI between two groups is shown in Table II. There were no significant changes in ventricular function at times of valve insufficiency detection. AVV stenosis was not detected in any patient at pre- or postimplantation evaluations.

### Systemic Venous Effects and Lead Number

Since many patients with ventricular also had atrial leads, venous effects were evaluated based on lead number and type. SVC or INN luminal diameters more than 60% narrowing were defined as having obstruction.[23] All patients had baseline venograms performed at the time of lead placement. Postlead implant venograms to

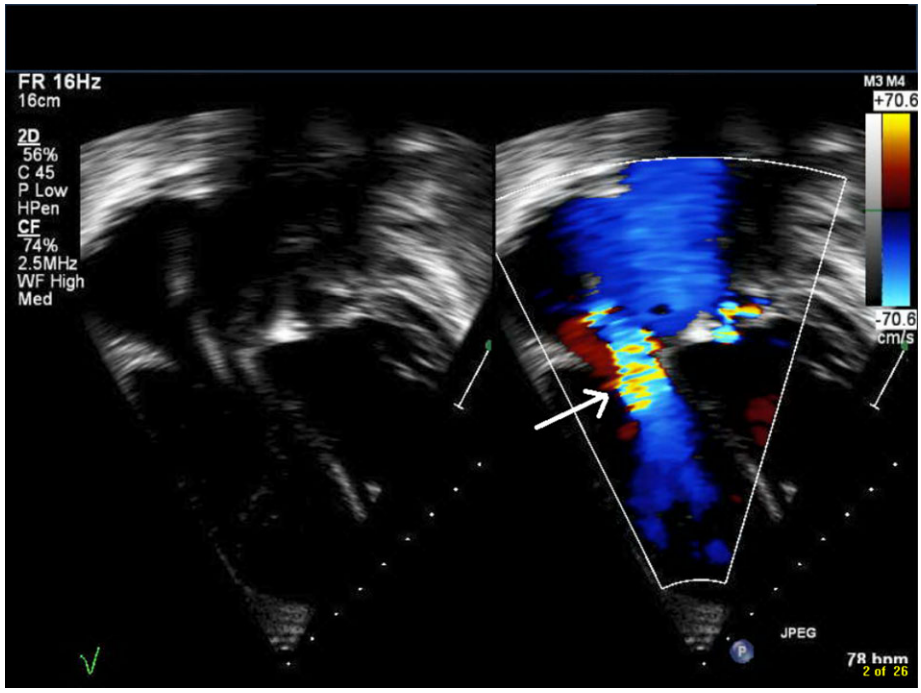


**Figure 2.** *Right: Transthoracic echocardiogram/Doppler study in the four-chamber view, demonstrating a moderate-severe degree of tricuspid valve insufficiency (arrow) associated with a 6-Fr diameter lead. Left: With the Doppler signal off, the 6-Fr diameter pacemaker lead is readily visible traversing through the tricuspid valve.*

evaluate vascular patency were obtained at the time of any required generator change or other catheterization studies performed for clinical reasons. Due to the time frame of this review, all 69 patients (100%) who had received standard 5–7-Fr PLs (Group 2) underwent postlead implant venograms at mean follow-up interval of 6.8 ± 2.6 years. There were 61 patients with either one or two transvenous 4.1-Fr PLs (Group 1). However, due to the shorter postimplant time intervals, and limited availability of repeat catheterization studies, venograms were available in only 35 (57%) patients from this group at mean follow-up interval of 4.2 ± 1.8 years.

The overall incidence of angiographically significant venous obstruction was 21% among all patients. As a comparison between lumenless versus standard leads, there was significantly less venous obstruction associated with the 4.1-Fr diameter lead (P = 0.0004). Of those 35 patients from Group 1 with follow-up venograms, there were 10 patients with only one 4.1-Fr diameter lead and none of these patients showed any evidence of venous obstruction. On the other hand, among patients with two 4.1-Fr diameter leads (N = 25), four (16%) met criteria for obstruction. Among the 69 patients with the larger diameter (5-6-7 Fr) leads, 33 had received one and 63 patients had two leads. Of these, eight of 33 (24%) and 15 of 63 (23%) showed obstruction (Fig. 3). Finally there were 18 patients with both the lumenless as well as standard leads. Of these, a postimplant venogram was performed in all patients at a mean follow-up interval of 5.4 ± 2.3 years. This demonstrated obstruction in four of 18 (22%) patients.

In comparison among lead type and number, there was significantly less obstruction associated with a single 4.1 Fr versus single 5–7-Fr leads (P = 0.0002) as well as between two 4.1 Fr versus two 5–7-Fr diameter leads (P = 0.0002). All 10 patients with 4.1-Fr leads implanted when they were less than 12 years of age showed no difference in luminal venous diameters on follow-up, adjusted for height, between pre- and postimplant, while six of 12 patients (50 %) younger than 12 years with 5–7-Fr diameter leads showed stunted growth of the SVC and INN veins, as evident by less than a 60% diameter increase upon follow-up venograms (P < 0.05).

## Discussion

Compared with pacemaker implantation among older patients with normal cardiac anatomy, PLs in children and patients with CHDs

**Table II.**

Atrioventricular Valve Insufficiency Data

| | Group 1 | Group 2 | P Value |
|---|---|---|---|
| n = 134 | 65 | 69 | |
| Preexisting AVVI | 9 (14%) | 11 (16%) | NS |
| AVVI worsening postimplant | 8 (12%) | 19 (27%) | 0.001 |
| Change in 1 grade | | | |
| • None/trivial to mild | 3 (5%) | 6 (7%) | |
| • Mild to moderate | 4 (6%) | 8(12%) | |
| • Moderate to severe | 1 (2%) | 3 (4%) | |
| Extreme change | | | |
| • None/trivial to moderate | – | 2 (3%) | |
| • Mild to severe | – | – | |

AVVI = atrioventricular valve insufficiency.

often pose a challenge to implanting physicians, due to relatively smaller patient size, venous dimensions, wide anatomic cardiac diversity, and venous anatomy, as well as preexisting valvular issues. Special consideration also needs to be given to this unique population due to somatic growth, requirements for long-term pacing, and potential alterations in the cardiovascular hemodynamics. Advancement in endocardial transvenous leads technology has offered pediatric/congenital heart electrophysiologists improved equipment to perform lead implant in younger patients and those with complex CHD without significant increases in procedure-related short- and long-term

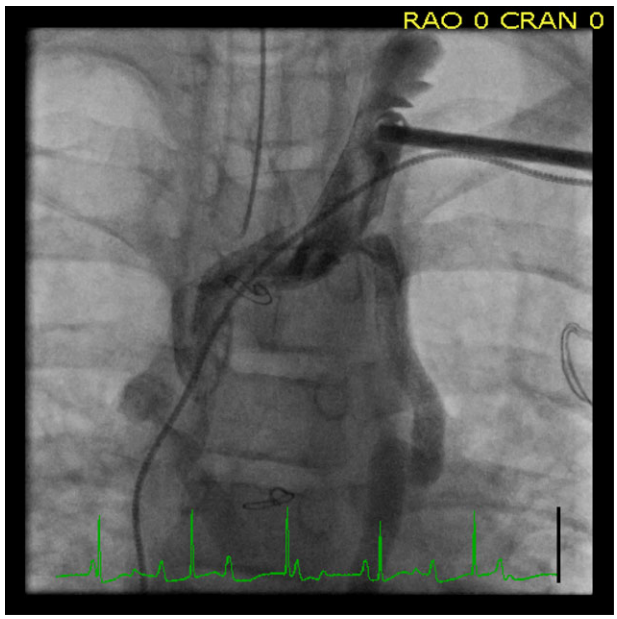


**Figure 3.** *Venous complications associated with use of larger diameter pacing leads in the young is illustrated in this venogram from a patient post-CHD repair viewed in the anteroposterior projection. The 6-Fr diameter lead was implanted when the patient was 9 years of age and the viewed venogram was taken 4 years later. There is severe narrowing and nearly complete occlusion at the superior vena cava-innominate vein junction with compensatory enlargement of both azygous and hemiazygous veins acting to decompress the obstruction. CHD = congenital heart defects.*

complications. Additionally, selective-site pacing and cardiac resynchronization therapy offers better pacing outcomes on cardiac function.[24]

Due to the nature of the coaxial lead design, the 4.1-Fr bipolar lumenless lead's cross-sectional area is reduced by 40% when compared to standard, stylet-delivered, 5–7-Fr diameter leads. This facilitates use in pediatric patients with relatively smaller cardiac sizes, venous dimensions, and AVV cross-sectional areas. The lead diameters are visually different (Fig. 1). The 4.1-Fr M3830 has demonstrated favorable acute and chronic performance with low complication rates.[13–19,27,28] A more recent study by Garnreiter et al. also showed encouraging results for ease of manual lead extraction.[18] Webster et al. have reported the association between PLs and tricuspid insufficiency in children and patients with congenital heart disease.[6] AVVI following ventricular lead placement can occur via multiple mechanisms, including mechanical causes such as lead adherence to the valve tissue and scar formation as well as changes in ventricular activation

timing causing poor valve leaflet coaptation.[1,2,5] Our study further expands this finding by demonstrating worsening of AVVI only after 2–3 years postimplantation and not within the first month. We speculate that late-onset insufficiency is related to chronic lead-tissue interactions, not acute mechanical leaflet tethering.

Our study is the first to directly compare both the chronic impact on AVVs and venous diameters between the 4.1 and standard 5–7-Fr leads in the same cohort of young and congenital heart patients. The overall incidence of AVVI for all leads in our patient population was 20%, similar to the pediatric CHD study published previously.[6] However, this study shows that most adverse valve issues were associated with larger diameter leads. The lumenless lead was associated with less adverse impact on AVV integrity. Due to simple timing logistics, long-term echocardiographic data, up to 9 years postimplant, were not available on all of the 4.1-Fr leads when both lead diameter groups were compared. Since the 4.1 leads first became available in 2005, the mean follow-up in this group was 4.3 years compared to the standard 5–7 Fr implanted prior to 2005 which had the mean follow-up of 6 years. However, worsening in AVVI degree occurred within the first 2 years postimplant, during which data on all leads were available. Therefore, we considered this as adequate long-term follow-up data. Our study did not suggest that change in ventricular function impacted the degree of AVVI since there were no changes in ventricular contractility at the time of detection. Although the majority of worsening AVVI occurred on one nominal scale in our study, patients in the larger diameter 5–7-Fr lead group demonstrated more severe AVVI within a 5-year follow-up interval, with significant hemodynamic impact requiring some patients to undergo tricuspid valvuloplasty or replacement. As indicated above, leads were implanted to achieve optimal ventricular contractility response.[24] Therefore, implant locations were variable along the ventricular septum. However, due to concerns for elevated pacing thresholds, especially among younger patients, none of the leads were implanted to attempt "His bundle/para-His" pacing, so lead insertion in very close proximetry to the septal leaflet did not occur.

Partial venous obstruction is another pacing-related complication associated with transvenous PLs, especially in the young and those with abnormal venous anatomy. Detection of venous obstruction or occlusion can be challenging as patients often do not develop any symptoms initially due to development of collaterals and potential azygous vein decompression. Although significant venous abnormalities can be detected echocardiographically, this is not as sensitive as venography for milder degrees of venous obstruction.[23] In addition, some repaired CHDs preclude accurate echocardiographic evaluations of venous patency.[29]

Our study quantitatively reviewed the chronic systemic venous effects among patients with both the 4.1 and more standard 5–7-Fr diameter PLs alone and in combination. The overall incidence of angiographically significant systemic venous obstruction was 17%, similar to the previously published pediatric cohorts.[23] However, the incidence of narrowing was more prevalent in the standard 5–7-Fr lead group compared to the 4.1-Fr lumenless lead group. Also, this study demonstrates the adverse impact of larger diameter PLs on vascular growth in the young.

### Limitations

A limitation for this study was the smaller number of the Group 1 patients who completed 9-year follow-up echocardiogram/Doppler studies when compared to Group 2 patients. This is a chronological logistics issue that continued clinical evaluations will rectify. All patients in both groups had an echocardiogram performed at baseline and at 1-month follow-up. However, all patients had 3 years and the majority at least up to 4 years of serial echocardiogram/Doppler studies for evaluation of AVV function. Based on clinical findings, it would be anticipated that any chronic AVV change should become evident during this available follow-up period. Unfortunately, due to limited number of patients in the Group 2 lead diameter subsets, further breakdown comparisons with the 4-Fr diameter lead was not feasible to provide any valid statistical significance. So the 5-6-7-Fr diameter leads were grouped together.

### Conclusions

Cardiac pacing initiated in the young inherently is associated with potentially more adverse issues than if pacing is initiated later in life. Recent improvements and advances in PL technology have benefitted pacing applications in the young. The lumenless 4.1-Fr design has previously been shown to be associated with stable chronic pacing/sensing characteristics as well as ease of extraction. This study contributes additional information by demonstrating improved clinical benefits on AVV and venous integrities. This is a very important concept in growing children who require long-term permanent pacing for which any PL-induced adverse AVV issues or delayed or arrest in systemic venous growth as well as obstruction can be associated with clinical morbidities and negatively impact patient well-being.

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