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Impact of Implantable Transvenous Device Lead Location on Severity of Tricuspid Regurgitation

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Abstract

Background—Implantable device leads can cause tricuspid regurgitation (TR) when they interfere with leaflet motion. The aim of this study was to determine whether lead-leaflet interference is associated with TR severity, independent of other causative factors of functional TR.

Methods—A total of 100 patients who underwent transthoracic two-dimensional and three-dimensional (3D) echocardiography of the tricuspid valve before and after lead placement were studied. Lead position was classified on 3D echocardiography as leaflet-interfering or noninterfering. TR severity was estimated by vena contracta (VC) width. Logistic regression analysis was used to identify factors associated with postdevice TR, including predevice VC width, right ventricular end-diastolic and end-systolic areas, fractional area change, right atrial size, tricuspid annular diameter, TR gradient, device lead age, and presence or absence of lead interference. Odds ratios were used to describe the association with moderate (VC width ≥ 0.5 cm) or severe (VC width ≥ 0.7 cm) TR, separately, using bivariate and stepwise multivariate logistic regression analysis.

Results—Forty-five of 100 patients showed device lead tricuspid valve leaflet interference. The septal leaflet was the most commonly affected (23 patients). On bivariate analysis, preimplantation VC width, right atrial size, tricuspid annular diameter, and lead-leaflet interference were significantly associated with postdevice TR. On multivariate analysis, preimplantation VC width and the presence of an interfering lead were independently associated with postdevice TR. Furthermore, the presence of an interfering lead was the only factor associated with TR worsening, increasing the likelihood of developing moderate or severe TR by 15- and 11-fold, respectively.

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SUPPLEMENTARY DATA

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Conclusion—Lead-leaflet interference as seen on 3D echocardiography is associated with TR after device lead placement, suggesting that 3D echocardiography should be used to assess for lead interference in patients with significant TR.

Keywords

Three-dimensional echocardiography; Pacemaker; Intracardiac defibrillator; Tricuspid regurgitation

A growing number of permanent pacemakers (PPMs), implantable cardioverter-defibrillators (ICDs), and cardiac resynchronization therapy (CRT) devices are being inserted each year[1] in the United States, as the indications for device therapy expand.[2] Despite the increasing number of implanted devices, relatively little information is available on how they interact with the tricuspid valve (TV) apparatus. Some studies suggest that they induce tricuspid regurgitation (TR),[3–8] a valvular lesion that is increasingly appreciated to be associated with high morbidity and mortality,[2,9] whereas other studies suggest that they do not.[10–12]

Functional TR, commonly encountered in clinical practice,[13] usually occurs in the presence of an anatomically preserved valve. It is frequently directly associated with an enlarged tricuspid annulus and/or a dilated right ventricle but can also be related to leaflet tethering and right ventricular (RV) remodeling as a consequence of increased right-sided pressures often secondary to left-sided heart disease.[14]

We recently reported on an observational study[15] that described the use of three-dimensional (3D) echocardiography for the visualization of device leads as they traverse the TV. We found that TR was present when implanted device leads were interfering with the normal TV leaflet motion. Although that study showed an association between the presence of a device lead and TR, it did not prove that the lead was a major cause of TR, because preimplantation studies were not available in the majority of patients. In addition, other factors were not considered as potential causes of TR, and the strengths of their associations with the severity of TR were not studied.

Accordingly, in the present study, we sought to determine whether device lead interference with the TV leaflets, as depicted by 3D echocardiography, is an important contributor to TR severity by comparing pre- and postimplantation studies or whether other factors known to increase TR severity, such as RV size and systolic function, tricuspid annular (TA) diameter, systolic pulmonary artery pressure (sPAP), and right atrial (RA) size, are more important predictors of TR in these patients.

METHODS

Patient Population

We retrospectively studied 100 consecutive patients (49 men, 51 women; mean age, 67 ± 16 years; range, 26–93 years) with morphologically normal TVs who had device lead implantation in the right ventricle (PPM, ICD, or CRT with defibrillator or pacemaker) and had undergone complete two-dimensional (2D) and 3D transthoracic echocardiography

before and after device implantation. Fifty-three of these patients were included in our previous publication.[15] Demographic information, date and site of device generator implantation (left vs right chest pocket), and type of device were obtained by chart review. This study was approved by the institutional review board.

2D Transthoracic Echocardiography

Comprehensive 2D and color Doppler evaluation was performed by an experienced sonographer using an iE33 imaging system equipped with an S5 transducer (Philips Medical Systems, Andover, MA). Digital loops were stored and analyzed (Figure 1) offline (Xcelera Workstation; Philips Medical Systems). TR was quantified using vena contracta (VC) width according to published guidelines.[16] It represented the narrowest portion of the TR jet at or downstream from the orifice in mid-systole. The largest VC width obtained from either the RV inflow or apical four-chamber view was reported on both the pre- and postimplantation echocardiograms (Figure 1C). In addition, RA dimensions and area, RV basal and mid-end-diastolic dimensions and fractional area change, sPAP, and RA pressure were recorded according to published guidelines.[17,18]

Maximal RA long-axis and short-axis dimensions were measured at ventricular end-systole, defined as the frame immediately before TV opening, when the RA chamber was at its greatest size in the apical four-chamber view. A straight line was traced connecting both sides of the tricuspid leaflet attachment points. The RA long-axis dimension was measured from the midpoint of this line to the superior border of the right atrium parallel to the interatrial septum. The short-axis dimension was then measured from the lateral wall of the RA to the interatrial septum, perpendicular to the RA long-axis dimension (Figure 1D).[17] RA area was obtained from the apical four-chamber view by tracing the endocardial border at end-systole excluding the RA appendage (Figure 1D). TA diameter was also obtained from the apical four-chamber view at end-diastole starting at the lateral hinge point of the nonseptal TV leaflet and ending at the hinge point of the septal TV leaflet (Figure 1B). RV basal (maximal dimension in the lower third) and middle (maximal dimension in the middle third) cavity measurements were made in the RV-focused apical four-chamber view in end-diastole (Figure 1A).[18] Fractional area change, defined as (end-diastolic area − end-systolic area)/end-diastolic area × 100, was also measured using the RV-focused apical four-chamber view. End-diastole and end-systole were defined as the frames depicting the largest and smallest RV cavity sizes, respectively (Figure 1E). Peak TR gradient was measured using the modified Bernoulli equation, from the maximal TR jet velocity (Figure 1F). sPAP was estimated using the TR gradient and adding an approximation of RA pressure based on inferior vena cava size and collapsibility[18] (Figure 1G).

3D Transthoracic Echocardiography

Three-dimensional transthoracic echocardiographic studies were performed by using a Phillips iE33 ultrasound system (Philips Medical Systems) with a fully sampled matrix-array transducer (model X7-2t). The TV was imaged from the apical four-chamber view using full-volume and 3D zoom modes, as previously described.[15] Three-dimensional acquisitions were performed using electrocardiographic gating over four consecutive cardiac cycles with a single breath-hold.[19] Once acquired, the image was cropped and oriented to visualize the

TV leaflets in the en face view (RV or RA perspective), depending on which orientation best depicted the device lead. For display, the TV was oriented with the septum in the 6 o'clock position, in accordance with American Society of Echocardiography guidelines.[19]

Device Lead Location Relative to the TV Leaflets

Three-dimensional data sets were cropped (QLAB version 9.0; Phillips Medical Systems) and displayed to enable identification of the device lead position at the level of the tricuspid annulus. The device lead was described as interfering with leaflet motion (anterior, posterior, or septal), if it was noted to be impinging or adhering to a leaflet (Videos 1–3; available at www.onlinejase.com). If the device lead was in the commissure, the lead position was named according to the two leaflets between which it was located (anteroseptal, anteroposterior, or posteroseptal) (Videos 4 and 5; available at www.onlinejase.com). A lead was described as mobile or centrally located when it was neither in a commissural position nor interfering with leaflet function.[15] In addition, device pulse generator position was determined as "left" or "right" using the chest x-ray taken closest to the time of 3D transthoracic echocardiography.

Statistical Analysis

Continuous variables are summarized as mean ± SD, and categorical variables are presented as absolute numbers and percentages, unless otherwise stated. Pre- and postdevice measurements were compared using paired *t* tests. Differences between leads resulting in "leaflet-interfering" and "noninterfering" groups were tested using the unpaired *t* test. Independent factors associated with postdevice VC width increase were established using multivariate linear regression analysis, including individual factors with significant associations in bivariate analysis. The association between each independent factor and VC width is reported as β, representing the slope of the regression line and describing the expected change of VC width as a consequence of a unitary increase of the relevant predictor.

Postdevice TR VC width was dichotomized to separate moderate (VC width ≥ 0.5 cm) from less than moderate (VC width < 0.5 cm) TR and severe (VC width ≥ 0.7 cm) from less than severe (VC width < 0.7 cm) TR. Associations between predevice echocardiographic parameters and dichotomized TR severity were investigated using stepwise, backward and forward, multivariate logistic regression. Odds ratios (OR) were used to describe the associations of factors with moderate or severe TR, separately, using both the bivariate and multivariate logistic regression analyses.

Finally, the mitral regurgitation scale for VC severity was used to divide TR VC into mild (grade 1, 0–0.3 cm), mild to moderate (grade 2, 0.3–0.5 cm), moderate (grade 3, 0.5–0.7), and severe (grade 4, ≥0.7 cm) categories[16] to determine in which cases (interfering vs noninterfering leads) TR improved significantly (decreased by ≥1 grade) or worsened significantly (increased from grade 1 or 2 to grade 3 or 4 or from grade 3 to 4).

Statistical significance was defined as $P \leq .05$. Statistical analysis was performed using SPSS Statistics 20 (IBM, Armonk, NY).

RESULTS

Table 1 shows the summary of baseline characteristics. Of the 100 patients studied, 53% had ICDs, 20% had PPMs, and 27% had CRT devices. The majority of device generators (88%) were implanted in a left-sided chest pocket. The mean time between device implantation date and the postimplantation 2D or 3D echocardiographic study was 3.8 ± 3 years. The mean time between the pre-implantation echocardiographic examination and device lead placement was 0.9 ± 1.6 years. The majority of patients (79%) underwent preimplantation echocardiography <1 year before device lead implantation.

When comparing pre- and postimplantation echocardiograms, parameters that were significantly increased included RV end-diastolic and end-systolic areas and dimensions, RA area, and TR VC width. None of the other parameters, including left ventricular ejection fraction and TA diameter, significantly changed between pre- and postimplantation (Table 1).

In accordance with clinical practice, 2D images were reviewed before 3D images. In cases with little or no TR device lead present, noninterference was assumed. In cases with moderate or more TR, often with the TR jet directed along the wire, device lead interference with normal leaflet motion was suggested. An attempt was not made to specify with which of the three TV leaflets the lead was interfering, as this could not be diagnosed with certainty. Leaflet interference was easier to detect on dynamic 3D images of the TV when visualized from the RV perspective (Videos 1–4; available at www.onlinejase.com). Forty-five patients (45%) showed device lead interference with the TV leaflet. The septal leaflet was the most commonly affected (23 cases, 51% of the total interferences) followed by the posterior leaflet (19 cases, 42% of the interferences) and anterior leaflet (three cases) (Figure 2). Leaflet interference was most commonly due to device lead impingement (Figure 2) or adherence to the leaflet (Figure 3). Fifty-five patients did not have device lead–associated leaflet interference. In these patients, device leads were seen in either the posteroseptal commissure (26 cases) (Figure 4) or the middle of the valve (26 cases) (Figure 5). Videos 3 and 4 (available at www.onlinejase.com) illustrate a device lead in the posteroseptal (Video 3) and middle (Video 4) locations. Figure 6 details the number of device leads in interfering and noninterfering locations.

Differences between patients with and without device lead–associated leaflet interference are summarized in Table 2. There were no significant intergroup differences on predevice echocardiograms. On the postdevice echocardiograms, in the presence of an interfering lead, the TR VC width was larger, as were the TR gradient, sPAP, and RA areas. Of note, the time between implantation and echocardiographic recording was similar between the interfering and noninterfering groups (Table 2).

In the device lead interfering group (n = 45), 27 had worsening in TR severity from grade 1 or 2 to grade 3 or 4 or from grade 3 to grade 4. In the noninterfering group (n = 55), nine had TR worsening, eight from mild to moderate and only one from mild to severe. This suggests that device lead interference is associated with a greater likelihood of developing significant TR after placement. Of note, eight of 55 patients who were classified as having

noninterfering leads decreased TR severity by ≥1 grade, whereas six patients in the interfering group decreased TR severity by ≥1 grade after device lead placement.

The results of bivariate analysis for pre–device lead–placement factors and post–device lead–placement TR VC width are summarized in Table 3. Preimplantation VC width, RA size (linear dimensions and area), TA diameter, and lead-leaflet interference were all factors significantly associated with postdevice TR worsening ($P < .01$). Interestingly, the age of the device lead was not a significant predictor of postimplantation TR. Similarly, device type (ICD, PPM, or CRT device) was not significantly associated with the presence of TR after device lead implantation. In multivariate analysis, only preimplantation VC width ($\beta = 0.35$; 95% confidence interval [CI], 0.24–0.71) and the presence of an interfering lead ($\beta = 0.47$; 95% CI, 0.18–0.52) were independent factors significantly associated with postimplantation VC width. These same factors were positively associated with a higher likelihood of developing moderate (TR VC width ≥ 0.5 cm) (Figure 7) or severe (TR VC width ≥ 0.7) (Figure 8) TR. Using a multivariate stepwise logistic regression analysis, the presence of an interfering lead was the only factor associated with TR worsening, increasing the likelihood of developing moderate or severe TR by 15- and 11-fold, respectively.

When assessing patients with noninterfering leads, bivariate analysis showed that larger preimplantation VC width, RA long-axis dimension, RA area, RV end-diastolic area, and TA diameter as well as higher sPAP and RA pressure were all significantly associated with increased postimplantation TR VC width. The likelihood of developing at least moderate TR was associated with larger preimplantation TR VC width (OR, 9.1; 95% CI, 1.7–48.6), RA long-axis dimension (OR, 1.13; 95% CI, 1.03–1.23), RA area (OR, 1.14; 95% CI, 1.01–1.27), and RA pressure (OR, 1.14; 95% CI, 1.01–1.28).

DISCUSSION

In this study, we found that both a device lead interfering with the TV leaflets on 3D echocardiography and the presence of preexisting TR as measured by VC width are associated with increased risk for developing significant TR after device lead placement. An interfering lead was 11 times more likely to result in moderate TR (VC width ≥ 0.5 cm) and 9 times more likely to result in severe TR (VC width ≥ 0.7 cm) than a noninterfering lead.

Approximately 80% of significant TR is functional in nature, resulting from RV or TA dilation and leaflet tethering in the setting of RV remodeling due to pressure and/or volume overload.[2,13,20] In this study, we measured RV size (RV basal end-diastolic dimension, RV middle end-diastolic dimension, RV end-diastolic area, and RV end-systolic area) and TA size (TA diameter) to assess the effects of changes in RV dimension and TA dilatation on TR VC width after device lead placement. To measure the effects of RV pressure overload on TR worsening after device lead placement, we assessed RV systolic pressure. With the addition of RA dimensions and area, we were able to account for most of the factors causing and maintaining functional TR.[21,22]

Before device implantation, both interfering and noninterfering device lead groups were similar in terms of RA and RV size, RV function, TA diameter, and sPAP. After device

placement, the group with interfering leads had significantly larger RA areas and higher sPAP, in keeping with the presence of more severe TR. Before device implantation, factors predicting postdevice TR worsening included RA size, RV size, TA diameter, and the presence of an interfering lead. Increases in both RA and RV size can occur secondary to TR, and both have been shown to be associated with TA dilatation[14,23] and thereby to cause TR. Therefore, from this analysis, we conclude that four factors are significantly associated with TR development after device placement: (1) RA dimensions, (2) RV dimensions, (3) TA dilatation, and (4) the presence of an interfering lead. These results are not surprising, because three of these factors (RA, RV, and TA sizes) are known to be associated with the development and worsening of functional TR.

Interestingly, predevice TRVC width showed ORs of 8.5 (Figure 7) and 5.9 (Figure 8) for the development of at least moderate and severe TR after device placement, respectively. This implies that implantation of a device lead in a patient with preexisting TR increases the likelihood of developing moderate or severe TR by 54% and 43%, respectively, for every 2 mm of VC width. In addition, an interfering lead would dramatically increase the likelihood of developing significant TR by a factor of 10 (Figure 7).

These findings suggest that it would be beneficial for patient outcomes if the device lead were placed under 3D echocardiographic guidance into a commissural or middle valve position, which is not associated with TR development. Indeed, when patients with non-interfering leads are analyzed as a subgroup, bivariate analysis revealed that preimplantation VC width, RV size, TA diameter, sPAP, and RA pressure were all associated with TR development after device lead placement. All these factors are well-established causes or perpetuators of functional TR, further reinforcing the observed association between an interfering device lead and TR worsening.

The number of interfering device leads in this study was relatively large (45 of 100 [45%]), likely because the population was retrospectively enrolled at a tertiary care center and therefore highly selected. Additionally, because patients at our institution are not routinely scheduled for either pre- or postimplantation echocardiography, those who underwent echocardiography were more likely to have developed symptoms after implantation or to have an associated valvular or myocardial dysfunction. For example, given that 36 patients had significant TR worsening, and 27 of these had interfering device leads, it is likely that in this group, transthoracic echocardiography was ordered for symptoms related to worsening TR in the presence of an interfering device lead. Of note, in a study published by Lin *et al.*, 64 of 156 patients (41%) who had surgical lead extraction or repositioning for severe TR had evidence of device lead interference with the TV leaflets. These data, although collected over a 10-year time span, represent the extreme situation in which device lead–associated TV interference led to severe valvular insufficiency requiring surgical intervention. This study underscores the fact that this clinical problem is not insignificant.[6]

Significant Changes in TR

In the present study, the association between device lead interference and the subsequent development of severe TR was robust. Clinically, it would be concerning if the degree of TR after device lead implantation increased from mild to moderate or severe, as this is

associated with increased morbidity and mortality.[9] The latter would also influence the clinical strategy used, as the patient would need either close follow-up or, in some cases, surgical intervention. It was difficult to assign a VC width value that would parallel a clinically significant degree of TR worsening. We graded TR severity using the mitral regurgitation grade scale, because TR lacks a severity scale in the current guidelines.[16] We noted that 36 of 100 patients (36%) worsened in TR severity (i.e., worsened from mild to moderate or severe or from moderate to severe). Of these, 27 (75%) had device leads interfering with TV leaflet motion, whereas the other 25% did not. Of note, eight of 55 patients (15%) without interfering leads improved their TR degrees compared with predevice placement echocardiograms. Surprisingly, six of 45 patients (13%) who had interfering leads improved their TR degrees. These six patients were reexamined. In two patients, the lead was adhering to the leaflet edge without restricting leaflet motion. In two patients, the TR grade decreased from severe to moderate without a ready explanation. In the remaining two patients, CRT devices were implanted, which could have alleviated TR in response to RV reverse remodeling.[24]

Value of 3D Imaging

Three-dimensional echocardiography continues to be an underused tool. Our results demonstrate that the position of device leads as they traverse the TV is an important cause of TR. Thus far, device lead–associated interference with the TV was a diagnosis made at autopsy or surgery.[6,25,26] With 3D echocardiography, it is possible to make this diagnosis noninvasively (Figures 9 and 10; Videos 5A and 5B; available at www.onlinejase.com). Admittedly, the clinical consequences of device lead–associated TR are unclear.[3] It is well appreciated, however, that TR is not a benign condition, because it is associated with reduced survival rates at 1 year.[9,27] Moreover, 2D transthoracic imaging is insufficient to fully visualize RV leads,[5,6,8] implying that 3D echocardiography is of incremental value in the assessment of TR after device lead placement.[15] Once device lead–associated TR is identified, lead repositioning may be the only way to correct the problem, particularly if recognized early, before inflammation and fibrosis cause lead adherence and fusion to the TV leaflets.[3,25,28] Our results could also stimulate more widespread consideration of subcutaneous ICD devices, which use epicardial and not transvenous leads, especially in patients who only need defibrillators and do not require pacemakers.[29,30]

This is one of the first studies evaluating pre- and postdevice echocardiograms, including postdevice 3D imaging of device lead location, allowing the categorization of lead position as leaflet-interfering versus noninterfering. Also, we assessed independent factors associated with TR worsening after device lead implantation. Several other studies have also assessed TR before and after device implantation using echocardiography.[4,5,8,11,31] However, only one of those studies[8] evaluated the TV after lead implantation using 3D imaging. Previous studies using 2D imaging were unable to define mechanisms associated with increased TR post device lead placement and were therefore unable to separate TR secondary to lead interference from other causes. The only study done with 3D imaging[8] did not systematically analyze and compare the impact of the different parameters associated with worsening TR. The strength of this study is that it was designed to determine, in a group of patients with normal TVs who developed TR after device lead placement, whether TR was

directly attributable to RA, RV, or TA dilatation, increased RV systolic pressure, or device lead interference with normal TV leaflet motion. By systematically comparing the contributions of these factors in a multivariate model, we found that the independent factors associated with TR after device lead implantation were predevice TR and the presence of an interfering lead. These results add to those of our previous study,[15] which demonstrated that 3D imaging of device leads was feasible, illustrated common lead locations at the level of the TV leaflets, and showed an association between increased TR and interfering device leads.

Limitations

Because of the retrospective nature of this study, the intervals between echocardiographic examinations before device lead implantation and after device lead follow-up imaging were variable. Nevertheless, 79% of the predevice echocardiograms were acquired <1 year before device placement, thereby minimizing the effects of other factors, such as RV and TA size on TR worsening. Another potential limitation is the fact that TR VC width and not a composite of multiple indices was used to determine TR severity. However, TR VC width has been shown to be a more reliable method of TR assessment than jet size, correlating well with effective regurgitant orifice.[32] Furthermore, although it has been shown that VC width is load dependent, on bivariate analysis, VC width was independent of TR gradient, sPAP, and RA pressure and therefore not a factor significantly associated with postimplantation VC width. Of note, we were not able to exclude the possibility, although unlikely, that the TR jet itself, as it becomes more significant, may cause the device lead to impinge on a leaflet. Finally, a word of caution about the implications of our results for the general population of patients who have pacemakers inserted: this study included a highly selected population, and therefore its results may not be an accurate representation of the true prevalence of significant TR after device implantation. To study the latter, a prospective study would be required, which should include consecutive patients referred for device implantation.

CONCLUSIONS

When a device lead is introduced into the right ventricle, its position relative to the TV leaflets is important and can in most patients be easily determined using 3D echocardiography. Compared with other factors known to be associated with TR worsening, the presence of a device lead interfering with normal TV leaflet motion is the most significant factor associated with the development of postdevice TR. These findings suggest that 3D echocardiography of the TV should be used to determine whether device lead interference is present in patients with PPMs, ICDs, or CRT devices who present with significant TR and/or right heart failure.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations

CI	Confidence interval
CRT	Cardiac resynchronization therapy
ICD	Implantable cardioverter-defibrillator
OR	Odds ratio
PPM	Permanent pacemaker
RA	Right atrial
RV	Right ventricular
sPAP	Systolic pulmonary artery pressure
TA	Tricuspid annular
3D	Three-dimensional
TR	Tricuspid regurgitation
TV	Tricuspid valve
2D	Two-dimensional
VC	Vena contracta

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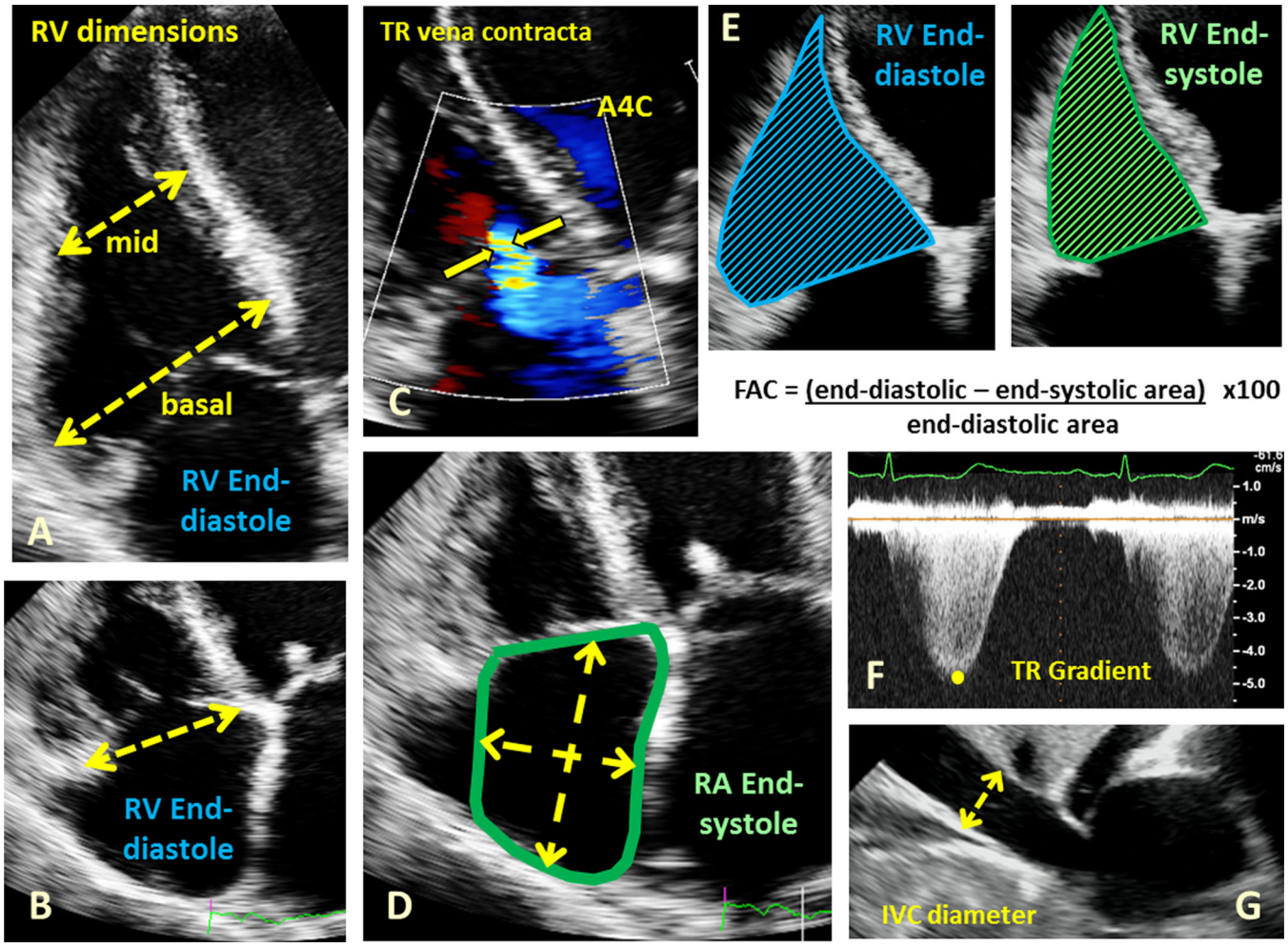

Figure 1.

Right heart measurements. **(A)** RV basal and middle dimensions as measured from the RV-focused apical four-chamber (A4C) view in end-diastole. **(B)** TA diameter was obtained at end-diastole from the lateral hinge point of the nonseptal TV leaflet to the hinge point of the septal leaflet. **(C)** TR was quantified using VC width. **(D)** RA dimensions and area were measured at RA end-systole. A straight line connected both sides of the TV leaflet hinge points. The RA long-axis and short-axis dimensions were perpendicular to each other. RA area was obtained by tracing the endocardial border excluding the RA appendage. **(E)** Fractional area change (FAC) was measured using the RV-focused A4C view as defined. End-diastolic (*blue hatch marks*) and end-systolic (*green hatch marks*) areas are shown. **(F)** Peak TR gradient was measured using the modified Bernoulli equation ($4 \times v^2$), from the maximal TR jet velocity (v) in centimeters per second shown. **(G)** Inferior vena cava (IVC) size was estimated from the subcostal view just proximal to the hepatic vein. Collapsibility was assessed over the cardiac cycle. Measurements were used to estimate sPAP.

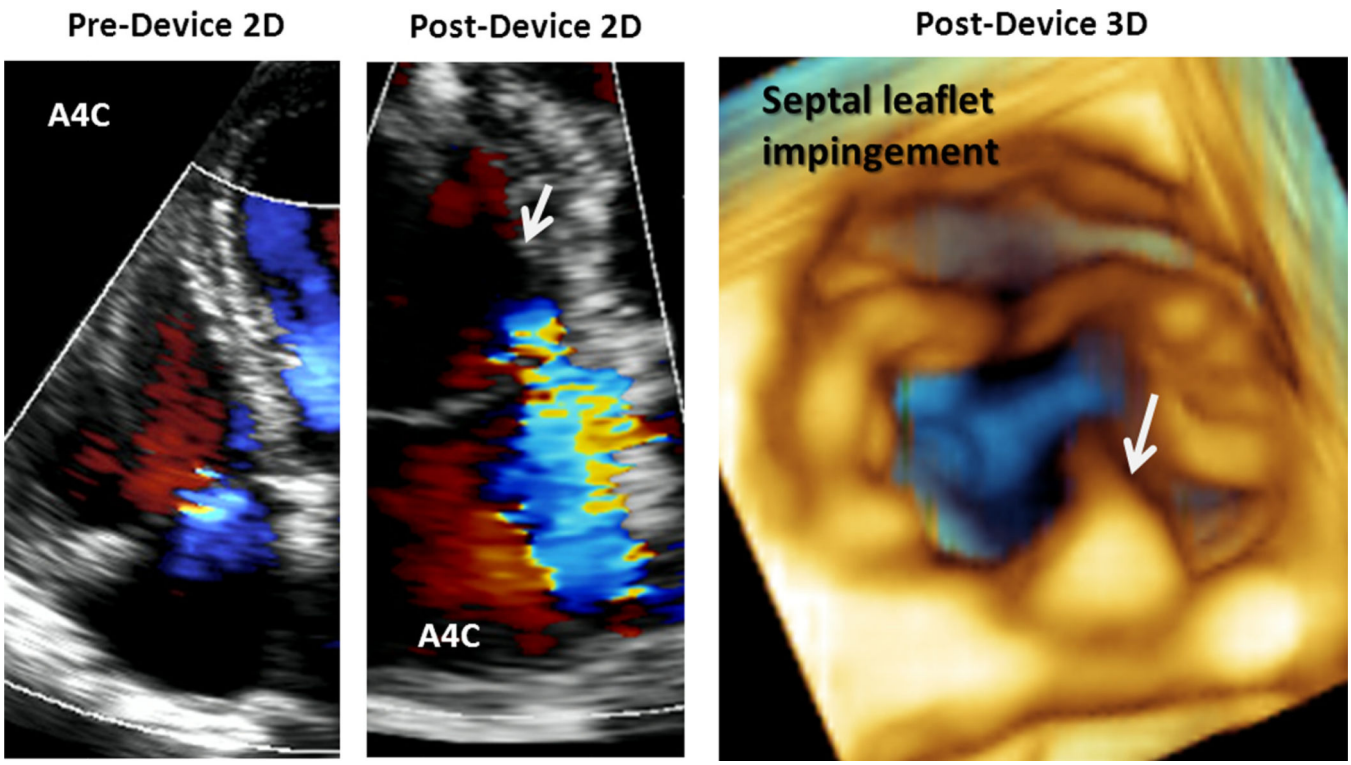

Figure 2.

Septal leaflet impingement. Color flow recordings of the TV showing mild TR before device lead placement (*left*) and moderate TR after device lead implantation (*middle*). Three-dimensional zoom (*right*) confirms septal leaflet impingement. *White arrow* points to device lead. *A4C*, Apical four-chamber. See also Video 1.

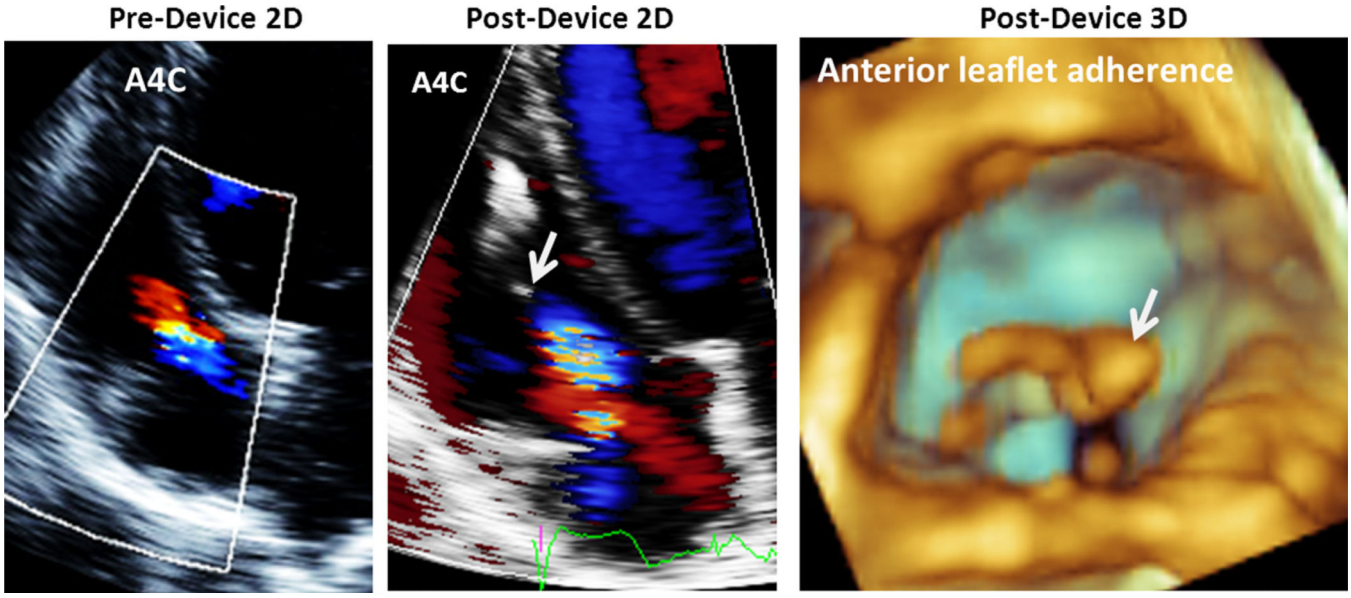

Figure 3.

Anterior leaflet adherence. Color flow recordings of the TV showing trace TR before device lead placement (*left*) and mild to moderate TR after device lead implantation (*middle*). Three-dimensional zoom (*right*) confirms anterior leaflet adherence of the device lead. *White arrow* points to device lead. *A4C*, Apical four-chamber. See also Video 2.

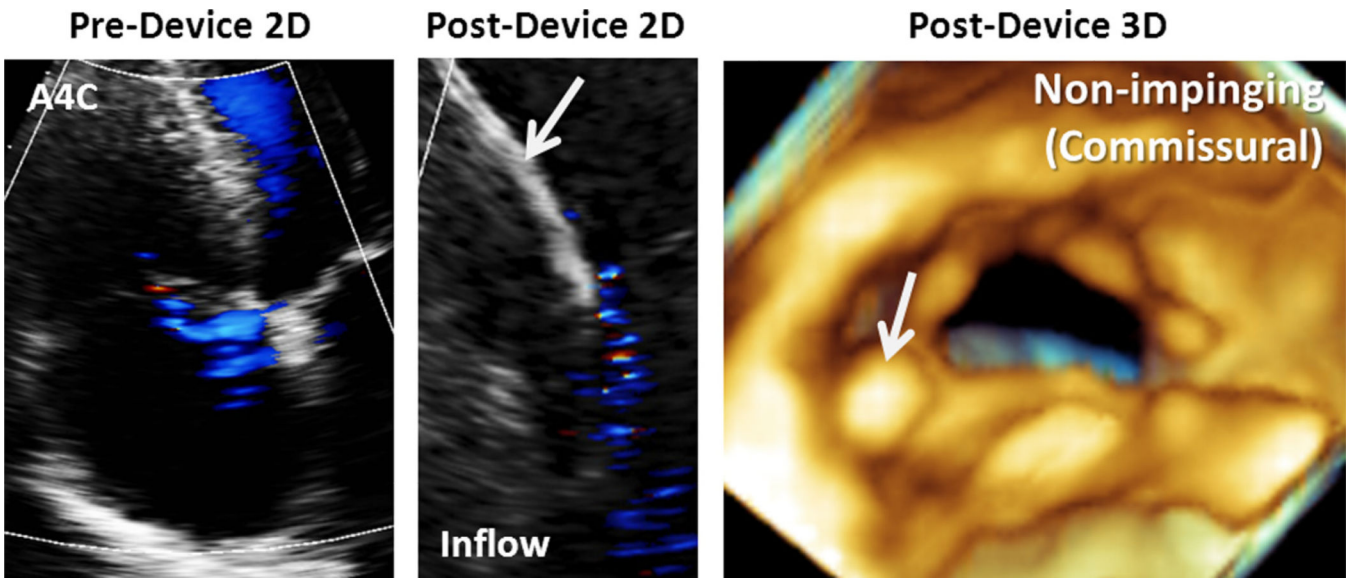

Figure 4.

Noninterfering lead. Color flow recordings of the TV showing trace TR before device lead placement (*left*) and trace TR after device lead implantation (*middle*). Three-dimensional zoom (*right*) confirms device lead in the posteroseptal position. *White arrow* points to device lead. *A4C*, Apical four-chamber; *inflow*, RV inflow view. See also Video 3.

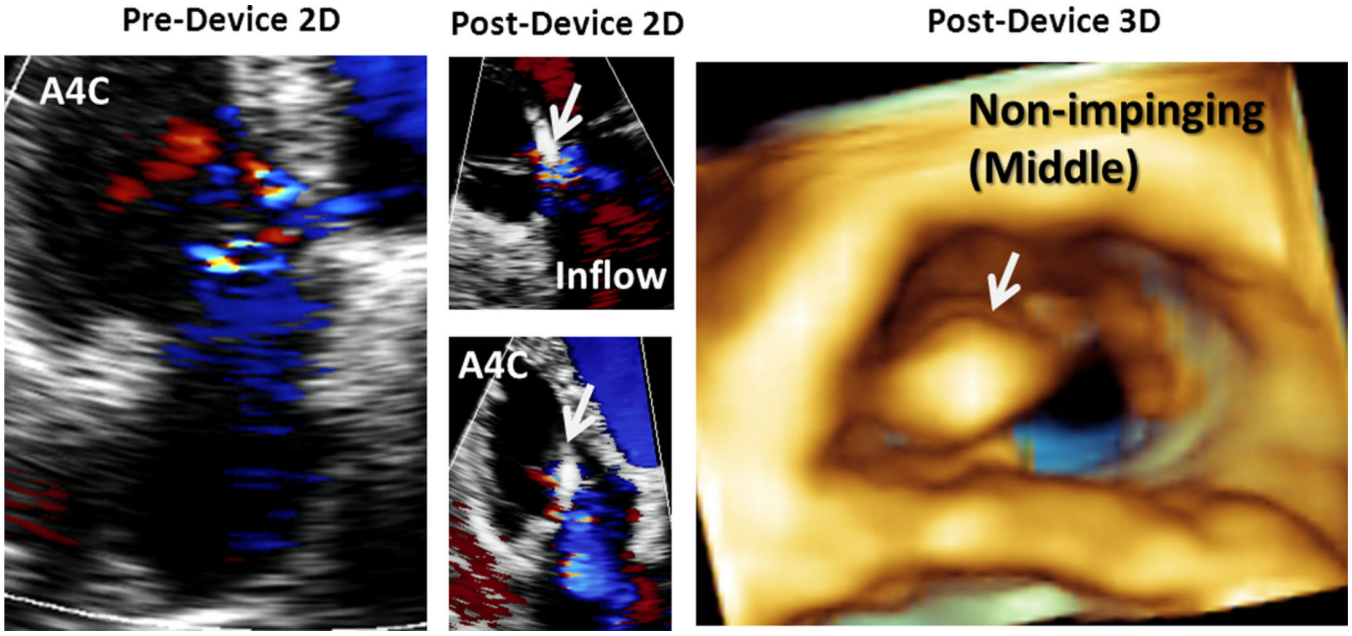

Figure 5.

Noninterfering lead. Color flow recordings of the TV with trace TR before device lead placement (*left*) and mild TR after device lead implantation (*middle*). Three-dimensional zoom (*right*) confirms device lead in the central position. *White arrow* points to device lead. *A4C*, Apical four-chamber; *inflow*, RV inflow view. See also Video 4.

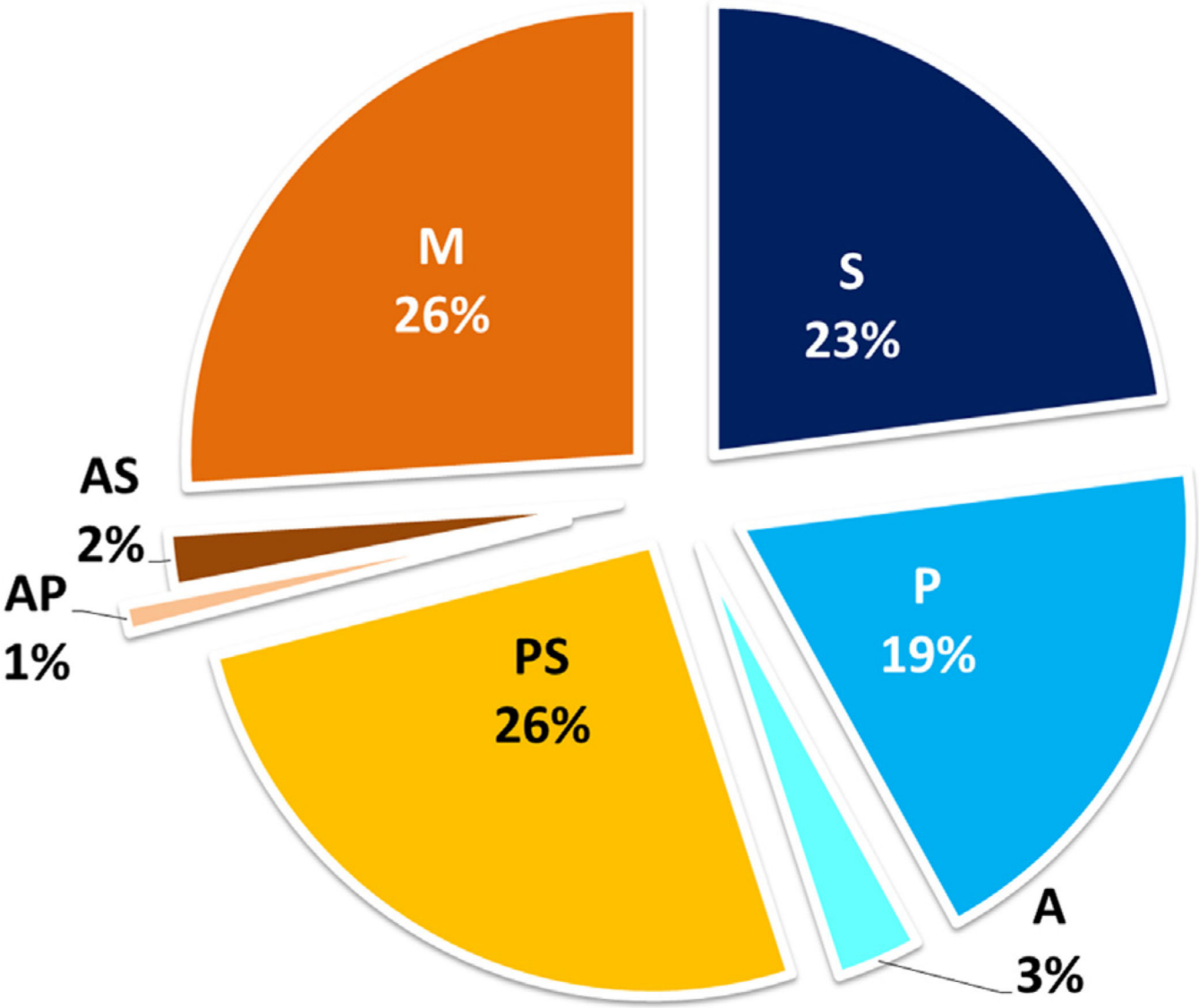

Figure 6.

Chart illustrating spectrum of device lead locations for patients enrolled in the study. Interfering leads are in *blue* (in the anterior [A], posterior [P], and septal [S]) positions on the *righthand side* of the pie chart. Noninterfering leads are in *green* in the center of the valve (M), posteroseptal (PS), anteroposterior (AP), and anteroseptal (AS) positions on the *left-hand side* of the pie chart.

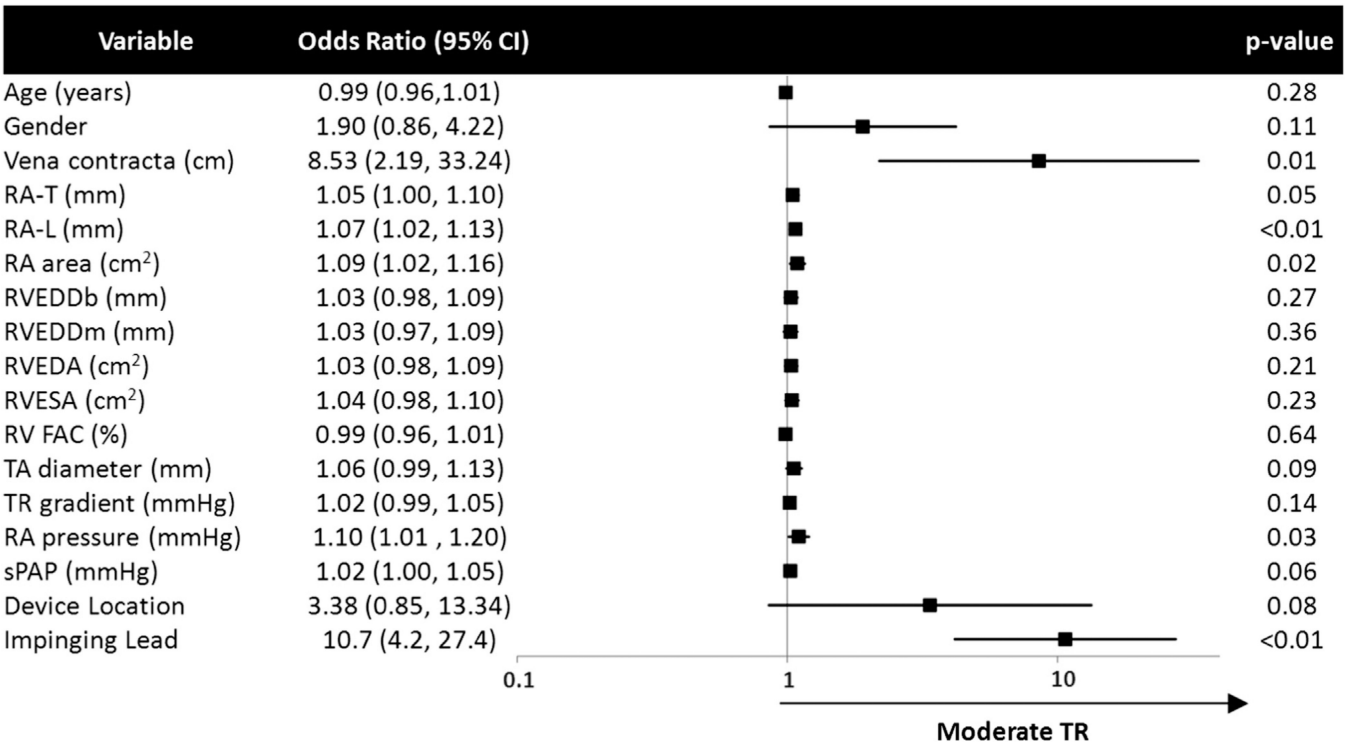

Figure 7.

Factors associated with postimplantation moderate TR (VC width ≥ 0.5 cm). *FAC*, Fractional area change; *RA-L*, RA longitudinal dimension; *RA-T*, RA transverse dimension; *RVEDA*, RV end-diastolic area; *RVEDDb*, RV enddiastolic diameter at the base; *RVEDDm*, RV end-diastolic diameter at the middle level; *RVESA*, RV end-systolic area.

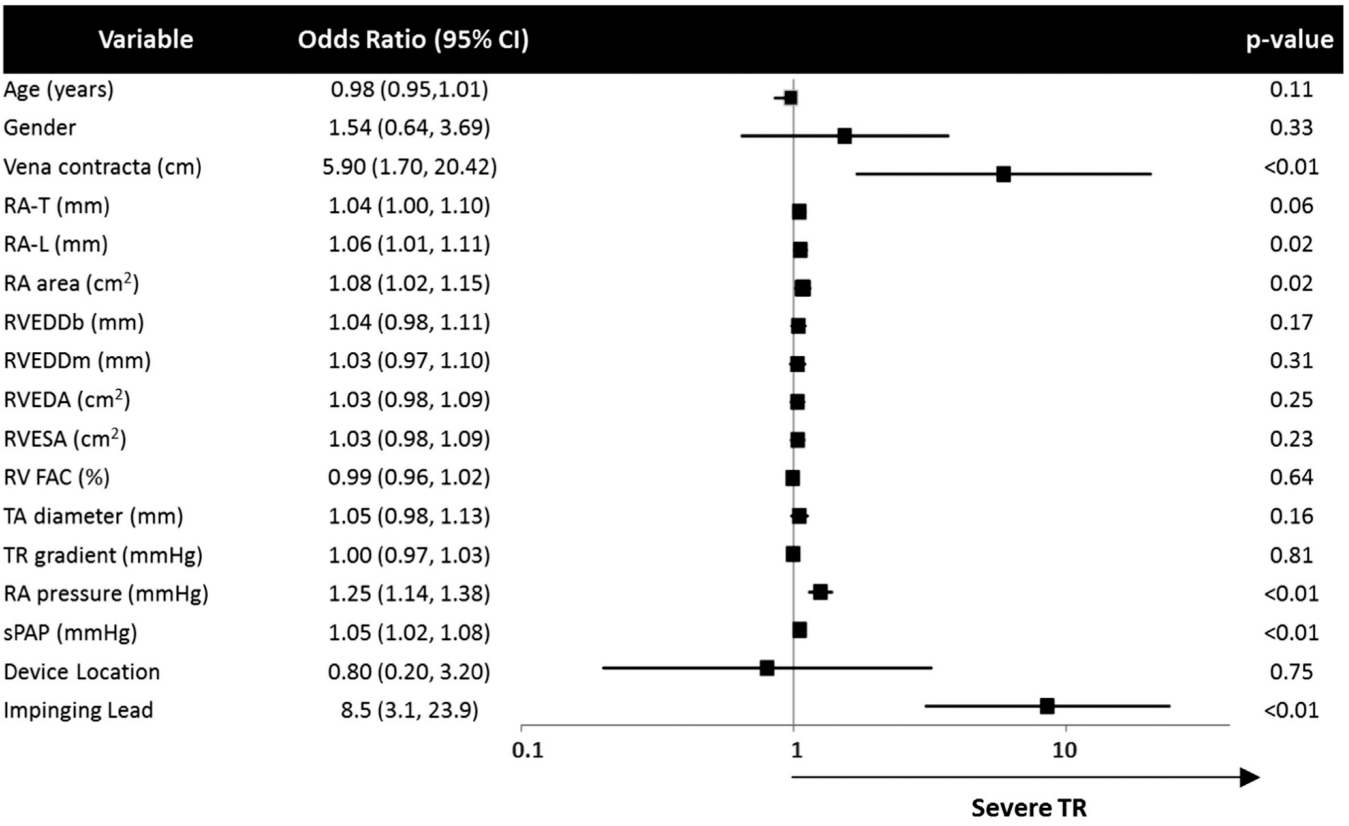

Figure 8.

Factors associated with postimplantation severe TR (VC width ≥ 0.7 cm). *FAC*, Fractional area change; *RA-L*, RA longitudinal dimension; *RA-T*, RA transverse dimension; *RVEDA*, RV end-diastolic area; *RVEDDb*, RV end-diastolic diameter at the base; *RVEDDm*, RV end-diastolic diameter at the middle level; *RVESA*, RV end-systolic area.

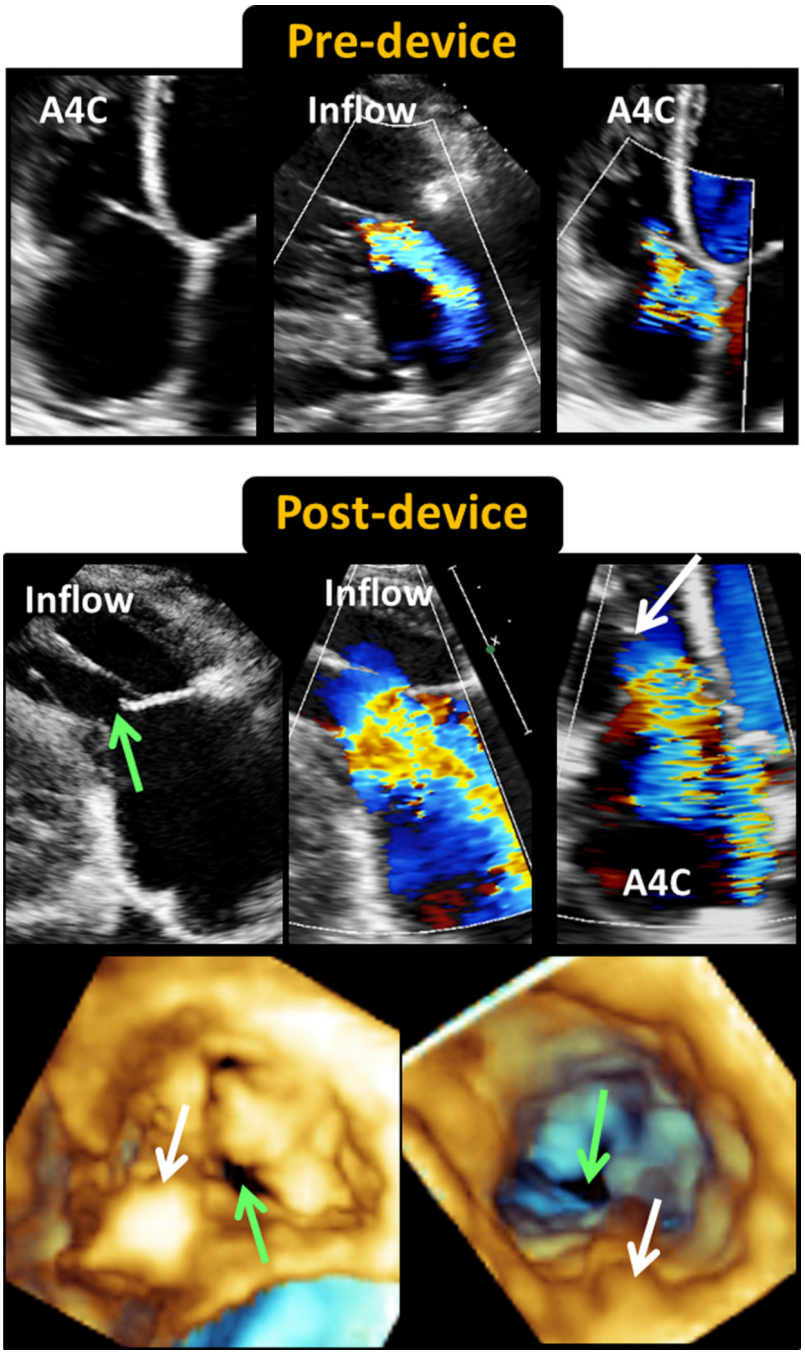

Figure 9.

Case illustrating predevice 2D and color Doppler recordings of the TV (*top row*) in a patient with moderate TR. One day after device lead implantation (*middle and bottom rows*), the patient presented with severe malcoaptation of the TV leaflets (*green arrow*) and massive TR. The device lead was impinging on the septal leaflet (*white arrows* on 3D image) and possibly entangled in the chordal apparatus. Malcoaptation can be seen on 3D images from the RV and RA perspectives (*green arrows*). *A4C*, Apical four-chamber; *inflow*, right ventricular inflow view.

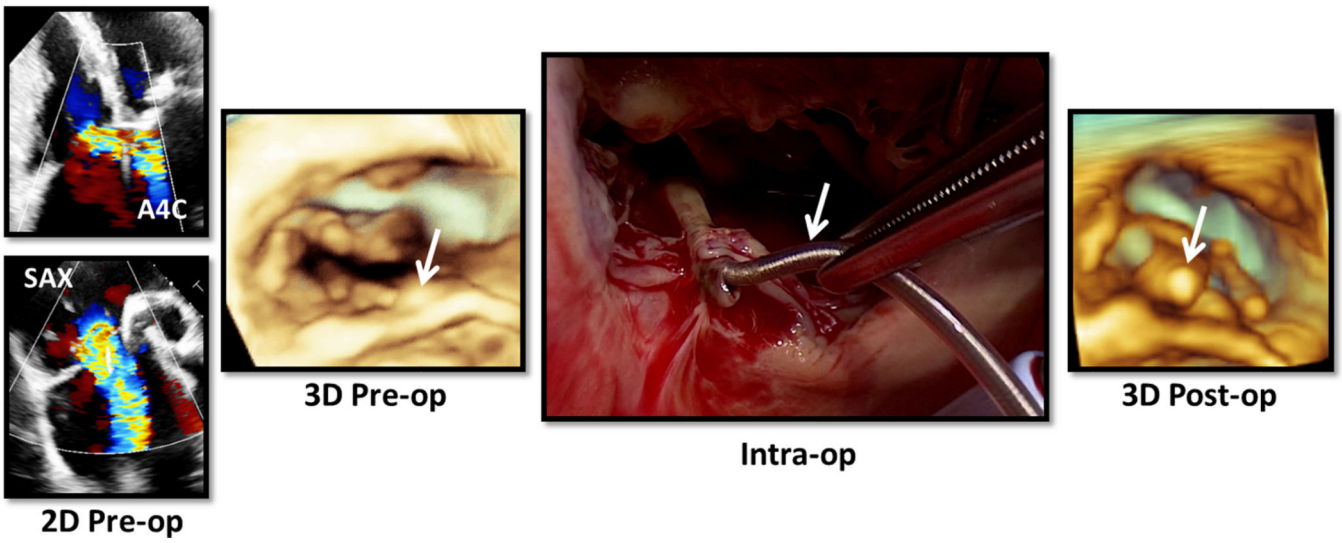

Figure 10.

Case of septal leaflet impingement on 3D echocardiography with intraoperative confirmation. Preoperative 2D color Doppler recordings showing at least moderate TR with accompanying 3D zoom illustrating septal leaflet impingement are shown on the far left. Surgical confirmation of septal impingement is seen in the middle panel. During surgery, the wire was placed in the posteroseptal commissure, and the commissure was sutured closed. Postoperative 3D zoom is seen at far right. *A4C*, Apical four-chamber; *SAX*, basal short-axis view. See also Videos 5A and 5B.

Table 1

Baseline patient characteristics before and after implantable device lead placement

Characteristic	*n**	Before device placement (*n* = 100)	*n**	After device placement (*n* = 100)	*P*
Demographics					
Age (y)	100	—	—	67 ± 16 (26–93)	—
Men	100	—	—	49%	—
Device lead age† (years)	100	—	—	3.8 ± 3	—
Device information					
Pulse generator position on left	—	—	97	84 (85%)	—
ICD	—	—	100	53 (53%)	—
PM	—	—		20 (20%)	—
CRT defibrillator	—	—		27 (27%)	—
Echocardiographic parameters					
LVEF (%)	98	35 ± 16	97	36 ± 16	.79
RA-T (mm)	96	44 ± 9	100	46 ± 10	.32
RA-L (mm)	95	53 ± 9	100	53 ± 11	.78
RA area (cm^2)	95	22 ± 7	100	23 ± 9	.02
TA diameter (mm)	98	37 ± 6	99	37 ± 7	.76
RVEDDb (mm)	97	46 ± 7	100	50 ± 9	<.01
RVEDDm (mm)	96	33 ± 7	99	39 ± 8	<.01
RVEDA (cm^2)	98	27 ± 8	100	30 ± 9	<.01
RVESA (cm^2)	98	17 ± 8	100	20 ± 9	<.01
RV FAC (%)	98	38 ± 14	100	36 ± 13	.13
TR gradient (mm Hg)	86	39 ± 16	93	40 ± 16	.51
RA pressure (mm Hg)	96	7 ± 5	94	7 ± 5	.90
sPAP (mm Hg)	86	46 ± 18	93	46 ± 18	.79
TR VC width (cm)	100	0.31 ± 0.36	100	0.57 ± 0.49	<.01
TR severity					
Mild (0–0.3 cm)		60		30	

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Characteristic	*n**	Before device placement (*n* = 100)	*n**	After device placement (*n* = 100)	*P*
Mild to moderate (0.3–0.5 cm)		15		19	
Moderate (0.5–0.7 cm)		8		22	
Severe (≥0.7 cm)		17		29	

FAC, Fractional area change; *RA-L*, RA longitudinal dimension; *RA-T*, RA transverse dimension; *RVEDA*, RV end-diastolic area; *RVEDDb*, RV end-diastolic diameter at the base; *RVEDDm*, RV end-diastolic diameter at the middle level; *RVESA*, RV end-systolic area.

Data are expressed as mean ± SD (range) or as number (percentage).

*Number of patients for whom data were available.

†Obtained from determining the time between 3D echocardiography and device implantation date.

Table 2

Differences in measured echocardiographic parameters in patients with interfering versus noninterfering device leads

	Before device implantation			After device implantation		
Parameter	**Noninterfering leads (*n* = 55)**	**Interfering leads (*n* = 45)**	***P*** (predevice)*	**Noninterfering leads (*n* = 55)**	**Interfering leads (*n* = 45)**	***P**† (postdevice)*
Age (y)	—	—	—	66 ± 14	67 ± 19	.94
Men	—	—	—	60%	36%	
Device age (y)	—	—	—	3.3 ± 2.7	4.5 ± 3.4	.07
LVEF (%)	35 ± 16	35 ± 17	.97	36 ± 16	36 ± 16	.92
RA-T (mm)	43 ± 8	45 ± 10	.24	44 ± 8	47 ± 13	.30
RA-L (mm)	52 ± 9	54 ± 10	.50	52 ± 10	55 ± 13	.16
RA area (cm^2)	21 ± 6	23 ± 9	.29	22 ± 7	25 ± 10	.03
TA diameter (mm)	37 ± 6	37 ± 7	.72	37 ± 8	38 ± 6	.46
RVEDDb (mm)	45 ± 7	46 ± 8	.79	49 ± 10	51 ± 8	.33
RVEDDm (mm)	33 ± 7	33 ± 8	.90	38 ± 8	40 ± 8	.30
RVEDA (cm^2)	27 ± 8	26 ± 8	.40	28 ± 9	31 ± 9	.18
RVESA (cm^2)	17 ± 7	17 ± 8	.97	19 ± 9	21 ± 9	.32
RV FAC (%)	39 ± 14	37 ± 14	.41	36 ± 13	35 ± 14	.80
TR gradient (mm Hg)	36 ± 14	42 ± 18	.13	36 ± 15	45 ± 16	<.01
RA pressure (mm Hg)	6 ± 5	7 ± 5	.21	7 ± 5	6.8 ± 5.0	.99
sPAP (mm Hg)	43 ± 15	50 ± 21	.08	42 ± 18	51 ± 18	.01
TR VC width (cm)	0.26 ± 0.38	0.35 ± 0.32	.25	0.38 ± 0.45	0.79 ± 0.44	<.01
TR severity						

Parameter	Before device implantation			After device implantation		
	Noninterfering leads (n = 55)	Interfering leads (n = 45)	P^* (predevice)	Noninterfering leads (n = 55)	Interfering leads (n = 45)	$P^\dagger$ (postdevice)
Mild (0–0.3 cm)	38	22		26	4	
Mild to moderate (0.3–0.5 cm)	7	8		14	5	
Moderate (0.5–0.7 cm)	1	7		9	13	
Severe (≥0.7 cm)	9	8		6	23	

FAC, Fractional area change; *RA-L*, RA longitudinal dimension; *RA-T*, RA transverse dimension; *RVEDA*, RV end-diastolic area; *RVEDDb*, RV end-diastolic diameter at the base; *RVEDDm*, RV end-diastolic diameter at the middle level; *RVESA*, RV end-systolic area.

Data are expressed as mean ± SD or as number or percentage.

*P value between preimplantation parameters.

†P value between postimplantation parameters.

Table 3

Results of bivariate analysis for factors associated with postimplantation VC width

Independent variable	Correlation coefficient*	β†	95% CI for β	*P*
Age	0.11	−0.003	−0.009 to 0.003	.28
Gender	−0.18	—	—	.21
Pulse generator location‡	0.10	—	—	.62
Device type§	−0.07	—	—	.29
Device age	0.06	−0.009	−0.041 to 0.023	.57
VC width	0.36	0.500	0.242 to 0.757	<.01
RA-T	0.29	0.015	0.005 to 0.026	<.01
RA-L	0.31	0.016	0.006 to 0.026	<.01
RA area	0.36	0.023	0.011 to 0.036	<.01
TA diameter	0.28	0.021	0.006 to 0.037	<.01
RVEDDb	0.20	0.012	0.000 to 0.025	.05
RVEDDm	0.15	0.010	−0.004 to 0.024	.15
RVEDA	0.20	0.013	0.000 to 0.025	.05
RVESA	0.17	0.011	−0.002 to 0.024	.09
RV FAC	0.06	−0.002	−0.009 to 0.005	.58
TR gradient (mm Hg)	0.09	0.003	−0.004 to 0.010	.39
RA pressure (mm Hg)	0.19	0.019	−0.001 to 0.038	.06
sPAP (mm Hg)	0.14	0.004	−0.002 to 0.010	.21
Presence of interfering lead	0.53	—	—	<.01

FAC, Fractional area change; *RA-L*, RA longitudinal dimension; *RA-T*, RA transverse dimension; *RVEDA*, RV end-diastolic area; *RVEDDb*, RV end-diastolic diameter at the base; *RVEDDm*, RV end-diastolic diameter at the middle level; *RVESA*, RV end-systolic area.

*Correlation coefficient refers to the Pearson correlation for continuous variables and Spearman correlation for categorical variables.

†The regression coefficient (β) is the average change in the dependent variable (postimplantation VC width) for a 1-unit change in the independent variable (RA-T, RA-L, etc). It is the slope of the regression line.

‡Location of pulse generator can be in a left-sided or right-sided pocket.

§Device type refers to ICD, PPM, or CRT device.