Chapter 10
Evaluation and Management of Tricuspid Regurgitation in Patients with Cardiac Implantable Electronic Devices

Donya Mohebali and James D. Chang

Introduction

Approximately 200,000 permanent pacemakers (PPMs) and 120,000 implantable cardioverter defibrillators (ICDs) are implanted annually in the United States [1]. Cardiac implantable electronic devices (CIEDs) have increased the quality and duration of life for millions of patients by providing support of heart rate, atrioventricular and interventricular synchrony, and prevention of sudden cardiac death [1, 2]. Until recently, with the advent of leadless pacing systems and His bundle pacing, the near-universal requirement for an endocardial lead to provide pacing or defibrillation, or both, in the right side of the heart has led to the recognition of adverse consequences of these leads with respect to tricuspid valve (TV) structure and function.

Tricuspid regurgitation (TR), of even moderate grade and of any etiology (primary or secondary), is associated with increased mortality rates, even after accounting for factors known to contribute to secondary, or functional, TR such as left ventricular (LV) dysfunction, right ventricular (RV) dilation and dysfunction, and pulmonary hypertension [3]. In patients with CIEDs, moderate-or-severe TR occurs at significantly higher rates [4, 5], and has been shown to be associated with increased heart failure hospitalizations and mortality [6–9].

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D. Mohebali
Division of Cardiology, Beth Israel Deaconess Medical Center, Boston, MA, USA

J. D. Chang (✉)
Cardiovascular Division, Beth Israel Deaconess Medical Center, Boston, MA, USA
e-mail: jchang@bidmc.harvard.edu

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Tricuspid valve dysfunction after CIED implantation can manifest clinically as right-sided heart failure secondary to TR (or less often to tricuspid stenosis) or as left-sided heart failure when RV volume overload impairs LV filling by direct ventricular interaction through the interventricular septum. Other structural consequences can include mechanical interference with normal leaflet coaptation, leaflet entrapment, subvalvular support structure entanglement, endocarditis, and damage during lead placement or manipulation, or at the time of lead extraction of infected or malfunctioning leads.

The diagnosis and differentiation of lead-related primary TR, as distinct from secondary/functional TR, poses unique challenges, but constitute a critical distinction in the management of patients with a CIED and right heart failure. Routine diagnostic imaging can be fraught with pitfalls and therefore a high level of clinical suspicion in conjunction with 3D echocardiography can alert the clinician to the possibility of worsening heart failure as a consequence of mechanical interference with TV leaflet mobility or coaptation. This form of TV dysfunction may be amenable to lead extraction or valve repair or replacement when performed in a timely fashion to avoid severe annular and chamber dilation as well as severe RV dysfunction that when present may preclude the desired outcome even if the TV is technically repairable or replaceable. Thus, corrective intervention for suspected lead-related TR should be undertaken ONLY when all five of the following conditions are met: (1) the RV (tricuspid annulus) is neither severely dilated nor severely dysfunctional, (2) there is robust echocardiographic and hemodynamic evidence supporting a primary lead-related etiology of TR, (3) the requirement for stroke work production is not expected to exceed the capacity of the RV if and when it is forced to eject its entire stroke volume in an antegrade direction, (4) any left-sided cardiac dysfunction that may be contributing to TR is optimally managed, and (5) a TV replacement or repair strategy is available if transvenous lead extraction is the contemplated intervention.

Mechanisms of Lead-Induced Tricuspid Regurgitation

In order to understand the mechanism of lead-induced TR, it is important to understand the basic morphology and structure of the TV. Tricuspid valve morphology and attachments make it prone to insufficiency, as does any preexisting chamber dilation or LV dysfunction. The TV apparatus is comprised of a nonplanar elliptical annulus, three leaflets (anterior, posterior, and septal), chordae tendineae, and two papillary muscles (anterior and posterior). The mural portion of the annulus subtends the RV free wall, is not supported by the semirigid fibrous cardiac skeleton, and therefore can elongate under chronic pressure or volume overload leading to annular dilation. This is in contrast to the septal portion of the annulus that subtends the right fibrous trigone and is supported by the cardiac skeleton. Some of the TV chordae tendineae attach directly to the interventricular septum and free wall without an intervening papillary muscle. As a result of this underlying structure, TR

tends to beget more TR as the effects of a chronically volume overloaded state leads to chamber and annular dilation, tethering of the leaflets, and loss of leaflet coaptation. Pre-existing left-sided cardiac dysfunction (including systolic or diastolic myocardial dysfunction, valvular dysfunction, and dyssynchrony) predisposes the patient to functional or secondary TR, and as a result even a modest increment in TR associated with CIED implantation can over time result in severe TR and right-sided heart failure due to the combined effect of primary lead-related and secondary factors.

Mechanisms of CIED-induced TR can be classified as implantation-related, device-mediated, and pacing-related. Damage to the TV leaflets and subvalvular structures can occur during lead implantation, removal, or manipulation. These include leaflet perforation, laceration, or avulsion (primarily occurring during lead extraction) (Video 10.1), and transection of papillary muscles or chordae tendinae [10–14].

Device-mediated TR results from mechanical interference with TV leaflet mobility and coaptation. This occurs by the presence of a lead traversing the TV, which can prevent leaflet coaptation by direct contact with the leaflets, impingement on leaflet mobility, or by entanglement with the chordae tendinae. Absent direct mechanical interference with leaflet coaptation, over the long term even intermittent contact between the endocardial leads and leaflet or chordal structure can result in a foreign body inflammatory and fibrotic response leading to encapsulation or entrapment of the lead with subsequent loss of leaflet mobility (Videos 10.2 and 10.3).

Additionally, the presence of hardware in the circulatory system in combination with damage to the TV predisposes the patient with a CIED to thrombosis and endocarditis, both of which can lead to TV dysfunction – TR or stenosis [15–20]. There has been an increase in device infections, representing an emerging problem. Infection due to CIED necessitates lead extraction almost all of the time. Tricuspid valve dysfunction in this setting can occur as a result of leaflet destruction by the infectious process itself or during lead extraction. It is estimated that up to 24,000 lead extractions occur annually worldwide, and device infections remain the leading indication for extraction [21, 22]. Over time, leaflet and/or supporting structures adhere to and encapsulate CIED leads. As a result, transvenous lead extractions can cause TV damage, including leaflet avulsion [23–27] (see Video 10.1). Tricuspid valve damage can also result from surgical lead extraction [28].

Pacing-related TV dysfunction can occur by way of various mechanisms. Dyssynchronous LV electromechanical activation induced by left bundle branch block or RV pacing can result in systolic or diastolic dysfunction of the LV or in mitral regurgitation. This results in increased left-sided filling pressure and pulmonary artery pressure, leading to functional TR [29, 30]. Among 89 consecutive patients undergoing their first PPM implantation, TR increased after dual-chamber, but not after biventricular, PPM implantation further supporting this mechanism [31]. Other studies suggest that the physical presence of the lead itself, and not pacing per se, plays the major role in TV dysfunction, as the percentage of paced beats does not correlate with worsening TR [8, 32, 33].

Diagnosis of Tricuspid Valve Dysfunction Associated with CIED Leads

Echocardiographic assessment, two-dimensional (2D), three-dimensional (3D), and Doppler, are the mainstays for diagnosis of CIED-associated TV dysfunction. The diagnosis of TR in patients with CIEDs is similar to that in patients without endocardial leads. The CIED leads can result in echocardiographic imaging artifacts and signal attenuation because of their high acoustic impedance and reflectivity, resulting in underestimation of TR by Doppler color-flow mapping [34]. Other associated artifacts include scattering and acoustic shadowing, similar phenomena that are encountered in Doppler echocardiographic assessment of prosthetic valve regurgitation. This is somewhat mitigated with the use of transesophageal echocardiography (TEE). When TR is caused by an asymmetric impairment of leaflet mobility, which is usually the case with lead-related TR, the regurgitant jet can assume an eccentric or wall-hugging rather than central trajectory, resulting in loss of Doppler color-flow signal—known as the Coanda effect—and therefore underestimation of TR, as is similarly the case with mitral regurgitation caused by asymmetric leaflet tethering or prolapse.

A high index of clinical suspicion is required in conjunction with a careful physical assessment in patients in whom CIED lead-induced severe TR is suspected, as routine echocardiographic assessment may miss this, for the reasons stated above. In patients found to have severe TR from CIED leads, only 63% had a correct diagnosis based upon routine preoperative TTE [34], whereas all were found to have severe TR by preoperative or intraoperative TEE. In cases of suspected CIED-associated TR, it is important to inspect the pattern of hepatic vein flow by spectral and color-flow Doppler assessments, which are not affected by lead-induced acoustic artifacts. Holosystolic hepatic vein flow reversal is diagnostic of severe TR, whereas normal antegrade systolic flow excludes moderate and severe TR, although if the right atrium is severely dilated, the negative predictive value of hepatic vein systolic flow reversal may be reduced [35]. Therefore, Doppler assessment of the hepatic vein is essential in all patients with a CIED and will reveal many instances of severe TR not disclosed by standard color-flow imaging of the valve itself.

Treatment of Lead-Related Tricuspid Regurgitation

Surgical corrective intervention of severe TR induced by CIED leads includes suture (DeVega) annuloplasty, ring annuloplasty, and valve replacement with or without lead retention. For valve repair with lead retention, the lead is first surgically detached from any adherent interaction with valve leaflets or chordae tendineae.

The lead is repositioned by securing it in a location abutting the tricuspid annulus in a cleft created by suture-bicuspidization of the TV, to prevent leaflet impingement. Lastly, DeVega-type suture or ring annuloplasty can be performed in cases where the annulus is dilated [36–39].

On the one hand, ring annuloplasty can be done with an open C-ring (band), rather than a closed O-ring, in order to accommodate the lead within the ring [40–43]. On the other hand, a circumferential O-ring, providing superior support of the entire tricuspid annulus, can be deployed. However, the use of a circumferential annuloplasty ring requires displacement of the lead outside of the annulus, thereby entrapping it. The majority of case series describing TV repair or replacement resulting in an entrapped lead report normal function of both the valve implant and device following surgery. However, this method can lead to the possibility of damage to the lead or adverse effects on pacing or defibrillation in addition to precluding subsequent performance of transvenous lead extraction in the case of a future device infection.

Transvenous Lead Extraction to Treat Lead-Related Tricuspid Regurgitation

Over time, leaflets and supporting structures can adhere to and encapsulate CIED leads (see Videos 10.2 and 10.3). RV lead extraction can therefore result in TV damage, including leaflet avulsion (see Video 10.1). Current lead extraction methods employing sheath extraction with mechanical and laser-assisted dissection allow for extrication of the lead from encapsulating or ensheathing valve material with a low incidence of complications, such as worsening TR (0–5.6%), and a high procedural success rate (94–100%) [44–46]. Predictors of worsening TR after RV lead extraction include removal of greater than 1 lead, endocarditis involving the TV as the reason for explantation, and longer dwell time [45, 47].

Ultimately, whether lead extraction alone, without valve repair or replacement, is adequate to improve lead-related TR cannot be determined a priori with certainty. Because further damage may occur as a result of lead extraction, a valve replacement or repair strategy must be in place prior to RV lead extraction.

Lastly, there are no prospectively acquired data to support TR, in the absence of TV or device infection, as an indication for transvenous lead extraction [48]. However, when operative risk is low and patients have severe symptomatic TR with compelling three-dimensional (3D) echocardiographic evidence of valve dysfunction attributable to the lead, extraction should be considered, since the significant increment in morbidity and mortality associated with TR, with or without an interfering CIED lead, is now widely recognized.

Future Directions Including Leadless Pacemakers and his Bundle Pacing

Lead-related TV dysfunction can be eliminated by foregoing the use of transvalvular leads altogether. Strategies to provide pacing to the heart without crossing the TV include placement of a coronary sinus pacing lead, surgical epicardial placement of leads, and leadless pacing systems.

His bundle pacing results in a physiological activation sequence in the ventricles, thus leading to a narrow QRS complex, and minimizes the deleterious effects of RV pacing [49]. Additionally, because the His bundle penetrates the membranous septum on the atrial side of the TV leaflet insertion, His bundle pacing can occur without affecting TV closure and function. Importantly, effective and direct His bundle pacing may result in (1) a narrower QRS complex compared to biventricular pacing; (2) improvement of LV dimension, functional status, ejection fraction, and quality of life; (3) reduction of heart failure hospitalization frequency [50–56], and (4) avoidance of LV dyssynchrony caused by RV pacing as well as of the need to upgrade an RV pacing system to a biventricular pacing system in patients with pacing-induced dyssynchrony. Tricuspid valve function has not been directly assessed, but the absence of a lead that interferes with leaflet coaptation and the improved electromechanical coupling obtained with His bundle pacing compared with RV pacing should, in principle, preserve TV function [57].

Other novel approaches to reduce or eliminate many of the complications of conventional pacemakers, like TR, include leadless pacemakers. At present, these are transvenous single-chamber devices implanted in the RV apex (Nanostim, St. Jude Medical, St. Paul, Minnesota; and Micra pacing system, Medtronic, Minneapolis, Minnesota) [58, 59]. Initial data suggest that they are associated with a 99.2% rate of successful implantation and a 4% complication rate at 6-month follow-up [58, 60].

Conclusion

Clinical consequences of tricuspid valve dysfunction secondary to CIED leads are developing increasing recognition in parallel with recognition of those of RV and TV dysfunction in general. A higher level of clinical suspicion than has prevailed in the past, in conjunction with 3D echocardiography, may alert the clinician to the possibility that worsening heart failure may be a consequence of mechanical interference with TV leaflet mobility or coaptation and therefore amenable to lead extraction or valve repair or replacement. When clinical, hemodynamic, and echocardiographic assessment provides compelling evidence of lead-related severe TR, corrective intervention should be provided in a timely fashion, before the onset of severe annular and chamber dilation and severe RV dysfunction because, by that time, the lead itself will no longer be the problem, and the extant problem may not

be as amenable to corrective intervention. The future of CIEDs in which endocardial leads are absent or non-transvalvular is likely to be associated with a reduction in lead-related cardiac dysfunction.

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