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REVIEW ARTICLE

Cardiac Implantable Electronic Device Lead-Induced Tricuspid Regurgitation

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ABSTRACT

Background: Tricuspid regurgitation (TR) has traditionally been regarded as benign condition, but moderate to severe TR is now recognized as a predictor of morbidity and mortality. Cardiac implantable electronic devices (CIEDs) including (pacemakers, Implantable cardioverter-defibrillators (ICD) & cardiac resynchronization therapy (CRT)) have been increasingly used in the treatment of bradyarrhythmias and some types of tachyarrhythmias. Lead-induced TR (LITR) has emerged as a clinically significant complication following CIED-lead implantation, often underdiagnosed and undertreated. Our objective is to review the mechanisms, diagnosis, outcomes, and management strategies of CIED-associated TR, with a focus on recent advances and clinical implications. This narrative review synthesizes findings from observational studies, cohort analyses, and recent guideline updates on CIED-related TR. Literature was identified through PubMed and MEDLINE. Reported incidence of new or worsened TR after CIED implantation ranges from 7% to 45%, depending on study design, device type, and follow-up duration. Mechanisms include direct interference with TV apparatus (entanglement, perforation, leaflet impingement), pacing-induced right ventricular dyssynchrony, and implantation-related trauma. Significant LITR is associated with right-sided heart failure, impaired functional capacity, and increased mortality. Diagnosis is challenging due to acoustic shadowing, but 3D echocardiography improves visualization and identification of lead–leaflet interactions. Management options range from medical therapy and careful monitoring to lead extraction, surgical repair or replacement, and newer approaches such as leadless or His-bundle pacing.

Conclusion: Lead-induced TR is an underrecognized complication and early detection through standardized imaging and a multidisciplinary approach to management are essential. Future prospective studies are needed to clarify incidence, predictors, and optimal therapeutic strategies.

Keywords: Tricuspid regurgitation; Cardiac implantable electronic device; Lead-induced tricuspid regurgitation; Pacemaker; Implantable cardioverter-defibrillator

INTRODUCTION

Tricuspid regurgitation (TR), after the implantation of a cardiac implanted electronic device (CIED), is becoming widely acknowledged as a clinically significant complication especially in patients who are receiving transvenous right ventricular leads. Although some patients may already have TR from structural or functional right heart disease, new or worsened TR after CIED placement is often attributed to both mechanical and functional mechanisms [1]. Lead-induced TR may occur through entanglement with chordae tendineae, adhesion and fibrosis along the valve apparatus, tricuspid leaflets perforation or impingement [2]. Annular dilatation and increasing regurgitation may also be caused by right ventricular dyssynchrony caused by chronic pacing [3]. Depending on the imaging modality, device type, patient selection, and follow-up period, reported incidence rates range greatly, from 7% to over 40% [4]. Crucially, negative consequences such as increasing right-sided heart failure, reduced exercise tolerance, and elevated all-cause mortality have been linked to substantial CIED lead-induced TR [5]. Despite these

consequences, TR following CIED implantation is frequently underdiagnosed due to echocardiographic limitations and the subtle onset of symptoms. Standardized imaging, particularly with 3D echocardiography, along with careful clinical follow-up is therefore essential to identify high-risk patients [6]. Determining the best time for intervention, enhancing long-term prognosis, and directing preventive measures all depend on identifying predictors and comprehending the clinical effects of lead-induced TR [5, 6]. Although Lead-induced tricuspid valve interference was initially reported in the late 1900s, concentrated efforts to further quantify the extent of this interference have just recently been made.

ASSOCIATION BETWEEN CIED AND TRICUSPID REGURGITATION

Regurgitation and, less frequently, stenosis are examples of tricuspid valve (TV) dysfunction linked to cardiac resynchronization therapy (CRT), ICD, and PPM implantation. Both (regurgitation &stenosis) are of the primary causes of TV dysfunction. Tricuspid regurgitation (TR) following device lead implantation was originally documented almost fifty years ago. Since then, TR has been linked to the presence of device leads in many case reports, case series, and retrospective cohort studies. Since it is now known that TR is not a benign disease, interest in this area has grown. The reported incidence of acquiring considerable TR following CIED implantation ranges from 7% to 45% [7]. The results of early research employing 2-dimensional (2D) echocardiography revealed mixed findings about the connection between the degree of TR and the existence of a CIED. Some research suggested that CIED implantation was associated with an increase in TR severity, while other investigations reported no appreciable differences in TR severity before and after the implantation of endocardial leads. Other research, however, found that improved RV hemodynamics with pacing following right ventricular (RV) lead insertion was responsible for improving TR in subgroups of patients. Nevertheless, research that has included results in their methodology has demonstrated that CIED-mediated TR is linked to a worse prognosis [8].

Table 1: Summary of Major Studies Evaluating Tricuspid Regurgitation after Device Implantation

Author (Year)	Sample Size	Device Type	Imaging Method	Follow-up Duration	Incidence of New/Worsened TR	Notes
Seo et al. (2014) [1]	81	Pacemaker	TTE/TEE	Mean 3.5 yrs	24%	Mechanistic study; lead interference with tricuspid valve
Kim et al. (2016) [2]	146	Pacemaker	TTE	Median 2 yrs	21%	Lead impingement significantly associated with TR
Chang et al. (2018) [3]	1,502	PPM/ICD	TTE	Median 2.9 yrs	18%	TR increased risk of right-sided heart failure
Höke et al. (2014) [4]	239	Mixed (PPM/ICD/CRT)	TTE	5 yrs	38% (significant TR)	TR strongly associated with adverse prognosis
Delling et al. (2019) [5]	13,575	Pacemaker	TTE	5 yrs	24%	TR associated with increased long-term mortality
Lee et al. (2015) [20]	1,167	PPM/ICD	TTE	3 yrs	15%	Predictors: lead position and RA enlargement
Arabi et al. (2015) [16]	41	PPM/ICD	TTE	12 months	Progressive worsening (echo)	Small prospective cohort study

MECHANISMS OF CIED-INDUCED TRICUSPID REGURGITATION

Three categories of CIED-induced TR have been explained by the following mechanisms: device-mediated, pacing-related, and implantation-related.

Implantation-Related (Mechanical) Tricuspid Valve Dysfunction

The implantation technique used for CIEDs has changed throughout time and differs from operator to operator. Damage to the TV apparatus has been associated with several technological factors. Direct lead crossing through the tricuspid valve lowers the chance of trauma and tricuspid apparatus damage, which results in reduced TR. Nevertheless, the outcomes of this method have not been fully investigated. This contrasts with the conventional "prolapsing" method of lead implantation [9, 10]. According to certain studies, the more leads that flow through the tricuspid annulus, the higher the chance of developing worsening TR [11]. According to other research, defibrillator leads that are stiffer and bulkier have a larger risk of making TR worse than pacemaker leads [12]. As demonstrated by post-mortem examinations of hearts with device leads and in vivo 2D and more recently 3D echocardiographic studies, leads can interfere with the tricuspid valve apparatus by adhering to a leaflet, impinging upon a leaflet, interfering with the subvalvular apparatus, perforating or lacerating a leaflet, and avulsion of a leaflet. These events rarely occur during lead extraction [13].

Lead extraction also has the potential to damage the TV system and turn into an additional lead-mediated TR mechanism. It has been shown that employing more modern lead extraction methods, like laser-assisted lead dissection from adhering material, lowers the possibility of problems like worsening TR [7].

Pacing-Related (Electromechanical) Tricuspid Valve Dysfunction

One known cause of mitral regurgitation is dyssynchronous LV electromechanical activation brought on by RV pacing or left bundle branch obstruction. It is debatable if a comparable mechanism applies to TR [14]. Research links either pacing-induced LV or RV dyssynchrony to the development of TR following lead implantation. It is believed that MR or systolic or diastolic malfunction of the left ventricle, which increases pulmonary artery pressure and left-sided filling pressure and leads to secondary (functional) TR, are caused by pacing-induced dyssynchrony. This idea is supported by the observation that in 89 consecutive individuals having their first PPM implantation, TR increased following dual-chamber (as opposed to biventricular) PPM implantation [15].

Echocardiography to Diagnose CIED Interference

2D and 3D echocardiography can be used to image the TV from both TTE and TEE perspectives. All three leaflets can be viewed concurrently from the RA and RV views, and the location of the device lead in respect to the TV leaflets and annulus is frequently clearly visible.

Considering the high acoustic impedance of the device lead, color Doppler imaging may understate the severity of TR and significant reflectivity; this effect is more noticeable on TTE than TEE [17]. According to recent research, the size and quantity of tricuspid leaflets vary greatly, making it impossible to identify exactly which leaflet pair is being photographed in any of the common perspectives [18].

Consequently, a technique for accurately localizing TV anatomy has been proposed: 2D imaging of the TV leaflets focusing on specific neighboring anatomic landmarks. For instance:

1) The anterior leaflet is captured in the near field of the RV inflow image when the septum is visible, and the septal leaflet is captured in the far field.

2) If the aortic valve can be seen in the apical 4-chamber view, the anterior and septal tricuspid leaflets are photographed; if the coronary sinus can be seen in this imaging plane, the septal and posterior leaflets are scanned.

3) In the parasternal short-axis view, only the anterior leaflet is ever visible [18].

3D echocardiography makes it easier to see

the device lead, and CIEDs can be observed in different positions in relation to the tricuspid annulus and leaflets. They may be located in the center of the valve, against, impinging on, or adhering to a leaflet, or in the commissures (anteroseptal, posteroseptal, or anteroposterior).

Greater levels of TR were linked to leads that were designated as "impinging" or "adherent" While the "impinging" lead directly disrupts leaflet coaptation, the adherent lead is attached to the leaflet/tricuspid apparatus but still moves with it. Significant TR was less likely to be linked to leads that were positioned commissurally or in the "center of the valve." [19].

Anytime there is severe TR in a patient using a device lead, especially if an echocardiography obtained before the device lead was implanted showed little to no TR, lead-induced TR should be investigated. TR jet hugging the device lead, leaflet mal-coaptation, lead adherence to the sub-valvular structures which tend to move with the sub-valvular apparatus, extreme lead displacement against the septum and non-RV outflow tract lead position are some indicators of potential lead-induced TR on 2D echocardiography [20].

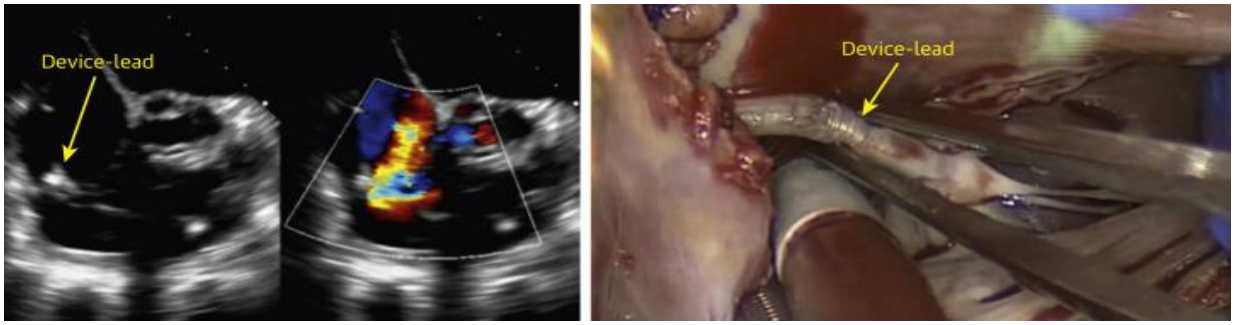

Figure (1): Patient with Device Lead-Induced TR, left: 2D and color Doppler showing the device lead traversing the TV with significant TR. Right: Intraoperative view showing the device lead entrapped by fibrous/inflammatory tissue and adherent to the TV leaflets and subvalvular apparatus [17].

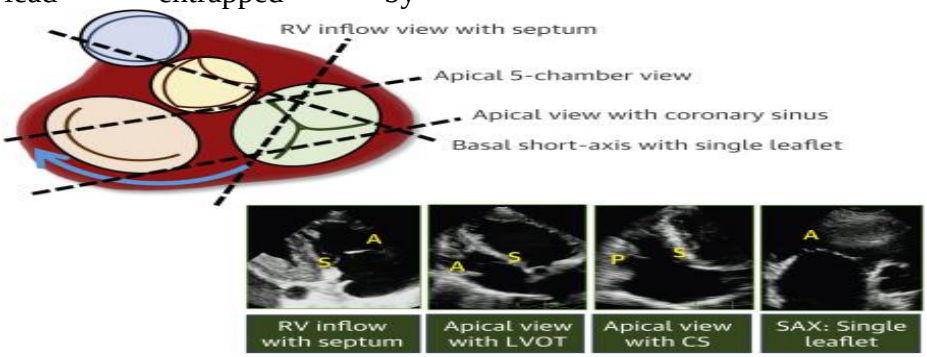

Figure (2): Targeted Imaging to Include Specific Landmarks to Allow More Predictable Imaging of TV Leaflets and Leaflet Pairs: In the right ventricular inflow view (bottom, far left), when the septum and coronary sinus are seen, the leaflets imaged are the septal (S) and anterior (A). In the apical 5-chamber view (bottom middle left) the S and A leaflets are visualized. In the apical view, when visualizing the coronary sinus (bottom middle right), the S and posterior (P) leaflets are seen. In the short-axis view (bottom far right) when a single leaflet is visualized, the A leaflet is imaged [17].

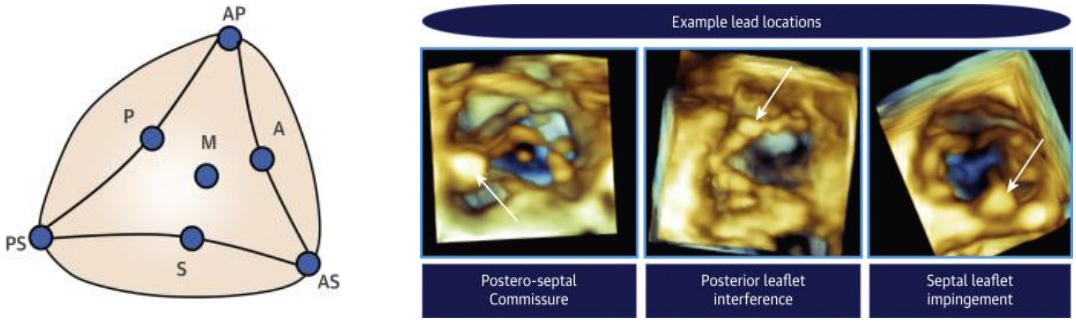

Figure (3): Three Possible Device Lead Tricuspid Leaflet/Valve Interactions. (Left) Device lead in the posteroseptal commissure. (Middle) Device lead adhering to the posterior leaflet. (Right) Septal leaflet impingement by the device lead [17].

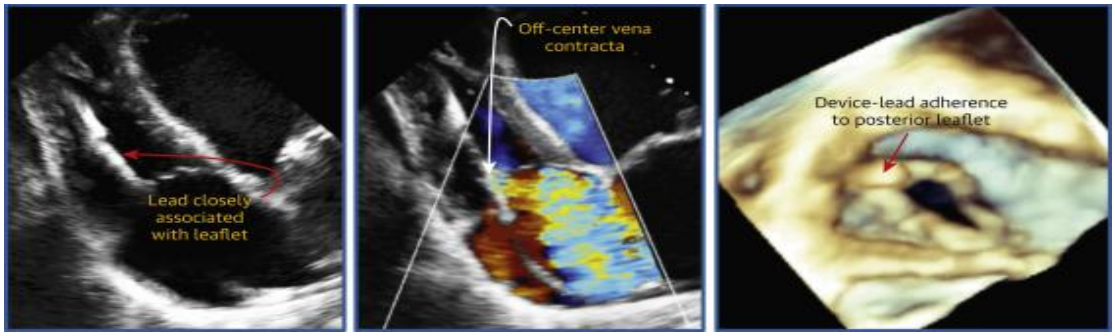

Figure (4): ICD Lead Interference with a Tricuspid Valve Leaflet. 2D (left), 2D color Doppler (middle), and 3D zoom (right) dataset of the tricuspid valve as seen from the right ventricular perspective in a patient with an ICD lead interfering with the posterior leaflet [20].

CIED-INDUCED TRICUSPID REGURGITATION OUTCOMES

TR linked to CIED causes the right heart to remodel, leading to reduced RV function and enlarged right atrial and ventricular sizes, in the worst case, the clinical manifestations of lead-related TR include peripheral edema, ascites, and hepatomegaly. About 50% of patients with severe lead-related TR who needed TV surgery initially had signs of acute right heart failure [13]. In terms of how TR affected CIED patients' survival, severe TR has also been associated with increased mortality [8].

Little information is available regarding when TR first appears or gets worse following lead implantation. A different pattern of worsening TR was found using the TR quantification method in a small report that included 41 patients who had repeat echocardiograms at 1-, 6-, and 12-month intervals as well as right after CIED.

Following implantation, the patients' Color Doppler assessments revealed an initial tendency of decline in first month, which stabilized six and twelve months later. On the other hand, quantitative evaluation showed that throughout the course of the follow-up period, the VC and PISA progressively grew while RV&RA sizes and RV function gradually declined [16].

MANAGEMENT OF PACEMAKER-INDUCED TRICUSPID REGURGITATION

The existence of RSHF symptoms, the severity of TR, the degree of lead-related valve damage, the degree of RV dysfunction and TA dilatation, and the differentiation between lead-related primary TR and secondary functional TR are some of the factors that determine how CIED-related severe TR is managed [7].

The frequent occurrence of both disorders, especially in this patient group, makes distinction challenging. Patients with left-sided illness, which is known to result in subsequent right-sided dysfunction and eventually functional TR, frequently have pacemaker leads placed.

Additionally, Lead-related severe primary TR

may cause RV remodeling with tricuspid annular (TA) dilatation and leaflet mal-coaptation if treatment is not received promptly. Even if the lead is eliminated and the leaflets' functionality is restored, this leads to a superimposed secondary TR that frequently does not get better (figure 5) [7].

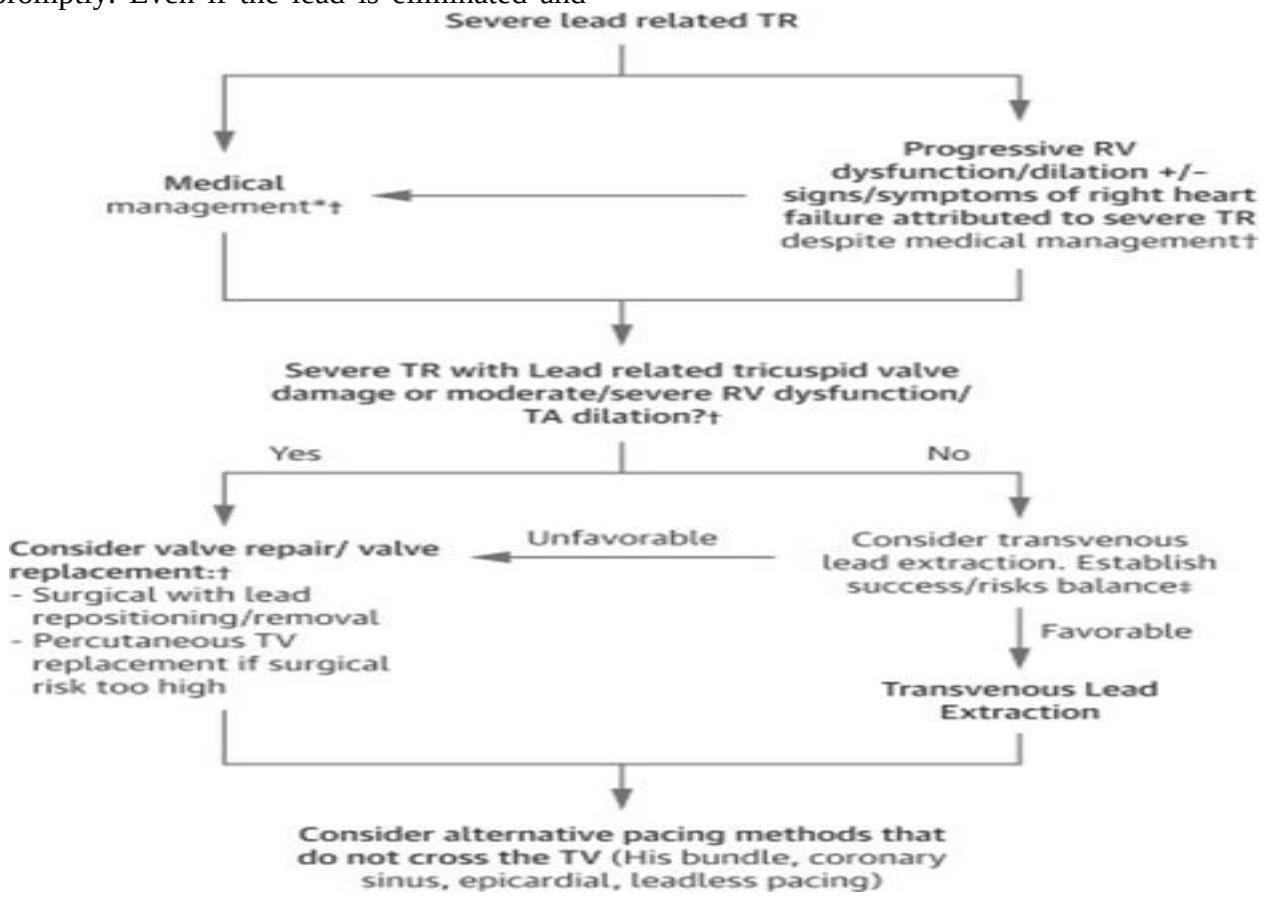

Figure (5): Management strategies for severe lead induced TR include medical therapy, consideration for transvenous lead extraction, and TV repair/replacement [7].

Medical Therapy

Diuretics are the mainstay of treatment for people having symptoms of right heart failure and severe TR. In addition to the commonly used loop diuretics, aldosterone antagonists are suggested as beneficial additional medications, especially when there are secondary aldosterone elevation and hepatic congestion. However, it is unknown what the long-term effects of this cautious therapeutic approach will be in patients with severe lead-induced TR [8].

Transvenous Lead Extraction

There is currently no prospective data and, hence, no guideline recommendations to support lead extraction for severe TR in the absence of device or lead infection, despite significant improvements in the safety and efficacy of lead extraction over the past ten years. This is demonstrated by the fact that most extractions are still caused by device infection [8].

Additionally, TV damage can make lead extraction more difficult and aggravate TR. The removal of several leads, advanced age, and endocarditis involving the TV as the indication for device explanation all predict TR getting worse after TLE [21].

Irreversible severe TR can result from unfavorable RV remodeling and TA dilatation [7]. Lead extraction should be beneficial if there is no severe RV dysfunction or TA dilatation, the TR cause is believed to be lead-related, and the surgical risk is minimal [7].

Leads inserted within a year may normally be extracted using basic traction techniques, while leads that have been there for more than a year typically require more modern equipment (mechanical and laser-assisted dissection which allow removing lead from encasing or ensheathing valve material with low incidence of complications [21, 22].

Surgical Treatment

There are some differences in the intensity of the recommendations between the ESC valvular guidelines and the American Heart Association, and the rationale for TV surgery in patients of severe primary TR without left-sided illness are not well supported by evidence.

For severe primary TR, TV surgery should be considered in two situations: first, in patients who continue to have symptoms after taking diuretics (strongly recommended by ESC and weakly recommended by AHA/ACC); and second, in patients who show signs of progressive RV dilatation or dysfunction (strongly recommended by ESC and very weakly recommended by AHA/ACC).

In these situations, both the lead and the valve should be addressed by any potential operation. Lead damage to valves can be fixed via suture (DeVega) annuloplasty, ring annuloplasty, or valve replacement with or without lead retention.

Lead management options include lead repositioning, which ensures that the lead is fastened in a position that does not impede valve function, or extraction and substitution with a pacing method (coronary sinus, epicardial, leadless pacing) that avoids crossing the TV.

The latter can be achieved by using suture approximation of both leaflets to secure the lead in the leaflet commissure, usually between the septal and posterior leaflets [23].

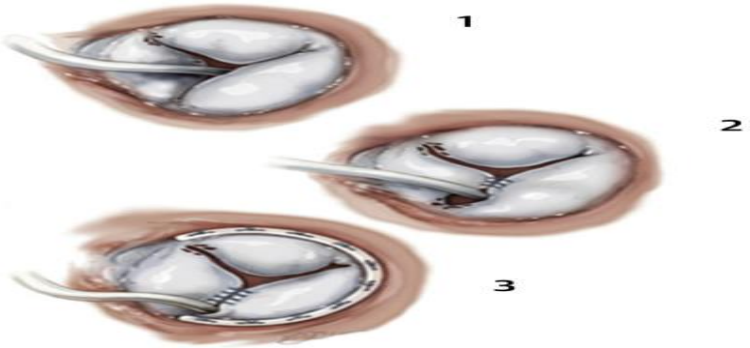

Figure (6): Steps of TV surgical Repair (TV annuloplasty). (1) Mobilized defibrillator lead and leaflets of the tricuspid valve. (2) Lead repositioned in the cleft between the septal and inferior and posterior leaflets, with suture approximation of the leaflets above the cleft. (3) Repositioned lead with cleft closure and tricuspid valve annuloplasty [23].

Percutaneous (Transcatheter) Treatment

The alternatives for percutaneous TV repair have increased recently. None of these are intended for severe TR caused by lead, though. Instead, the main goal of these methods is to cure functional TR; some of them have excluded patients who have defibrillator leads or PPM [24].

They are classified into 3 groups according to the mechanism and the anatomic target:

1) Annuloplasty devices: based either on transcatheter suture or ring implantation techniques, and include: **Trialign** device, **TriCinch** device and **Cardioband** tricuspid valve reconstruction system [27].

2) Coaptation devices: designed to improve leaflet coaptation in functional TR and include 3 devices: **Tri-Clip** (Used for Tricuspid transcatheter edge-to-edge repair (T-TEER), **FORMA** repair system and the **PASCAL** system [27].

3) Heterotopic caval valve implantation (CAVI): include techniques for heterotopic caval valve implantation, where self-expanding bioprosthetic valves or balloon-expandable valves are placed in the inferior and superior vena cava [28].

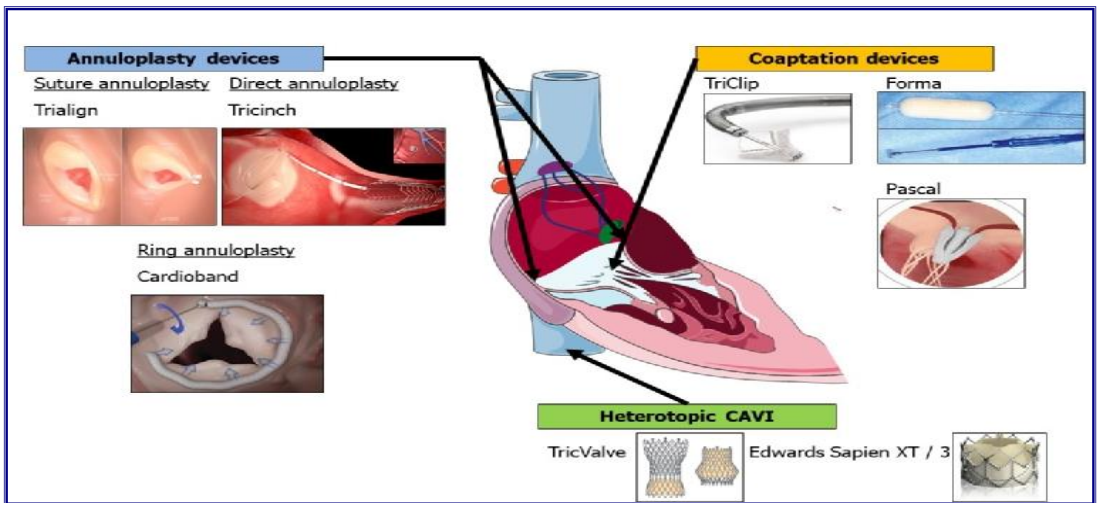

Figure (7): Transcatheter techniques targeting functional TR including: Annuloplasty devices (Trialign device, TriCinch device and Cardioband), Coaptation devices (Tri-Clip, FORMA repair system and the PASCAL system) and Heterotopic caval valve implantation (CAVI) [27].

TRANSVALVULAR LEAD SUBSTITUTES

TV dysfunction caused by lead can be lessened by:

1) Using polytetrafluoroethylene coated CIED-leads to decrease inflammatory and foreign body endocardial fibrotic reactions, preventing silicone-induced contact dermatitis.

2) Removing the transvalvular lead

Following the removal of the lead that was shown to be the perpetrator, alternatives to pace the heart without crossing the TV are needed.

Pacing the left ventricle through the **coronary sinus** is conceivable, but might be constrained by lead instability, high capture thresholds, and phrenic nerve stimulation.

Epicardial pacing, the invasive nature of the procedure and the somewhat greater lead failure rate as compared to transvenous leads are of the limitations [25].

His bundle pacing has been focused on in research since 1967, which reduces the negative effects of RV pacing by narrowing the QRS complex, preventing ventricular dyssynchrony, and giving the ventricles a more physiological activation sequence.

His bundle can be paced without impairing TV closure and function since it penetrates the membranous septum on the atrial side of the TV leaflet insertion.

Leadless pacemakers are another innovative way to lessen or completely eradicate many of the issues, such as TR, associated with traditional RV pacing.

Currently, the RV apex is implanted with transvenous single-chamber devices including: **Micra pacing system** (Medtronic, Minneapolis, Minnesota) and **Nano-stim** (St. Jude Medical, St. Paul, Minnesota) [26].

Table 2: Recent Trials and Guidelines Relevant to CIED and Tricuspid Regurgitation (2020–2024)

Item	Year	Type	Population / Intervention	Key Finding
Meta-analysis of TR after device implantation [29]	**2023**	**Meta-analysis**	**Patients with CIEDs (multiple observational studies)**	**Confirmed nontrivial incidence of new/worsened TR and identified predictors; highlighted heterogeneity.**
Systematic review — His-bundle pacing and TR [31]	**2022**	**Systematic review**	**Patients undergoing His-bundle pacing (HBP)**	**Mixed results: some studies report improvement, others worsening of TR.**
TRILUMINATE	**2023**	**Randomized**	**Patients with**	**T-TEER reduced TR severity**

Item	Year	Type	Population / Intervention	Key Finding
Pivotal (TriClip T-TEER) [30]		**controlled trial (pivotal)**	**symptomatic severe TR randomized to TriClip T-TEER vs medical therapy**	**and improved quality of life at 1 year; improved symptoms and TR grade though no clear 1-yr difference in mortality/HF hospitalization.**
CLASP II TR / PASCAL program (ongoing) [32]	**2021–2024**	**Ongoing pivotal trial**	**Patients with severe TR treated with PASCAL device vs medical therapy**	**Ongoing pivotal trial: preliminary reports show safety and feasibility of PASCAL repair.**
ESC/EACTS Guidelines — Management of Valvular Heart Disease [33]	**2021**	**International guideline**	**Patients with valvular disease (includes TR)**	**Re-emphasizes surgical indications for severe TR; notes emerging role of percutaneous options.**
ACC/AHA Guideline — Management of Valvular Heart Disease [34]	**2020/2021**	**Guideline (ACC/AHA)**	**Broad valvular disease guidance (includes tricuspid valve)**	**Provides recommendations for surgical TR management; acknowledge percutaneous therapies are emerging.**

LIMITATIONS AND FUTURE DIRECTIONS

The current evidence on CIED lead-induced tricuspid regurgitation (LITR) has several important limitations. First, most available studies are retrospective, single-center, and heterogeneous in design, making it difficult to draw firm conclusions about incidence and causality. Second, the diagnosis of TR is often based on conventional 2D echocardiography, which may underestimate severity due to lead-related artifacts and the complex anatomy of the tricuspid valve. The adoption of standardized imaging protocols, particularly 3D echocardiography and multimodality approaches, is needed to improve diagnostic accuracy. Third, patient populations and device types vary widely across studies, with inconsistent reporting of baseline TR and follow-up duration, which may contribute to the broad incidence range reported (7–45%). In addition, prospective data on the natural history of LITR and its optimal management remain limited. There is no consensus on the timing of intervention, and current guideline recommendations are based largely on expert opinion rather than randomized evidence. The role of transvenous lead extraction, leadless pacing systems, His-bundle pacing, and novel percutaneous tricuspid therapies require further investigation. Future research should focus on large, multicenter prospective trials to determine the true prevalence and predictors of LITR, as well as randomized studies to assess the efficacy of emerging treatment strategies. Establishing standardized definitions and echocardiographic criteria for lead-induced TR will also be critical to enable consistent reporting and facilitate clinical decision-making.

Conflict of Interest: None

Financial Disclosures: None

Availability of the data: Upon reasonable request, the associated author will make the datasets created and/or examined during the

current work available.

Authors contribution: In addition to writing and getting the book ready for publication, the writers oversaw gathering and analyzing the data. The final version was examined and approved by all authors.

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