Chapter 4
Tricuspid Regurgitation in Patients with Pacemakers and Implantable Cardiac Defibrillators

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Abstract Tricuspid regurgitation (TR) secondary to placement of implantable cardiac devices, including defibrillators and pacemakers, is now a definitive entity. TR usually occurs over time after lead implantation. Mechanisms include laceration of valve leaflets, entrapment of leads causing scar formation, interference with valve coaptation, and asynchronized activation of the right ventricle. Diagnosis by clinical exam and 2-dimensional echocardiography is further supported by 3-dimensional echocardiography and/or computed tomography. Management typically involves medical management as well as percutaneous extraction of the offending leads. Prospective methods to prevent or reduce the incidence of this complication include improved imaging modalities intraoperatively, procedural techniques, and particular lead placement. Newer technologies can help mitigate this problem but their effectiveness remains to be seen in larger prospective trials.

Keywords Tricuspid valve • Tricuspid regurgitation • Implantable cardiac defibrillator • Permanent pacemaker • Color Doppler flow • Ring annuloplasty • Bioprosthetic valves

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Introduction

Tricuspid regurgitation (TR) in the setting of permanent pacemakers (PPM) and implantable cardioverter defibrillators (ICD) is not an uncommon complication, with a prevalence reported to be between 25 and 29% and an incidence of worsening preexisting TR from 10 to 25% [1–12]. Our understanding of the problem is now emerging in the wake of increased PPM and ICD implantations worldwide [13–15]. The association was first described in 1980 by Gibson and colleagues, in which a case of a young 23-year-old woman was described to have developed TR following the placement of a PPM [16]. Since then, in the last three decades the mechanisms, incidence, risk factors, as well as management and prognosis of this curious yet potentially formidable repercussion have been further elucidated.

Pathophysiology and Mechanisms (See Fig. 4.1)

Various potential mechanisms have been described to explain the development of TR following PPM and/or ICD implantation. These mechanisms can be categorized as related to the tricuspid valve (TV) itself, the PPM or ICD leads, as well as cardiac structure and function.

Tricuspid valve: Tricuspid valve trauma, laceration or perforation, and scar formation as a consequence of the PPM or ICD leads potentiate and contribute to mal-apposition and improper coaptation of the valve leaflets [1, 17–20]. Leaflet perforations or lacerations are most notably present in the posterior leaflet. In TR which develops over years after PPM implantation, it has been suggested that adhesion of the TV leaflet itself to the pacer lead results in restricted movement, and therefore, improper coaptation of the posterior leaflet with the septal and anterior leaflets [21]. Infective endocarditis is also a potential complication of lead placement which similarly affects the TV leaflets via adhesions and vegetation, and therefore contributes to another mechanism of subsequent TR [17].

PPM and ICD leads: Physical and mechanical complications resulting from the introduction of PPM or ICD leads may also contribute to TR. Mechanisms (see Fig. 4.2) include thrombus and fibrous tissue formation on the leads, adherence of the lead to the TV apparatus, and impingement and entanglement in the TV as well as within the chorda apparatus. One small study in particular found that the mechanism of TR after pacemaker implantation was related to lead impingement in 39%, lead adherence in 34%, lead perforation in 17%, and lead entanglement in 39% [19].

Within 12 h of implantation of PPM or ICD, the formation of neoendocardium results in development of fibrous sheaths surrounding the electrode [1]. The consequence is multiple endocardial attachments, fibrosis, and adhesion which potentially affects TV function. A thin fibrin layer begins to develop around the wire during this time. Approximately 4–5 days following implantation, thrombosis on

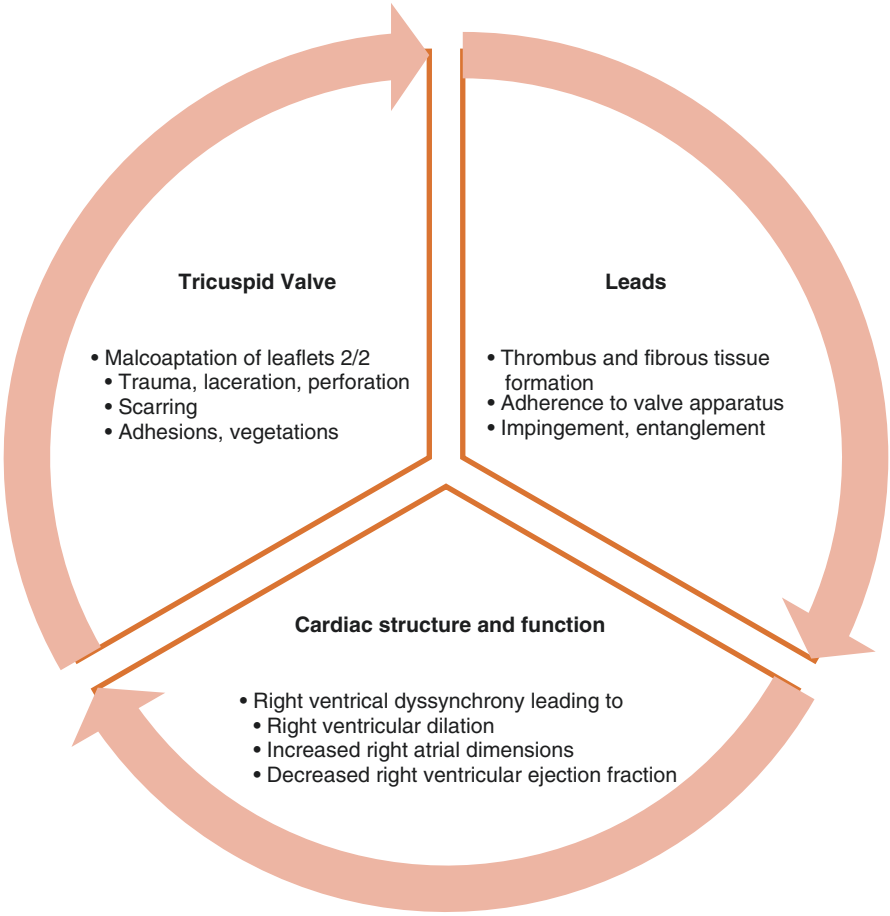

Fig. 4.1 Mechanisms and pathophysiology of lead-related tricuspid regurgitation

the lead and edema of the valve tissue itself often occurs [22]. Development of acute TR as a result of this remains to be controversial, as the frequency of acute TR varies in the literature [9–11, 23].

In addition to tissue homeostasis as a complicating and etiological factor, mechanical and physical complications of the leads coming in contact with the TV apparatus contribute to the development of TR. Leads positioned directly on the annulus or in the commissure between the leaflets may lead to obstruction and a subsequent progression of TR. In fact, it has been described that the majority of lead-related TR occurs when the leads are placed between the posterior and septal leaflets in particular [18]. One post-mortem study provided evidence of other mechanical complications, including leads fixed by fibrous tissue to the tricuspid orifice, as well as leads penetrated through the chordae tendinae [24]. Another less common mechanical etiology includes other valvular interventions leading to TR, such as one case of TR

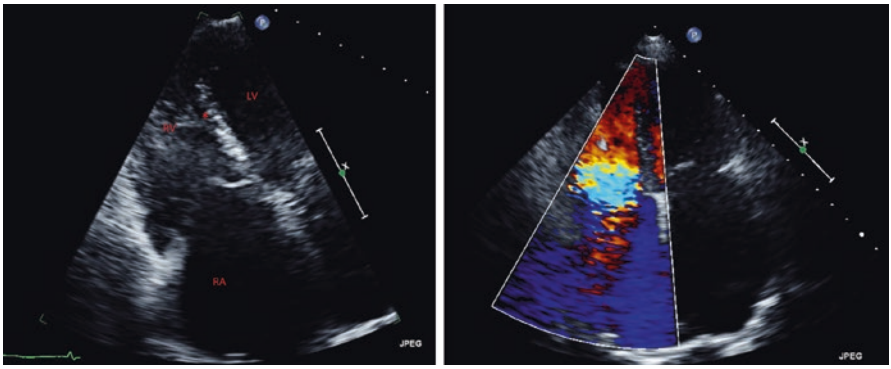

Fig. 4.2 (**a**) Section of the apical four-chamber view showing dilated right atrium (RA), dilated right ventricle (RV) and *pacemaker lead placed in the RV. (**b**) There is severe (4+) tricuspid valve regurgitation caused by annular dilatation. There is a centrally directed regurgitant jet. The left atrial cavity is severely dilated. RV systolic tissue Doppler velocity is 13.1 cm/s. Tricuspid annular displacement is 1.8 cm

years following PPM lead implantation and one month following aortic valve replacement [25]. It is postulated that the aortic valve replacement may have led to conformational changes between the tricuspid valve and the pacemaker leads.

Cardiac structure and function: Right ventricular (RV) dyssynchrony resulting from improper RV activation via the pacemaker has also been described as a potential mechanism. This may also be related to lead position, as one study showed a statistically significant increase in TR after PPM or ICD placement when the lead was apically placed versus in the right ventricular outflow tract (RVOT) [7]. Studies evaluating patients with 2-dimensional transthoracic echocardiography (2D TTE) prior to and following implantation of a PPM or ICD have also demonstrated significant RV dilation, increase in RA dimensions, as well as decrease in RV ejection fraction (RVEF) at up to one year following the procedure [4]. RV pacing frequency and dependence at follow up, however, has shown to have no effect on worsening of TR severity [6, 7].

Clinical Presentation

Clinical symptoms: The presentation of TR secondary to PPM/ICD placement may involve symptoms of decompensated right-sided congestive heart failure, such as abdominal distension and fullness, lower extremity edema, dyspnea on exertion, and palpitations related to atrial fibrillation [16, 21, 26]. An enlarged, pulsatile liver is a late finding [27]. In one study, patients with significant lead-induced TR following PPM or ICD implantation (increase of TR severity by ≥2 grades at follow up)

had more heart failure related events. This significant TR was even independently associated with increased all-cause mortality [5, 28]. Many patients may remain asymptomatic despite the presence of new or worsening TR. Larger studies have demonstrated that the majority of patients have new-onset or worsening of pre-existing TR several years following implantation, with some suggestion of acute worsening of TR in a small number of patients.

Physical examination: The physical examination may reveal the characteristic respirophasic, high-pitched, holosystolic murmur at the left lower sternal border that increases with inspiration (Carvallo's sign or maneuver). However, in many this murmur is unimpressive. In fact, the literature reports that only 28% of those with TR evidenced by echocardiography may have a regurgitant murmur on physical exam [29]. Nevertheless, when detected the Carvallo's maneuver has a sensitivity and specificity of 80% and 100%, respectively [30]. The TR murmurs that increase with inspiration are different from those which are associated with congestive heart failure, which often diminish with inspiration.

Other findings on exam may be consistent with isolated right-sided congestive heart failure, such as jugular venous distension, pulsatile liver, abdominal distension, and lower extremity pitting edema [19, 31]. Hepatojugular reflex may also be seen, with a sensitivity of 66% and specificity of 100% in detecting TR [30]. Likewise, the right atrial V wave is highly sensitive, yet it is not entirely specific for detecting the presence or severity of TR [32]. In addition to signs and symptoms of right-sided heart failure, some may have concurrent signs and symptoms of left-sided heart failure, especially those with some functional TR prior to the procedure and those who need ICD implantation for reduced left ventricular ejection fraction (LVEF).

Risk factors (see Table 4.1***):*** The risk factors for developing TR following PPM/ICD implantation are not entirely understood. Some predictors have been shown to be significant in recent studies. Advanced age is found to be a risk factor, with an average age of 73 years [7, 8]. Other predictors include body mass index, pre-device atrial fibrillation, heart rate, moderate or severe mitral regurgitation, history of mitral valve surgery, pulmonary artery systolic pressure ≥37 mmHg, elevated right ventricular systolic pressure and RV dilation [5, 7]. There is conflicting data in the literature regarding whether the placement of more than one lead predisposes to worsening of TR. In the pediatric population, a risk factor for lead-related TR was congenital heart disease which is not right-sided [9].

Table 4.1 Predictors of tricuspid regurgitation following PPM/ICD lead implantation

Predictors of lead related tricuspid regurgitation
Advanced age
High BMI
Atrial fibrillation
Tachycardia
Mitral valve disease
Pulmonary artery pressure ≥37 mmHg
Elevated right ventricular systolic pressure
Right ventricular dilation

Imaging

Diagnosing TR requires both 2D echocardiography (see Fig. 4.3) and color Doppler flow mapping. The severity of TR is graded based on the direction and size of the regurgitant jet, the presence of proximal flow convergence, and vena contracta width [33]. Using the vena contracta width of ≥6.5 mm, the sensitivity and specificity of detecting severe TR is 88.5% and 93.3%, respectively [34]. Other findings in new or worsening TR following PPM or ICD placement include increased RV and RA dimensions, greater pulmonary artery systolic pressure, elevated right ventricular systolic pressure, and decreased right ventricular ejection fraction (RVEF) compared to the pre-procedural values [4].

The utility of 2D echocardiography may be limited as it may underestimate the presence and severity of TR. It proves to be difficult to appreciate the full anatomical relationships between the TV and the ICD or PPM lead(s), as only two out of the three leaflets are visible simultaneously when using any 2D imaging plane [18, 35]. The posterior leaflet, which is implicated in most cases, is only visualized in some views, and is less commonly imaged during the routine echocardiographic examination [35]. In fact, the PPM lead may become entrapped in the thickened, fibrotic, and fused posterior and septal leaflets. These leads are visualized in only 12–17% of patients using 2D echocardiography [18, 19].

Three-dimensional transthoracic echocardiography (3D TTE) affords the ability to visualize all three TV leaflets and the short axis of the TV, not obtainable with 2D echocardiography. This allows the assessment of the route and position of the PPM/ICD lead within the TV apparatus [18, 31, 35, 36]. Mediratta et al. demonstrated that 3D TTE clearly depicted lead position in 90% of patients, in which 46% of patients had impinging leads visualized in the posterior (20%), septal (23%), and

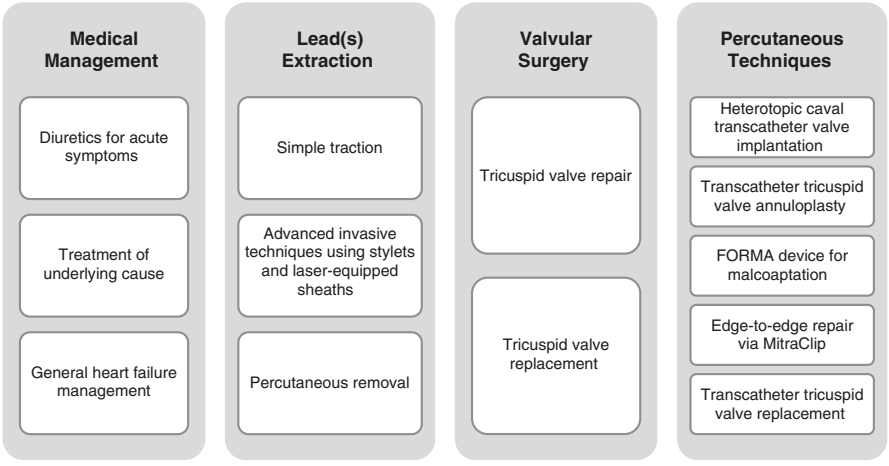

Fig. 4.3 Management of tricuspid regurgitation secondary to PPM/ICD lead implantation

anterior (4%) leaflets [36]. Those who did not have lead impingement, i.e. when the lead was visualized intercommisurally or in the middle of the tricuspid orifice, did not have evidence of significant TR compared to those with lead impingement. Due to this strong association between lead impingement and post-procedural TR, it has been suggested that 3D TTE targeted guidance of device- lead placement may be beneficial to avoid lead impingement, as lead placement is solely done under fluoroscopic guidance as of now [36]. It is undetermined, however, whether the lead would maintain its position from the time of placement to the time of development of TR, and therefore, the ultimate utility of intraprocedural 3D TTE is unclear. Additionally, due to the need of dedicated probes and image analysis software, as well as higher cost, 3D echocardiography is not widely used at the moment.

Other evolving imaging methods include contrast-enhanced multidetector computed tomography, which can indirectly be used to detect and grade TR. This is based on early opacification of hepatic veins or the inferior vena cava during first-pass intravenous contrast enhancement. In detecting echocardiographic TR, this particular method has a sensitivity of 90.4% and a specificity of 100% [37, 38].

An additional imaging modality is cardiac magnetic resonance (CMR), which can be used to detect and quantify TR based on the regurgitant jet area and volume. The sensitivity and specificity of this is 88% and 94%, respectively, compared to right ventricular angiography. Despite the favorable detection of this imaging modality, most pacemaker devices and leads are not compatible with CMR [39].

Management (See Fig. 4.4)

Medical management: Medical management has been largely studied in patients with functional TR, which involves treating the underlying cause as well as management of congestive heart failure [37]. Aggressive volume diuresis in acute decompensation and general balance of hemodynamics largely by the use of diuretics may be beneficial. There is, however, a paucity of data elucidating the outcomes of these patients with lead-related TR who are medically managed.

Lead(s) extraction: Lead placement is acutely associated with inflammatory changes, as well as chronically with fibrosis and scar tissue formation, which allows for lead adherence to the TV. The main indication for lead extraction is device and lead related infection [40]. The methods and techniques for lead extraction have become more sophisticated and specialized over time. Some leads can be removed by simple traction alone, while others require advanced techniques using locking stylets and laser-equipped sheaths. Percutaneous removal of PPM or ICD leads is often performed in large specialty centers with advanced technical skill and experience. However this carries significant and potentially fatal risk [31]. Major complications of lead extraction include cardiac or vascular avulsion requiring open chest interventions, pulmonary embolism, respiratory distress, stroke, and even death in up to 0.5% of patients.

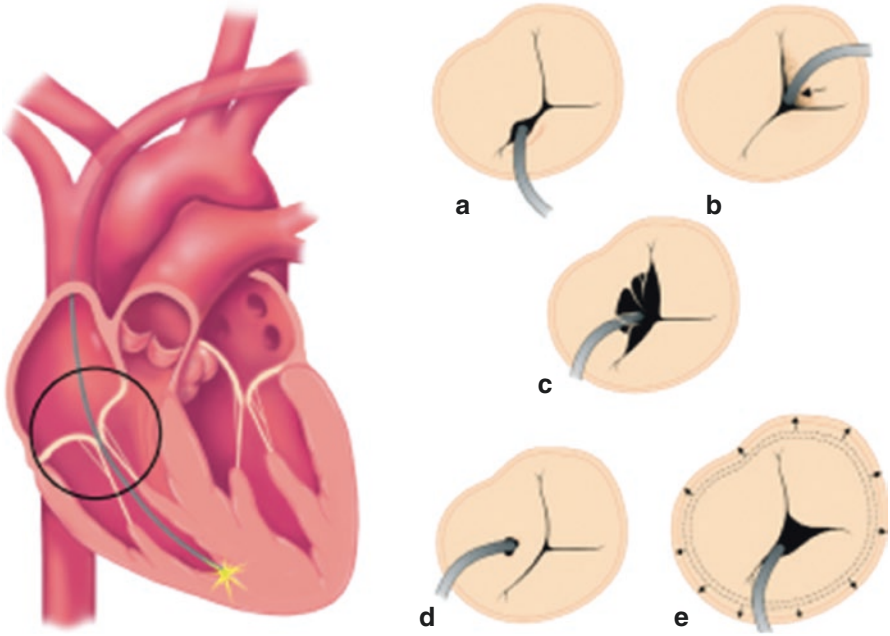

Fig. 4.4 Mechanisms of mechanical tricuspid regurgitation in the setting of permanent pacemaker or implantable cardioverter-defibrillator leads. (**a**) Valve obstruction caused by lead placed in between leaflets. (**b**) Lead adherence due to fibrosis and scar formation to valve causing incomplete closure. (**c**) Lead entrapment in the tricuspid valve apparatus. (**d**) Valve perforation or laceration. (**e**) Annular dilatation

However, in the last decade, success rate of lead extraction has been between 95% and 97%, and the complication rate has remained low at 0.4–1% [40].

Lead extraction itself may paradoxically lead to worsening TR [31, 35, 41]. Major risk factors and predictors of developing TR after extraction are the use of laser sheath or any additional tools for extraction beyond simple traction, extraction of more than two leads, female sex, and patients with longer duration of implantation [42]. Fortunately, There is no significantly increased mortality in those who develop TR post-extraction compared to those who do not develop TR. Tricuspid regurgitation is more likely to occur after PPM extraction than ICD lead extraction. This may be due to a longer duration of implantation or more fibrous tissue deposition and adherence to the TV [41].

Valvular surgery: The decision to operate on a regurgitant TV depends on the severity and clinical situation [27]. Tricuspid valve surgery is clearly indicated in primary severe TR at the time of left-sided valve surgery. It can also be considered in those with symptomatic severe TR who are unresponsive to medical management. Additionally, it may be considered in those who are asymptomatic or have minimal symptoms but have increasing RV dilation and dysfunction prior to any clinical right-sided heart failure.

Tricuspid regurgitation is managed surgically either via surgical repair or replacement depending on the etiology and mechanisms of valve dysfunction. Tricuspid valve repair often involves suture or ring annuloplasty as well as additional adjunctive techniques. It is often used in the case of secondary TR, with the goal of restoring tricuspid annulus geometry as well as concurrently reducing RV afterload by correcting left-sided valvular dysfunction [27]. On the other hand, tricuspid valve replacement is generally done when valve repair is technically not feasible, as in the setting of complex lesions causing severe primary TR and severe tricuspid stenosis. Those with secondary TR with marked RV remodeling and leaflet tethering may also benefit from replacement rather than repair.

It is estimated that approximately 8000 surgical tricuspid repairs occur annually in the United States, the majority of these cases involving patients without ICD or PPM-related TV pathology. Tricuspid valve repair has a success rate of above 85%. Recurrence of TR is common and occurs in about 20–30% of patients [37, 43]. Tricuspid valve replacement is associated with a 6% 30-day post-operative mortality rate, as well as 8% in-hospital mortality [21, 23, 35]. The 10-year survival for patients after a tricuspid valve replacement combined with left-sided heart valve surgery is 78%, but is only 41% in those with triple valve surgery. This is lower than the 10-year survival of patients undergoing aortic valve replacement (65%), mitral valve replacement (55%), and combined aortic and mitral valve replacement (55%) [37, 38].

There is limited data in the literature about surgical treatment in patients with TR secondary to PPM/ICD leads. Some of the literature recommends the removal of the original pacemaker leads and placement of an epicardial or transcoronary sinus lead in those patients who require TV replacement [44]. The disadvantage of epicardial leads has been the relatively high capture thresholds as resulting in frequent battery changes. An alternative is to surgically position the original lead between the sewing ring and the native annulus, or to place it inside the posteroseptal annulus with Lembert-type sutures [26]. Although promising, this technique makes it difficult to remove the lead transvenously in the future.

In regards to TV repair, ring annuloplasty is preferred over suture repair as it lends a lower incidence of recurrent and residual TR, fewer reoperations, and an improved survival for functional TR [43]. Some studies supporting this have included patients who have needed TV repair due to lead-related TR, with favorable outcomes. There is ongoing discussion and controversy over whether flexible versus rigid annuloplasty rings are superior. Some of the literature has shown that a rigid annuloplasty ring does not result in worsening of any residual TR following TV repair, while worsening was appreciated with flexible ring annuloplasty [23]. However, residual TR is still a significant problem which exists following TV repair regardless of the type of annuloplasty ring used. Additionally, there is an associated risk of increased postoperative conduction disturbances with the prosthetic ring compared to the suture annuloplasty technique [27].

Average time to surgery has been described in the literature as 72 months following device implantation [19]. This allows for the argument that lead-related TR is likely to occur over a longer period of time, although a small number of patients

have demonstrated acute decompensation within a shorter time frame. These patients fare well and show significant improvement postoperatively [19].

There is some recent data in the literature which outlines and demonstrates significant mortality associated with ≥3+ TR related to PPM's in particular [5]. Another study described that patients with device-related infection had an 18% all-cause mortality after 6 months of infection [45]. Overall, however, there is a paucity in data in the literature in order to come to a firm conclusion on these patients' potential benefits from surgery. Specifically, the long-term durability of surgical results remains unknown.

Percutaneous techniques: Percutaneous transcatheter techniques for TV repair or replacement are currently in their infancy, with feasibility studies in animal models and few in-human studies performed. Interest in this route remains high due to the significant risks of isolated TV surgery especially in the setting of reoperation following left-sided valve surgery [27]. The development of percutaneous techniques faces unique anatomic hurdles, such as the elliptical shape of the TV annulus, absence of calcium for good deployment and tethering, and proximity to important structures including the AV node/bundle of His, right coronary artery, and RVOT. Despite this, some important techniques and devices have been developed and have even shown procedural success and clinical improvement in small, early in-human studies.

Among these is the heterotopic caval transcatheter valve implantation. This procedure involves implantation of bioprosthetic valves at the level of the superior and inferior cavoatrial junctions. This aims to reduce the reflux of severe TR and improves heart failure symptoms but it does not affect the magnitude of the TR itself. Two prototypes have been developed: the self-expandable TricValve (P + F Products + Features Vertriebs GmbH, Vienna, Austria in cooperation with Braile Biomedica, São José do Rio Preto, Brazil) and the balloon-expandable Edwards valve (Edwards Lifesciences, Irvine, CA, USA). In addition, the self-expandable Edwards SAPIEN XT and SAPIEN 3 valves, which are primarily used for transcatheter aortic valve replacement (TAVR), have been used off-label for chronic severe TR, in which case a large self-expandable peripheral stent is implanted at the cavoatrial junction prior to implantation for proper "landing". Early studies have demonstrated successful valve implantation, improvement of heart failure symptoms, and no residual transvalvular or perivalvular leak with the above mentioned bioprostheses [27].

Transcatheter tricuscpid valve annuloplasty is another method being explored, with early studies using the Mitralign (Mitralign Inc., Tewksbury, MA, USA) and TriCinch (4Tech Cardio Ltd., Galway, Ireland) devices. The Mitralign device functions in the plication of the anterior and posterior aspects of the TV annulus, creating a functionally bicuspid valve as seen in the Kay surgical technique. The TriCinch device, currently being studied in the PREVENT registry, consists of a corkscrew anchor as well as a self-expandable stent which are connected by a Dacron band. The anchor is implanted into the TV annulus in the anterior and

posterior aspects, and the self-expandable stent is placed in the inferior vena cava. The modification and decrease of TV annular size is achieved by applying tension to the Dacron band.

Other methods include the FORMA device (Edwards Lifesciences) which aims to improve leaflet coaptation, the use of the MitraClip for TR, as well as transcatheter TV replacement. The FORMA device consists of a foam-filled balloon which acts as a spacer and is anchored at the RV apex. This has shown to be implanted successfully with improved outcomes in early studies [27]. Similarly, improved outcomes without any major complications have been demonstrated with the use of MitraClip on the TV for severe TR. In regards to transcatheter TV replacement, successful implantation have been demonstrated in animal models, however no in-human studies have been performed.

As of now, the above mentioned modalities have demonstrated favorable outcomes but these studies involve very small study populations. Studies involving human subjects and larger patient populations are needed to further elucidate the feasibility of these techniques.

Prevention (See Table 4.2)

Some suggestions have been made on ways to potentially prevent post-procedural TR, although the data behind this is overall limited. These involve various methods involving the type of leads used, procedural technique and location of lead implantation. Alternatives to the traditional intracardiac pacing devices have also been suggested as a possibility in some patients.

Lead type: Some authors have postulated that the lead type may be related to lead-related TR. One animal-based study found that expanded polytetrafluoroethylene-coated coils are easily extracted compared to backfilled with medical adhesive coils and uncoated coils, as these are commonly associated

Table 4.2 Prevention of lead related tricuspid regurgitation

Lead type	• Expanded polytetrafluoroethylene-coated coils • Polyurethane leads
Procedural technique	• Prolapsing technique
Lead location	• Septal or RVOT • Transvenous epicardial lead in coronary sinus • Intercommisural placement across tricuspid valve orifice
Imaging	• Intraoperative 3D TTE along with fluoroscopy
Alternative devices	• Leadless pacing • Subcutaneous cardioverter defibrillators
Surveillance	• Echocardiographic monitoring in select patients

with less fibrosis and, therefore, potentially less TR [46]. Whereas, Lin G et al. has reported higher prevalence of silicone versus polyurethane leads in those with lead-related TR [19].

Procedural technique: There are three methods to lead implantation during the procedure [47]. The first is the prolapsing technique, in which the lead is prolapsed across the tricuspid valve by first creating a loop in the right atrium, then subsequently advancing the loop with the inner stylet until the lead falls into the valve. The second is to cross the valve directly, aiming towards the target location with a shaped stylet. The third and final technique is also to cross the valve directly, however towards the RVOT with a curved stylet in place, then bringing the lead back until it is aimed towards the target location. Some experts suggest that the prolapsing technique is sometimes the preferred method, as the leads are not directly placed, decreasing risk of damage and trauma to the tricuspid apparatus. One disadvantage of the prolapsing technique, however, is the possibility of causing trauma to the structures surrounding the TV prior to advancement into the RV. Nevertheless, data still remains to be minimal on which method truly in fact causes the least damage to the TV apparatus.

Lead location: location of the lead itself may play a factor in the development or worsening of TR. Expert opinion explains that apically placed leads have the potential of tethering to the posterior TV leaflet more than septally placed leads. Recent literature has shown a higher incidence of lead-related TR with leads placed apically versus those placed in the RVOT [7]. Apical pacing can contribute to RV dyssynchrony and alterations in RV geometry. This may explain some literature that has shown a higher incidence of lead-related TR following ICD placement versus PPM as ICD leads must be placed apically. However, whether there is a higher incidence of TR with ICDs versus PPMs remains controversial.

Case reports in the literature have demonstrated the possibility and feasibility of transvenous epicardial leads in the coronary sinus or middle cardiac vein rather than intracardiac lead placement for permanent pacing [17, 48]. These cases were primarily those patients with whom a lead could not be placed across the TV, such as those with a history of TV surgery. This method is already accepted as an indication for cardiac resynchronization therapy; extension of the indication to permanent pacing may provide an alternate option to avoid trauma to the TV with intracardiac leads, and therefore, prevent lead-related TR.

As mentioned previously, 3D TTE studies have demonstrated that much of lead-related TR is associated with leads interfering with the posterior and/or septal TV leaflets [36]. Those without lead-related TR were found to have the lead placed intercommisurally or in the center of the TV orifice. This information could be potentially used for prevention of TR following PPM or ICD placement. It is suggested that intra-operative 3D TTE along with traditional fluoroscopy may be beneficial to adequately place the leads in a more optimal location and, therefore, potentially prevent post-procedural TR. However, this has not been studied head-to-head with purely intra-operative fluoroscopy, and the possibility of lead displacement following the procedure has not been assessed. Given the large

number of device implants, intra-operative echocardiography for lead placement would pose a significant logistical and financial challenge.

Alternative devices: Newer technologies can help in reducing lead-related TR. Leadless pacing has been possible with a self-contained encapsulated unit which can be attached to the endocardium of the right ventricle. Feasibility trials, such as the LEADLESS, LEADLESS II, and Micra Transcatheter Pacing Study have achieved positive results [17, 49–51]. The Micra Transcatheter Pacing Study in particular reported a 96% Kaplan-Meier estimate of freedom from device-related adverse events at 6 months [49]. One of these device-related adverse events was cardiac failure, which occurred in 0.9% of the patients, however device-related TR was not specifically reported.

Subcutaneous cardioverter defibrillators are also another alternative which do not involve leads that would cross the TV [17]. The generator is implanted subcutaneously in the lateral chest wall, the lead running also subcutaneously across the left side of the chest and along the parasternal area, thus avoiding any intravascular intervention. The major limitation of subcutaneous defibrillators is the inability to pace. In addition, the patient population in which subcutaneous defibrillators may be used is limited to those with appropriate recoding of cardiac signals according to the orientation of the device in the chest.

Surveillance: Although there are no data regarding the specific benefits and clinical effect of surveillance, some patients may benefit from close echocardiographic monitoring. These include those who develop signs and symptoms of new-onset right-sided heart failure, have pre-existing TR, or those who have more than one apical lead [1].

Summary

Device-related TR is not an uncommon complication of PPM or ICD placement. Although a previously unrecognized entity, it is now better understood and recognized with emerging literature. TR following PPM or ICD placement is most often secondary to either mechanical or physiological mechanisms which involve damage or deformity to the TV apparatus, leads, and/or cardiac structure and function. Clinically significant lead-related TR involves the signs and symptoms of right-sided heart failure, which is further exemplified and evidenced by 2D TTE and color Doppler flow mapping. Management typically involves medical management as well as percutaneous extraction of the offending leads. Prospective methods to prevent or reduce the incidence of this complication include improved imaging modalities intraoperatively, procedural techniques, particular lead placement, and the use of the cardiac venous system. In the future, devices which do not involve leads may be another alternative. As of now, the literature is continuing to emerge, while case studies and retrospective studies predominate and prospective studies are just beginning to surface. Further studies are needed to solidify our understanding of lead-related TR incidence/risk, prognosis and mortality, proper management, and possible prevention.

Review Questions

28. Which of the following mechanisms of mechanical tricuspid regurgitation in the setting of permanent pacemaker or implantable cardioverter-defibrillator leads is **not** correct?

    (a) Valve obstruction caused by lead placed in between leaflets.
    (b) Lead entrapment in the tricuspid valve apparatus.
    (c) Annular dilatation.
    (d) None of the above.

29. Which of the following statements about mechanical tricuspid regurgitation in the setting of permanent pacemaker or implantable cardioverter-defibrillator leads is **not** correct?

    (a) A prevalence reported to be between 25 and 29%.
    (b) It causes worsening of pre-existing TR in 10 to 25%.
    (c) TR is more often seen in apically implanted pacemaker leads.
    (d) TR is caused by lead perforation in 17%.

30. Which of the following are potential risk factors for mechanical tricuspid regurgitation in the setting of permanent pacemaker or implantable cardioverter-defibrillator leads?

    (a) Advanced age
    (b) Obesity
    (c) Preexisting atrial fibrillation
    (d) All of the above

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