Cath Lab Digest

A product, news & clinical update for the cardiac catheterization laboratory specialist

CCL BASED PROCEDURES:		FY23	FY24	FY25	2YRDIFF
Diagnostic Catherization:					
286	CIRC DIS EXC AMI, W CATH W MCC	$14,549	$15,093	$15,752	4.4%
287	CIRC DIS EXC AMI, W CATH W/O MCC	$7,547	$7,573	$7,755	2.4%
Coronary Intervention:					
321	PCI W/ INTRALUMINAL DEVICE W/MCC OR 4+ ARTERIES	-	$20,127	$20,260	0.7%
322	PCI W/ INTRALUMINAL DEVICE W/O MCC	-	$12,767	$12,875	0.9%
323	CORONARY INTRAVASCULAR LITHOTRIPSY W/ INTRALUMINAL DEVICE W/	-	$28,987	$30,313	4.6%
324	CORONARY INTRAVASCULAR LITHOTRIPSY W/O INTRALUMINAL DEVICE	-	$20,785	$22,739	9.4%
325	CORONARY INTRAVASCULAR LITHOTRIPSY W/ INTRALUMINAL DEVICE W/O	-	$18,514	$20,369	10.0%
250	PCI W/O STENT W MCC	$16,598	$16,459	$16,460	0.0%
251	PCI W/O STENT W/O MCC	$11,149	$11,111	$11,120	0.1%
Peripheral Vascular:					
252	OTHER VASC PROCEDURES W MCC	$22,933	$23,482	$24,413	4.0%
253	OTHER VASC PROCEDURES W CC	$18,342	$17,862	$18,169	1.7%
254	OTHER VASC PROCEDURES W/O CC/MCC	$12,543	$12,148	$12,450	2.5%
278	ULTRASOUND ACCELERATED AND OTHER THROMBOLYSIS OF PERIPHERAL	-	$31,230	$35,606	14.0%
279	ULTRASOUND ACCELERATED AND OTHER THROMBOLYSIS OF PERIPHERAL	-	$22,409	$22,804	1.8%

REIMBURSEMENT UPDATE

CMS 2025 Updates – Inpatient and Outpatient

Jackie Shepard, Business Consultant, Corazon

The Centers for Medicare & Medicaid Services (CMS) recently released its 2025 updates, introducing significant changes to the Medicare payment systems that will impact healthcare providers nationwide. These updates, which include adjustments to the Inpatient Prospective Payment System (IPPS), Outpatient Prospective Payment System (OPPS), Ambulatory Surgery Centers (ASCs), and Physician Fee Schedule (PFS), are designed to address current challenges and trends within the healthcare landscape. As providers navigate these updates, understanding the detailed revisions and their implications will be essential for strategic planning and operational efficiency in the year ahead.

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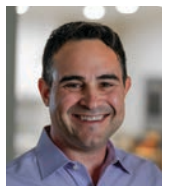
In This Issue

STRUCTURAL HEART & EP

Lead Management in Patients With Cardiac Implantable Devices and Tricuspid Regurgitation

CLD talks with Electrophysiologist Mark D. Metzl, MD, and Structural Interventionalist Mark J. Ricciardi, MD.



How often do cardiac implantable electronic device (CIED) leads cause significant tricuspid regurgitation (TR) and what are the causes?

Dr. Ricciardi (Structural Interventionalist): While there are cases where the lead can impinge on the valve leaflets and cause tricuspid valve dysfunction, it is not very common. We see many patients in valve clinic with significant TR and a lead in place, where the leads themselves are not causal; they just happen to be present. A review summarizing the frequency of occurrence or worsening of TR following CIED implantation found that its occurrence ranged from 7% to 45% of studied patients.[1]

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MEETING UPDATE

Fall Meeting Roundup: Highlighted Research From the Latest Interventional Symposia

From the 2024 European Society of Cardiology (ESC), Transcatheter Cardiovascular Therapeutics (TCT), and Vascular InterVentional Advances (VIVA) meetings

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Lead Management in Patients With Cardiac Implantable Devices and Tricuspid Regurgitation

CLD talks with Electrophysiologist Mark D. Metzl, MD, and Structural Interventionalist Mark J. Ricciardi, MD.

Continued from cover

Dr. Metzl (Electrophysiologist): A few years ago, there was a push to recognize CIED lead-related TR, which became quite a hot topic. Certainly, lead-related TR exists. A lead can perforate a leaflet and cause TR. Leads can interact with a part of the tricuspid valve apparatus, and prevent leaflets from closing or prop them open. The mechanisms are important, because when we talk about this issue, there is an almost gut reaction on the part of an electrophysiologist that if the lead is implicated as the etiology of the TR, then removing it will fix the TR. Sometimes that is true. A study of 208 consecutive patients by Park et al found that patients with severe TR experienced up to a 37% reduction in severe TR post transvenous lead extraction.[2] However, there are also many patients with severe TR in which the lead is an innocent bystander and there are many cases in which severe TR can be fixed by our structural colleagues with the leads in place. We have to approach these patients from multiple perspectives, which is why this topic is so important for both electrophysiologists and for our structural colleagues. As an electrophysiologist, I am an expert in implanting and removing these leads, but I am not an expert in fixing tricuspid valves. We need a team approach in order to treat these patients.

Can you describe how leads are placed and might interact with the tricuspid valve?

Dr. Metzl: The generator is typically placed in the upper chest and the venous system is accessed at this location. Leads are placed through access site, through the central venous system down through the right atrium, through the tricuspid valve, and into the ventricular myocardium. At the tip of the lead, there is a fixation mechanism, either a tine or a little screw to fixate the tip. The lead is also secured at the venous access site with a suture sleeve. When leads are placed through the tricuspid valve, they can perforate a leaflet, and lead slack resting on the endocardial surface can also pin a leaflet open. Over time, leads become more fixed as fibrotic tissue forms and this tissue can also interfere with the tricuspid valve apparatus, preventing the leaflet or the annulus from moving in its proper setting.

Why might tricuspid regurgitation occur in patients who end up with a CIED?

Dr. Ricciardi: Of the patients who are referred for tricuspid valve evaluation and repair/replacement, the vast majority will have a preexisting electrical problem; namely, atrial fibrillation (AF). And when the AF is associated with the development of bradyarrhythmia, treatment often includes the placement of a pacemaker. AF can cause the upper chambers of the heart to dilate. When the upper right atrial chamber dilates, the tricuspid valve annulus dilates — the annulus is the door frame within which those leaflets sit. The leaflets become stretched apart from each other so they don't coapt or meet at the middle, and they leak. In a normal heart, the annulus of the tricuspid valve is shaped like a horse saddle, and it has upper and lower scallop-shaped areas. With longstanding AF, that scalloped or saddle- shaped valve starts to flatten, which is called annular flattening. As it flattens, the annulus also dilates and pulls those leaflets apart, causing malcoaptation. There are three main components of the tricuspid valve: the leaflets, the annulus, and the subannular apparatus (the chordae and papillary muscles that attach the leaflets to the ventricle). A distortion of any of those three things can cause TR, but the annular flattening and enlargement happens in a lot of people with secondary TR. In secondary TR, leaflets are not the primary problem. In fact, these patients' leaflets are fairly normal. It is the structure around the leaflets that is causing problems. A much smaller percentage of people will have a primary leaflet problem causing the TR.

Dr. Metzl: Yes, and regarding the example Dr. Ricciardi just gave showing that it is common for patients to develop TR as a result of AF, in longstanding persistent or permanent AF, patients will often burn out their atrioventricular (AV) nodes and need pacemakers for bradyarrhythmias that are a result of the natural history of the disease. We see these patients in later years of life and as a result of their longstanding arrhythmia, they both need pacing and have developed severe TR.

Heart failure is another patient population in whom we are often putting CIEDs with leads, so we are seeing both heart failure and AF patient populations.

Dr. Ricciardi: Historically, the number-one cause for TR is left-sided heart disease and that could include left ventricular dysfunction, cardiomyopathies, mitral regurgitation, or aortic stenosis. In some ways, the tricuspid valve is "sympathetic" to what goes on in the left side of the heart, directly impacting the right side of the heart, leading to chamber dilation, which could lead to TR. Sprinkle in some atrial fibrillation and it is a perfect recipe for TR, which is why we see so many patients with this problem.

How can imaging help identify the interaction between leads and the tricuspid valve?

Dr. Metzl: Imaging is a key component. It is extremely important to get a transesophageal echocardiogram (TEE) and have an expert structural imager look at the leaflets, the interaction between the leads and the leaflets, and determine the mechanism of the TR. In our institution, specialized cardiac structural imagers do these studies. In some cases, the interaction of the leads and the tricuspid valve cannot be well-imaged from faraway areas like through the chest (transthoracic echocardiography) or esophagus (TEE). Recently, we have been using 4D intracardiac echocardiography (ICE) in some of our EP and structural procedures, which has the advantage of imaging this lead-valve interaction from an up-close viewpoint with incredible detail.

Dr. Ricciardi: A simple transthoracic echo is a great tool to measure the degree of TR. If there is a lead across that valve, it will be visible. To determine whether the lead is causal for the TR, a TEE can determine the mechanism of the TR. Sometimes we will use ancillary testing like computed tomography, but mostly imaging is echo-based — transthoracic and then a TEE.

At present, this is a complex field that requires a ton of thought beforehand and great deal of discussion amongst the two disciplines, EP and interventional. It is a drawn-out process. First, the problem is identified, usually with a transthoracic echo. Irrespective of the etiology, we often will put these patients on medical therapy and treat whatever cause may be leading to the TR, such as left heart disease. Maybe they have concomitant mitral regurgitation or left ventricular dysfunction. We treat that, then reassess the tricuspid valve for improvement. If the TR hasn't improved, a TEE shows us the mechanisms and helps determine what therapy we might offer. The FDA recently approved two transcatheter treatments for TR repair and replacement. With these approvals, we expect this field will pick up a lot of steam; hence the need for conversations between the electrophysiologist and the valvular cardiologist.

Dr. Metzl: Having a TEE is extremely valuable. Treatment isn't cookie cutter and there isn't one option. To have time, thought, and conversation as a team is essential. From an EP perspective, we have several options that can be used to pace the ventricle without traversing the tricuspid valve. We can place traditional leads in the coronary sinus to pace the ventricle, and now we have single and dual chamber leadless pacemakers that can provide arteriovenous (AV) synchrony without

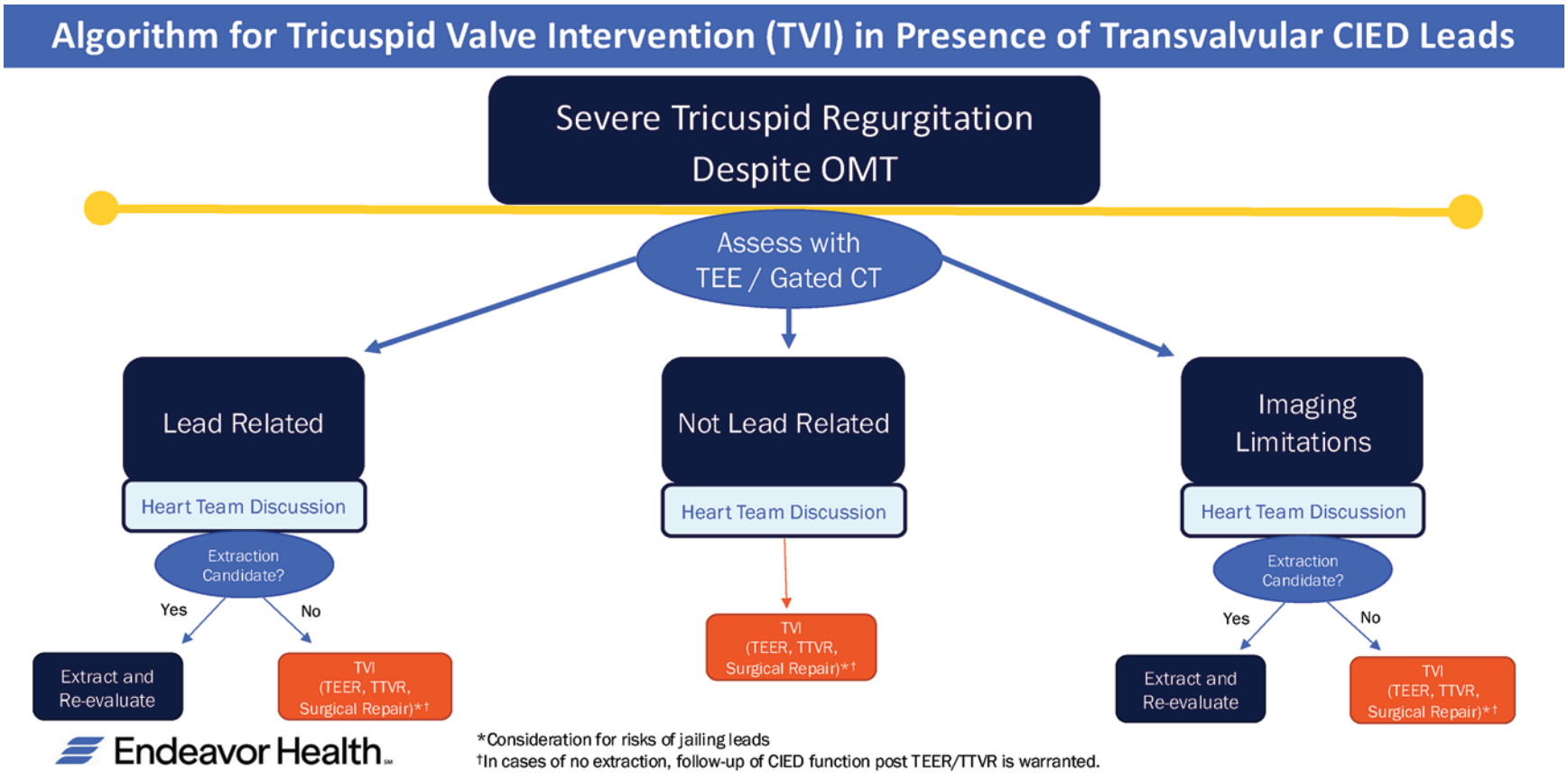

Figure. A simplified look at the decision pathways for patients on optimal medical therapy with severe symptomatic tricuspid regurgitation and a cardiac implantable device. *CIED, cardiac implantable electronic device; OMT, optical medical therapy; CT, computed tomography; TEE, transesophageal electrocardiography; TVI, tricuspid valve intervention; TEER, transcatheter edge-to-edge repair; TTVR, transcatheter tricuspid valve replacement.*

any leads. We have subcutaneous and extravascular defibrillators that have the ability to provide life-saving therapies without any hardware in the heart. Understanding the indication for the patient's device and the alternative options is very important.

Extraction of a pacemaker or defibrillator is also nuanced. Physicians that perform extraction constantly weigh the risks and benefits of removing leads in patients. Lead extraction is extremely safe overall. The complication rate is about 1%, with mortality reported at about 0.3%,[3] which is not much different than standard EP procedures or even percutaneous coronary intervention. But the decision needs to be all about the patient and their characteristics, and the characteristics of the hardware in place. A 40-year-old with a 4-year-old system is a different patient than a 90-year-old with a 20-year-old system. There are certain pacemaker and defibrillator leads that are easier and less challenging to remove than others, and there are patient characteristics that make patients higher and lower risk. The key is having someone involved that understands this perspective, and the accompanying risks and benefits. It also depends on the pacing indication. We've had patients whose pacing indication, when we look back, doesn't actually require pacing the ventricle, meaning we are removing a wire that is traversing the tricuspid valve and potentially does not need to be replaced. We've had patients where their leads are low risk and the patients themselves are low risk, and we've had the opposite. EP involvement is valuable for this procedure. Having someone knowledgeable about lead extraction available to talk with the structural team is something we didn't need before, but now we do, because there are so many ways to tackle these problems.

On the structural side, there are options to fix severe TR that are under-recognized by the EP team.

Dr. Ricciardi: If you imagine a decision tree for a patient with TR and a pacer lead, the first question would be, is the lead causing the TR or could it be impacting our ability to fix the valve? If the answer is no, then typically we leave that lead alone and repair/replace the valve. For these leads to be deemed truly "innocent", they are neither causing the TR nor interfering with the imaging needed to treat the TR. This approach has been demonstrated in the repair and replacement trials (TRILUMINATE and TRISCEND), where approximately one in three patients had a pacer lead and underwent successful tricuspid valve implantation or repair despite the presence of the lead.[4,5] For patients in whom the lead may be causing the TR or impacting our ability to repair/replace the valve, we need to think seriously about lead removal. Lead removal can thereby reset the table for a possible intervention. Rarely, however, does that lead removal in and of itself improve the TR, unless the lead was just recently placed. It is more common that lead removal simply allows us to work on the valve.

Dr. Metzl: There are some data showing that after lead removal in patients with severe TR, the TR improves into the moderate range about 37% of the time.[2] Another small study of mild to moderate TR patients showed no significant TR post lead extraction several months later, confirmed with transthoracic echocardiography (TTE).[3] That improvement may be enough for some patients. To go from severe to mild, however, is rare, and happens in under 5% of cases.[2] The problem these patients face is that over time, the annulus dilates and the right ventricle fails, and even if the lead originally caused the TR, removal may not be enough to fix the TR to a point where it is clinically relevant.

Dr. Ricciardi: The problem with looking at that study in isolation is that it included preselected patients, those where the team thought removing the lead would probably bring an improvement in their TR. There are many, many TR cases out there where you can remove the lead — but don't expect that the TR will automatically improve. That is why the heart team, including the electrophysiologist and the interventionist, is so important. If we were to assume that all leads are innocent or that all leads are causative, then we would be either keeping in too many leads or removing too many leads.

Dr. Metzl: That is a great point. There is a misperception that if we remove the lead, the TR will get better, or better enough. There is also a misperception that every patient dies with an extraction, whereas the mortality is only 0.3%.[4] These areas of expertise, these silos, need to be broken down,

which is why the heart team approach to these patients is so valuable. I'll come at these patients with one perspective. Dr. Ricciardi and his team have another. When we talk about the patients, we learn so much from each other about the disease processes that are contributing, how we each can help, and the benefit and risk of the things that we do. For example, when we started these conversations, I had no idea that you could successfully clip a tricuspid valve with a lead in place. I assumed that patients would need to have their leads removed every time, because how could you possibly work around this hardware? It turns out that a third of the patients in the TRILUMINATE and TRISCEND trials had leads in place and experienced successful repair/replacement procedures.[5,6] My perception of structural cardiology was different from reality, and I am sure that Dr. Ricciardi and his team felt the same way about the things that we in EP have to offer. It is only when we have these conversations that we learn the truth.

Is it safe to "jail" leads with transcatheter tricuspid valve replacement devices?

Dr. Riccardi: You can jail the lead if you put it in an annular tricuspid valve replacement apparatus. You push the lead to the side and have it lean against the annulus, and usually it is fine. When you repair the valve, you are repairing around the leads. The lead becomes isolated and often is not a problem. But even if you decide that the lead doesn't need to be removed because you can jail it or work around it, the next question becomes, what if we take a lead that is somewhat tethered and pull it away from its contact point, or what if the lead fractures as a result of our manipulations? Suddenly that patient may no longer have a functioning pacemaker. If they are pacer dependent, that is a disaster, because you will have just repaired or replaced the valve. Now you have to tell your EP colleagues, "I've ruined your lead. Can you put in a new one?" But where are they going to put it? Are they going to put it through the new valve I just worked on? Then there are all the other leadless pacing options. There are many things to consider. As an aside, we will be investigating the use of heterotopic (non-annular) bicaval valve implants. But this may be outside the scope of this conversation.

Dr. Metzl: We published a case report of a lead that was jailed and the patient went on to have a pocket infection, which is another concern.[7] Now you have jailed the lead and even though it seems to be or is functional, we put the person at risk for infection for the rest of their life.

Dr. Ricciardi: Although it is rare to see infections involving the pacer leads or pockets, it does often require removal of the lead.[8] If you jail a lead behind a valve, extracting the lead is near impossible and puts the patient in a dire situation. Infection is rare, but were it to happen, you have really pinned the patient into a corner.

Dr. Metzl: Device infection is a lifetime risk of perhaps 4%, which increases with each generator replacement.[9]

You are emphasizing the importance of cross-disciplinary conversations and the involvement of a heart team approach. Who should be involved besides electrophysiology (EP), structural heart, and imaging?

Dr. Ricciardi: The cardiothoracic surgeon has been involved in valvular intervention, because in the pre-transcatheter intervention era, all things valve were managed by cardiothoracic surgeons. The tricuspid valve has never really fallen into that realm, however, because isolated tricuspid valve surgery is the least performed cardiac surgery and has a high mortality rate. However, there are still a decent number of people who have tricuspid valve operations and the cardiac surgeon's perspective can be very helpful. For example, when patients undergo open-chest valve surgeries that include treatment of the tricuspid valve, there are some inventive things that the surgeon can do at the time of the open procedure that could solve some of these problems.

By definition, people who have symptomatic TR also have heart failure — their heart is failing because of valve regurgitation — so heart failure specialists often help us manage these patients, especially when it comes to optimizing their medical therapy before and after surgery.

In the absence of guidelines, what are you considering as you move forward in treating these patients?

Dr. Metzl: Earlier this year, the Heart Valve Collaboratory and Heart Rhythm Society published a review that provides an excellent place to start learning about these complex patients and how we can best help them.[10] It is an emerging field. We have all been learning together over the last few years and need to continue to share that knowledge with others. Right now, we work on a case-by-case basis. We have put together an algorithm (Figure), but it is not a simple recipe.

Dr. Ricciardi: You can't be terribly prescriptive. At the margins is probably where we want to make the most impact, and by that, I mean let's not insist that our EP colleagues pull out leads when we know it will not help, but might avulse or damage the tricuspid valve. Let's also not assume that the lead is innocent. Those 80% of the patients in the middle are the ones that require a lot of imaging, thought, and back-and-forth discussion.

If hospitals don't offer lead extraction, what should they do?

Dr. Ricciardi: The short answer is, send it to someone who does have lead extraction. The multisociety valvular heart disease guidelines, for instance, say that patients should be managed in a center of excellence or a valve heart center. Patients with a CIED and TR need this as well. Hospitals may offer EP and do extractions, but perhaps don't have anyone who handles the tricuspid valve. Vice versa, hospitals may have a good valve center, but are without EPs comfortable with lead extraction. You need a center that does and thinks about both these things on a daily basis.

Dr. Metzl: If a center plans to do tricuspid valves and doesn't have an extraction program, then it would be valuable to develop a closer relationship with an extraction program. I would imagine that most centers doing implants already have a relationship in order to take care of patients with class one indications for extractions. Involving EP from that center and building bridges will be very important as this field emerges in structural cardiology.

Can you share more about the Cardiovascular Institute at the Endeavor Health?

Dr. Metzl: Endeavor Health is a nine-hospital system situated in the Chicago region. We are extremely fortunate to have the recently built Endeavor Heath Cardiovascular Institute at Glenbrook Hospital in Glenview, Illinois. It is exciting not only because the Cardiovascular Institute is truly state-of-the-art with nine EP and cath labs, but because it provides us with a physical location where our structural team, cardiac imagers, and EP team can work together. These types of conversations, which before were more fragmented, are much easier to have in a single location. We and our patients are better for it.

Dr. Ricciardi: It is a great place to work because there is collaboration that just is born out of simple geography. It is good for patients, too, because they get everything they need in one location. Our patients are from all over the Chicagoland area. We are a tightknit group in general, but by being all under one roof, the new center enhances our collaboration, which is a necessity for these patients. ■

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References are available online with the article.

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Online Only

References

1. Addetia K, Harb SC, Hahn RT, Kapadia S, Lang RM. Cardiac Implantable Electronic Device Lead-Induced Tricuspid Regurgitation. *JACC Cardiovasc Imaging.* 2019 Apr; 12(4): 622-636. doi:10.1016/j.jcmg.2018.09.028
2. Park SJ, Gentry JL 3rd, Varma N, et al. Transvenous extraction of pacemaker and defibrillator leads and the risk of tricuspid valve regurgitation. *JACC Clin Electrophysiol.* 2018 Nov; 4(11): 1421-1428. doi:10.1016/j.jacep.2018.07.011
3. Arora Y, D'Angelo L, Carrillo RG. A novel protocol for tricuspid regurgitation reversal with transvenous lead extraction. *JACC*. 2022 Mar; 79 (9_Supplement): 102. https://www.jacc.org/doi/10.1016/S0735-1097%2822%2901093-2
4. Pokorney SD, Zepel L, Greiner MA, et al. Lead extraction and mortality among patients with cardiac implanted electronic device infection. *JAMA Cardiol.* 2023 Dec 1; 8(12): 1165-1173. doi:10.1001/jamacardio.2023.3379
5. Nickenig G, Lurz P, Sorajja P, et al; TRILUMINATE Investigators. Percutaneous Edge-to-Edge Repair for Tricuspid Regurgitation: 3-Year Outcomes From the TRILUMINATE Study. *JACC Cardiovasc Interv.* 2024 Jul 26:S1936-8798(24)00851-3. doi: 10.1016/j.jcin.2024.05.036
6. Kodali S, Hahn RT, Makkar R, et al. Transfemoral tricuspid valve replacement and one-year outcomes: the TRISCEND study. *Eur Heart J.* 2023 Dec 7; 44(46): 4862-4873. doi:10.1093/eurheartj/ehad667
7. Paz Rios LH, Alsaad AA, Guerrero M, Metzl MD. Tricuspid valve-in-valve jailing right ventricular lead is not free of risk. *Catheter Cardiovasc Interv*. 2020 Dec; 96(7): E758-E760. doi:10.1002/ccd.28622
8. Kusumoto FM, Schoenfeld MH, Wilkoff BL, et al. 2017 HRS expert consensus statement on cardiovascular implantable electronic device lead management and extraction. *Heart Rhythm*. 2017 Dec;14(12):e503-e551. doi:10.1016/j.hrthm.2017.09.001
9. Arana-Rueda E, Pedrote A, Frutos-López M, et al. Repeated procedures at the generator pocket are a determinant of implantable cardioverter-defibrillator infection. *Clin Cardiol.* 2017 Oct; 40(10): 892-898. doi:10.1002/clc.22743
10. Hahn RT, Wilkoff BL, Kodali S, et al; Heart Valve Collaboratory and Heart R hythm Society. Managing implanted cardiac electronic devices in patients with severe tricuspid regurgitation: JACC State-of-the-Art Review. *J Am Coll Cardiol.* 2024 May 21; 83(20): 2002-2014. doi:10.1016/j.jacc.2024.02.045