

Tricuspid Valve A Forbidden Fruit?

From overlooked disease to timely care

Audience: RN/APRN | **Contact hrs:** 1.75 | **Pharmacology hrs:** 0.0

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Pharmacology hours are included in the total contact hours.

The valve that should not be forgotten

A patient returns for the third time with swollen legs, abdominal fullness, and declining exercise tolerance. The left ventricular ejection fraction is preserved, so the symptoms are attributed to age, kidney disease, or an inadequate diuretic dose. Yet the jugular veins are elevated, the liver is congested, and severe tricuspid regurgitation has been present on several echocardiograms. The central problem is not simply excess fluid: a damaged or distorted valve apparatus is allowing systolic flow back into the systemic venous circulation (Mukherjee et al., 2025; Praz et al., 2025).

The phrase “forbidden fruit” captures an older reluctance to intervene on the tricuspid valve. That reluctance was reinforced by technically challenging anatomy, late referrals, and poor outcomes in patients who already had advanced right ventricular and end-organ dysfunction. Contemporary care replaces automatic avoidance with earlier recognition, integrated imaging, individualized risk assessment, and a choice among medical, surgical, and transcatheter approaches. An intervention is not appropriate for every patient, but severe symptomatic disease deserves a deliberate evaluation rather than indefinite escalation of diuretics (Praz et al., 2025).

Purpose, audience, and learning outcomes

This activity prepares registered nurses and advanced practice registered nurses to connect tricuspid anatomy and hemodynamics with assessment, referral, treatment selection, and safe longitudinal care. The principal management discussion concerns adults; congenital conditions are included to explain anatomy and the need for pediatric or adult congenital heart disease expertise. RNs assess, administer authorized treatment, monitor, educate, and escalate concerns. Diagnostic and prescribing decisions require appropriate professional authority, privileges, and local protocols. Device

authorizations identified as U.S. approvals do not establish authorization elsewhere.

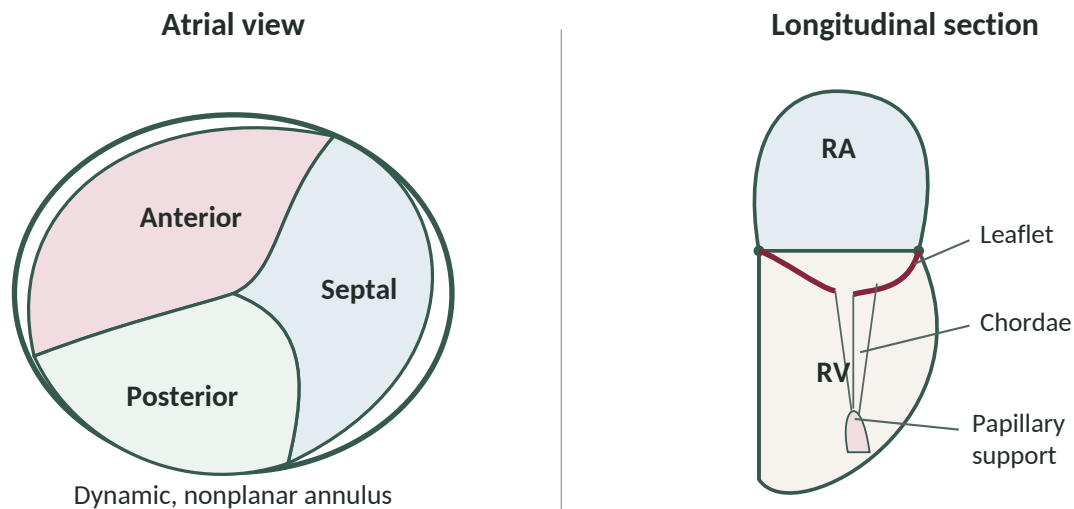
After completing the activity, learners should be able to: explain how valve, atrial, and ventricular abnormalities produce tricuspid dysfunction; distinguish primary, atrial secondary, ventricular secondary, and device-related disease; interpret a multiparametric imaging report without relying on one measurement; recognize congenital, infectious, neoplastic, and systemic causes that change the care pathway; compare the rationale and limitations of surgery, transcatheter repair, replacement, and medical treatment; and prioritize nursing surveillance, medication reconciliation, patient teaching, and timely reassessment. The five worked cases are fictional teaching examples.

Anatomy, development, and right-heart physiology

The tricuspid valve connects the right atrium to the morphological right ventricle. “Morphological” matters: in congenital malposition, the right ventricle is identified by its internal structure rather than by which side of the chest it occupies. Its inlet, trabeculated apex, moderator band, and outflow region form a complex chamber that cannot be described adequately by a single diameter or a left-ventricular geometric model. The valve is part of this integrated chamber, not an isolated three-flap structure (Dahou et al., 2019; Mukherjee et al., 2025).

The familiar anterior, posterior or inferior, and septal leaflet names are useful, but real valves vary in leaflet number, scallops, commissures, and chordal attachments. The annulus is dynamic and nonplanar, with a relatively constrained septal segment and a more distensible free-wall component. Chordae connect the leaflets to variable papillary muscles and, particularly along the septum, directly to ventricular myocardium. A

One valve, an integrated apparatus



RA = right atrium; RV = right ventricle. Three principal leaflet names are useful, but leaflet, chordal, and papillary muscle anatomy varies.

Figure 1. The tricuspid valve works with the annulus, chordae, papillary supports, and right-heart myocardium. Original educational schematic, not a patient image; anatomy is simplified and not to scale. Content: Dahou et al. (2019); Mukherjee et al. (2025).

rigid claim that every patient has exactly three leaflets and two papillary muscles can mislead procedural planning. Figure 1 relates these components to the sites of coaptation and regurgitation (Dahou et al., 2019).

Development involves atrioventricular cushion tissue, myocardial remodeling, and separation or delamination of leaflet tissue from the ventricular wall. Incomplete delamination helps explain Ebstein anomaly. Absent development of a patent right atrioventricular connection produces tricuspid atresia. Developmental abnormalities often involve more than the valve: associated atrial or ventricular communications, ventricular size, outflow obstruction, and great-artery connections determine how blood reaches the pulmonary and systemic circulations (Shah et al., 2016).

During diastole, right atrial pressure exceeds right ventricular pressure and the valve opens. During systole, pressure reversal closes the leaflets, while the chordal apparatus prevents excessive prolapse. Competence requires adequate leaflet tissue, appropriate annular size, suitable chordal geometry, and coordinated ventricular contraction. Annular enlargement or apical tethering can prevent normally formed leaflets from meeting. In stenosis, the obstacle instead lies in diastolic

inflow, raising right atrial pressure and limiting ventricular filling (Dahou et al., 2019; Shah et al., 2016).

The right ventricle normally ejects into a relatively low-pressure pulmonary circulation. Regurgitation provides an additional low-impedance route into the atrium. A ventricle may therefore appear to eject well while delivering an inadequate forward stroke volume. Eliminating a large regurgitant orifice changes loading conditions abruptly; apparent contraction before treatment does not guarantee adequate reserve afterward. This helps explain why severe pulmonary vascular disease and advanced right ventricular dysfunction complicate intervention (Mukherjee et al., 2025; Praz et al., 2025).

The septal annulus lies close to the atrioventricular conduction system, while the right coronary artery courses near the atrioventricular groove. These relationships explain why annuloplasty and replacement can cause conduction injury or coronary compromise. They also explain why a new rhythm disturbance, ischemic change, or hemodynamic deterioration after tricuspid treatment must be assessed as a possible procedural complication rather than dismissed as a

routine postoperative finding (Dahou et al., 2019; Praz et al., 2025).

Mechanisms, progression, and clinical recognition

Primary regurgitation begins with an abnormal leaflet or supporting apparatus. Examples include prolapse or flail tissue, rheumatic restriction, infective destruction, carcinoid fibrosis, congenital dysplasia, and traumatic injury. Secondary regurgitation begins mainly with altered atrial, annular, or ventricular geometry. Mixed disease is common: a lead may interfere with a leaflet in a patient who also has atrial fibrillation, annular enlargement, and pulmonary hypertension. Naming the mechanism is more informative than labeling every nonrheumatic lesion “functional.” (Andreas et al., 2024; Praz et al., 2025).

Atrial secondary regurgitation is characterized by prominent right atrial and annular enlargement, often in association with longstanding atrial fibrillation or heart failure with preserved ejection fraction. Ventricular secondary regurgitation involves right ventricular enlargement, leaflet tethering, or both, commonly in the setting of left-heart disease or pulmonary hypertension. These are useful phenotypes, not mutually exclusive diagnoses. A patient may evolve from predominantly atrial disease to significant ventricular remodeling. Figure 2 shows why similar regurgitant jets can arise from different structural problems (Mukherjee et al., 2025).

Regurgitant volume increases atrial and ventricular volume loading. Progressive annular enlargement and papillary muscle displacement worsen leaflet malcoaptation, which further increases regurgitation. The clinical consequence is a self-reinforcing cycle of congestion, reduced effective forward output, and organ dysfunction. Correcting a left-sided lesion can improve right-sided loading, but established tricuspid remodeling does not reliably disappear afterward. Persistent or late regurgitation after mitral surgery is therefore a reason for surveillance, not proof that the original operation failed (Gammie et al., 2022; Praz et al., 2025).

Systemic venous congestion may present as dependent edema, ascites, abdominal discomfort, early satiety, hepatic enlargement, or worsening kidney function. Fatigue and exercise intolerance may reflect reduced forward output as well as fluid accumulation. Dyspnea can coexist with lung disease or left-sided heart failure. The absence of pulmonary crackles does not exclude serious right-sided disease. Ask what the patient can no

longer do, how quickly symptoms have changed, and whether apparently successful diuresis is followed by rapid recurrence (Mukherjee et al., 2025; Praz et al., 2025).

Assessment includes blood pressure, heart rate and rhythm, oxygen saturation, perfusion, jugular venous pressure, edema, weight trajectory, urine output, and abdominal findings. Large systolic jugular venous waves or hepatic pulsation support regurgitation, but bedside signs vary with body habitus, rhythm, and severity. A soft murmur is not reassuring in a patient with advanced low-output disease. Laboratory review should include renal function, electrolytes, blood count, and hepatic measures when congestion or intervention is being evaluated (Dreyfus et al., 2022; Mukherjee et al., 2025).

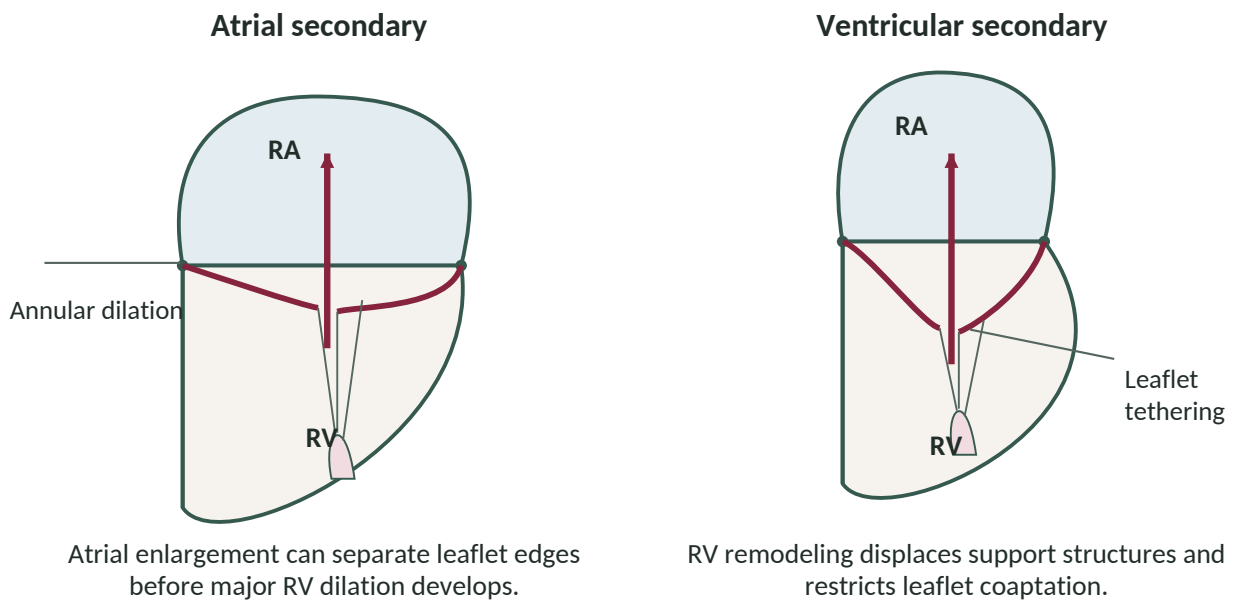
CASE 1 • Congestion with a preserved left ventricular ejection fraction

Maria, 78, has persistent atrial fibrillation and two recent admissions for edema. She reports eating only half a meal because of abdominal fullness. Her blood pressure is 112/68 mmHg, heart rate 84/min, and oxygen saturation 96% on room air. The echocardiogram reports a left ventricular ejection fraction of 60%, severe tricuspid regurgitation, marked right atrial enlargement, and a dilated annulus.

The preserved left ventricular ejection fraction does not explain away her illness. The pattern suggests an atrial contribution to secondary regurgitation, but the full report must also address right ventricular function, pulmonary pressures, and other valve lesions. The RN documents congestion and functional decline, checks medication use and laboratory trends, and communicates the repeated admissions. The APRN coordinates decongestion and valve-center assessment rather than treating each admission as an unrelated fluid episode. Her symptoms and trajectory, not one reassuring left-heart measurement, drive the next decision (Mukherjee et al., 2025; Praz et al., 2025).

Urgency increases with hypotension, new confusion, cool extremities, marked oliguria, severe hypoxemia, syncope, or rapidly worsening congestion. These findings require immediate assessment for low output, arrhythmia, sepsis, pulmonary embolism, or another

Two remodeling pathways to secondary regurgitation



Systolic backflow → venous congestion → further remodeling

Figure 2. Atrial and ventricular remodeling can both produce secondary regurgitation. Burgundy arrows show systolic backflow; the patterns may overlap in an individual patient. Original schematic. Content: Mukherjee et al. (2025); Praz et al. (2025).

acute process. The presence of chronic severe regurgitation must not become an explanation that obscures a new emergency. Escalation should communicate the baseline, the change, current vital signs, relevant procedure or device history, and the response to treatment.

Imaging: integrate severity, mechanism, and ventricular reserve

Transthoracic echocardiography is the starting examination. It should describe the valve mechanism, regurgitation or stenosis severity, right atrial and ventricular size, right ventricular function, inferior vena cava findings, estimated pulmonary pressures, and associated left-sided disease. Right-ventricular-focused and additional acoustic windows are needed because a routine four-chamber image may truncate the chamber or obscure a leaflet. A report that says only “severe TR” leaves several treatment-defining questions unanswered (Mukherjee et al., 2025; Zoghbi et al., 2017).

Tricuspid regurgitation is dynamic. Rhythm, respiration, blood pressure, preload, diuretic treatment, sedation, and ventilation can change its appearance. Compare studies in their clinical context, particularly when an

intraoperative or post-diuresis examination seems less severe than a previous study. Document heart rate and hemodynamic conditions. Do not discard a convincing history of substantial regurgitation simply because a single examination was obtained under different loading conditions (Praz et al., 2025; Zoghbi et al., 2017).

Grade regurgitation with converging evidence

Color Doppler identifies the jet but jet area alone is not a reliable severity measure. Eccentric wall-hugging jets, multiple orifices, low driving pressure, and instrument settings can distort the apparent size. Vena contracta measures the narrowest regurgitant stream near the orifice. Flow convergence, effective regurgitant orifice area, regurgitant volume, continuous-wave Doppler contour, hepatic venous flow, chamber remodeling, and leaflet morphology provide complementary information. Discordant findings should prompt reassessment, not selective use of whichever number fits a preferred conclusion (Zoghbi et al., 2017).

“Massive” and “torrential” extend the severe end of a five-grade research and interventional scale. These terms help describe very large lesions and treatment response; they do not make severe regurgitation benign or establish an automatic device indication. The

Table 1. Selected findings that support severe native tricuspid regurgitation

Finding	Interpretation and caution
Vena contracta width ≥ 7 mm	Supports severe TR when acquired correctly; a single width may miss a noncircular or multiple orifice.
EROA ≥ 0.40 cm ² or regurgitant volume ≥ 45 mL/beat	Conventional severe thresholds; PISA assumptions and loading conditions can introduce error.
Hepatic venous systolic flow reversal	Supports important TR; interpret with rhythm, respiration, and the full examination.
Dense, early-peaking triangular continuous-wave signal	Rapid RV–RA pressure equalization may accompany severe TR. Low jet velocity is not reassuring.
RA/RV enlargement, annular dilation, deficient coaptation	Describe mechanism and consequences; chamber enlargement alone does not grade severity.

Content: Zoghbi et al., 2017; Mukherjee et al., 2025.

laboratory should identify the grading method used. A reduction by two categories may still leave clinically important regurgitation, so improvement must be described alongside the residual grade, symptoms, and ventricular response (Hausleiter et al., 2026; Praz et al., 2025).

Assess the right ventricle beyond TAPSE

Tricuspid annular plane systolic excursion, or TAPSE, measures longitudinal motion rather than global ejection fraction. The 2025 ASE guidance identifies values greater than 1.70 cm as normal in the appropriate context; tissue Doppler systolic velocity greater than 9.5 cm/s and fractional area change greater than 35% are additional conventional reference points. None is sufficient alone. Integrate image quality, regional wall motion, chamber geometry, strain where available, three-dimensional measures, and the clinical state (Mukherjee et al., 2025).

Severe regurgitation may preserve or exaggerate annular excursion despite impaired effective forward performance. Surgery, pacing, tethering, and alignment can also alter TAPSE. There is no valid universal bedside conversion of TAPSE into right ventricular ejection fraction by multiplying by a fixed constant. Similarly, a single low TAPSE is not by itself an absolute intervention-futility threshold. The team needs a coherent assessment of contractile reserve and loading, especially before complete elimination of regurgitation (Mukherjee et al., 2025).

Doppler estimates of pulmonary systolic pressure use the tricuspid jet velocity and an estimate of right atrial pressure. With severe free-flowing regurgitation, rapid pressure equalization or an incomplete signal may underestimate the gradient. A low reported pressure therefore cannot exclude important pulmonary vascular disease. Inferior vena cava size and inspiratory collapse also have limitations, including ventilation and altered volume status. Right heart catheterization is used when accurate pulmonary hemodynamics, vascular resistance, or classification will change the intervention decision (Mukherjee et al., 2025; Praz et al., 2025).

Use complementary modalities for specific questions

Transesophageal and three-dimensional echocardiography clarify leaflet number, grasping zones, coaptation gaps, lead interaction, and procedural anatomy. The tricuspid valve is anterior and relatively distant from the esophageal probe; shadowing and poor acoustic windows can still limit visualization. Inadequate imaging is itself a procedural concern because secure leaflet capture cannot be assumed from fluoroscopy alone. Selected centers use intracardiac imaging when it improves an otherwise limited assessment (Praz et al., 2025; Zoghbi et al., 2024).

Cardiac magnetic resonance provides reproducible right ventricular volumes and ejection fraction, quantifies flow, and helps characterize congenital connections and myocardial disease. An indirect regurgitant volume can be calculated as right ventricular stroke volume minus pulmonary forward flow; regurgitant fraction equals that difference divided by right ventricular stroke

volume, multiplied by 100. For example, 120 mL minus 70 mL gives 50 mL, and 50/120 gives approximately 42%. This calculation requires valid acquisition and attention to shunts or other confounding flow lesions; it is not a substitute for integrated grading (Shah et al., 2016; Zoghbi et al., 2017).

Computed tomography is particularly useful for annular dimensions, venous access, relationships to adjacent structures, prosthetic anatomy, and replacement planning. Review kidney function, previous contrast reactions, rhythm, and the planned acquisition before testing. Contrast reflux into the hepatic veins can suggest elevated right-sided pressures or regurgitation but is not a standalone severity test. CT and magnetic resonance tissue characterization can help distinguish thrombus from tumor, yet histology or microbiology may still be required when the diagnosis changes treatment (Shah et al., 2016; Zoghbi et al., 2024).

A useful nursing imaging handoff contains the reason for the study, current rhythm and blood pressure, recent diuresis, oxygen or ventilatory support, implanted devices, contrast history, renal results, and any swallowing or sedation concern. After testing, reconcile the report with the patient: an anatomically suitable valve does not automatically mean the patient will benefit, and an inconclusive scan does not mean disease is absent.

Congenital disease changes the entire circulation

Ebstein anomaly is more than a low-positioned valve. Failed delamination produces abnormal attachment and apical displacement of leaflet tissue, particularly the septal and inferior components, with an atrialized portion of the right ventricle and variable dysfunction of the remaining functional ventricle. Regurgitation may coexist with an atrial communication, right-to-left shunting, cyanosis, or accessory electrical pathways. The relative sizes and functions of the atrialized and functional ventricular portions matter when planning treatment. Figure 3 contrasts this arrangement with a normally positioned valve (Gurvitz et al., 2026; Shah et al., 2016).

Evaluation combines echocardiography, magnetic resonance when useful, rhythm assessment, oxygenation, and functional status. Palpitations or syncope need careful investigation because accessory pathways and atrial tachyarrhythmias may complicate the disease. New desaturation should not be attributed automatically to respiratory infection. Valve

reconstruction, including cone-type repair in suitable anatomy, replacement, arrhythmia treatment, and shunt management are specialized decisions. Closing an atrial communication without understanding its hemodynamic role can be harmful (Gurvitz et al., 2026).

CASE 2 • Palpitations in an adult with Ebstein anomaly

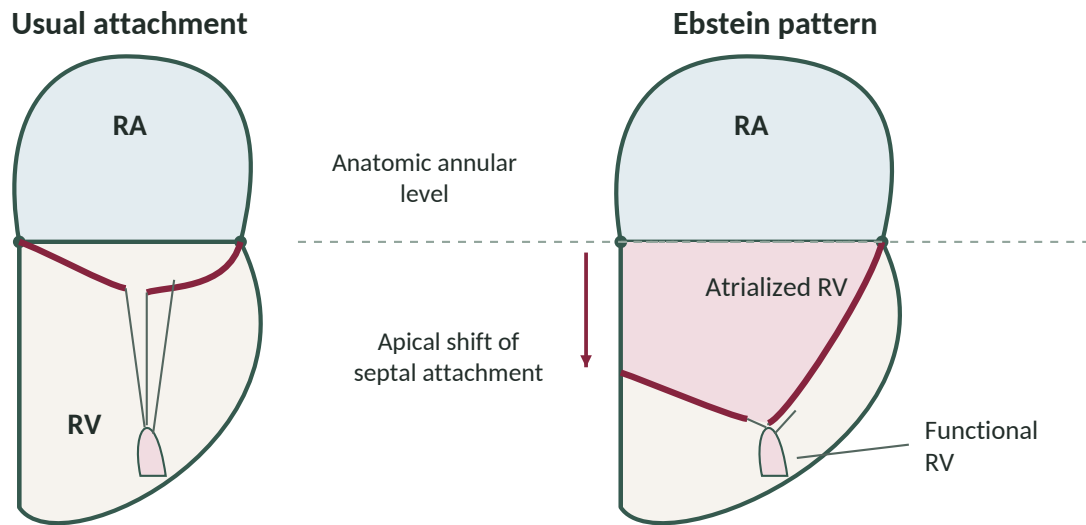
Daniel, 29, has known Ebstein anomaly and misses several years of congenital cardiology follow-up. He presents with episodic rapid palpitations and near-syncope. Oxygen saturation is 91%, lower than the 96% recorded at his last visit. He is currently alert with a blood pressure of 118/72 mmHg.

The next step is not routine reassurance because he is young. The team obtains rhythm documentation, compares oxygenation with baseline, and evaluates valve function, ventricular performance, and possible shunting. Sustained instability would require emergency arrhythmia care. Even after symptoms settle, congenital and electrophysiology review is warranted. The RN emphasizes that symptom resolution does not remove the need for evaluation. Decisions about valve repair, ablation, or closure of an atrial communication must account for the complete congenital anatomy (Gurvitz et al., 2026).

Tricuspid atresia means there is no patent direct connection from the right atrium to the right ventricle. Systemic venous blood must reach the left atrium through an atrial communication; pulmonary blood flow then depends on the associated ventricular communication, outflow anatomy, great-artery arrangement, and ductal or surgically created connections. The right ventricle is usually underdeveloped. Neonatal stabilization and staged palliation are tailored to the circulation rather than performed as one identical sequence in every infant (Shah et al., 2016).

Many survivors have a Fontan-type circulation rather than a repaired normal biventricular heart. Later care must account for elevated systemic venous pressure, rhythm problems, thrombosis risk, exercise limitation, and hepatic complications. A routine adult tricuspid-regurgitation pathway cannot be transferred uncritically to this population. Pediatric congenital specialists guide childhood care, while a planned

Ebstein anomaly: the functional valve plane is displaced



Associated shunts and accessory electrical pathways can change oxygenation and rhythm risk. New symptoms require congenital heart disease expertise.

Figure 3. Apical displacement of the septal attachment in Ebstein anomaly creates an atrialized portion of the anatomical RV. The anterior leaflet and associated anatomy vary. Original schematic, not a diagnostic measurement. Content: Shah et al. (2016); Gurvitz et al. (2026).

transition to adult congenital services helps prevent loss to follow-up (Gurvitz et al., 2026).

In congenitally corrected transposition, both atrioventricular and ventriculoarterial connections are discordant. The morphological right ventricle and its tricuspid valve may support the systemic circulation. Systemic tricuspid regurgitation therefore has different loading and intervention considerations from the usual subpulmonary lesion. An atrioventricular septal defect may involve a common atrioventricular valve rather than two normally formed valves. Crisscross arrangements require sequential description of chambers and connections, not an assumption that spatial position identifies ventricular morphology (Gurvitz et al., 2026; Shah et al., 2016).

A Gerbode communication directs blood from the left ventricle to the right atrium, directly or through a more complex pathway. Its high-velocity systolic jet can be mistaken for tricuspid regurgitation and lead to an erroneous estimate of pulmonary pressure. Membranous septal aneurysmal tissue and prior ventricular septal defect repair can further complicate imaging. Temporary tricuspid leaflet detachment is a specialized surgical exposure technique for selected septal defects;

postoperative assessment should look for residual shunt, valve distortion, and conduction injury rather than presume an uncomplicated valve result (Gurvitz et al., 2026; Shah et al., 2016).

Acquired structural lesions, infection, and cardiac devices

Infection and masses require a different pathway

Right-sided infective endocarditis can occur with intravascular devices, implanted cardiac hardware, healthcare exposure, or injection drug use. Valve destruction may cause acute regurgitation, while septic pulmonary emboli produce pleuritic pain, hypoxemia, or pulmonary infiltrates. Fever and a right-sided mass require synthesis of examination, blood cultures, microbiology, and imaging. A mobile echodensity is not automatically a vegetation, but a nondiagnostic scan does not exclude infection when clinical suspicion remains high (Delgado et al., 2023).

Obtain blood cultures before antimicrobial treatment when feasible without delaying urgently needed sepsis therapy. Transthoracic imaging may visualize the anterior tricuspid valve well; transesophageal imaging and additional modalities are used when findings are

inadequate or complications, prosthetic infection, or device infection are suspected. Treatment is organism- and susceptibility-directed with infectious disease and cardiology input. Source control, including management of infected hardware, may be essential. An elective transcatheter valve implant is not a treatment for uncontrolled active infection (Delgado et al., 2023; U.S. Food and Drug Administration [FDA], 2024a, 2024b).

Surgical assessment is appropriate when infection remains uncontrolled, severe valve dysfunction causes refractory right-heart failure, or recurrent septic pulmonary embolization and other complications make medical treatment inadequate. Vegetation size is considered with these features; it is not an isolated command to operate. Repair is desirable when feasible. Valve excision without replacement is an exceptional strategy with significant hemodynamic consequences, not routine management. Care should include respectful addiction treatment and harm-reduction support when relevant, without allowing stigma to delay necessary treatment (Delgado et al., 2023).

The differential diagnosis of a tricuspid or right atrial mass includes thrombus, vegetation, benign tumor, and malignancy. Thrombus may accompany venous thrombosis, intracardiac catheters, low-flow states, or hypercoagulability. A mobile right-heart thrombus can signal an embolic emergency. Fibroelastoma is a valvular tumor; myxoma often arises in a chamber and may prolapse through the valve. Lymphoma, metastases, and angiosarcoma can infiltrate or distort right-heart structures. Attachment, mobility, enhancement, invasion, systemic findings, and clinical history guide interpretation. A single magnetic resonance signal pattern cannot establish every diagnosis (Shah et al., 2016).

Distinguish a lead near the valve from a lead causing disease

A transvalvular pacing or defibrillator lead may impinge on a leaflet, entangle chordae, perforate tissue, or become adherent through fibrosis. Pacing-related changes in ventricular activation can also contribute indirectly. “CIED-associated” regurgitation describes coexistence with a cardiac implantable electronic device; “CIED-related” disease requires evidence that the device contributes causally. Three-dimensional imaging is particularly useful when the direction of the jet and leaflet motion suggest mechanical interference (Andreas et al., 2024).

Lead extraction or repositioning is not automatically the correct solution. A chronic adherent lead may damage the valve during extraction, and annular dilation may persist even after the mechanical obstacle is removed. Conversely, implanting a replacement valve across an existing lead can jail it, complicating future extraction, infection management, or lead failure. Electrophysiology, imaging, structural intervention, and surgical teams should consider pacing dependence, alternative pacing options, lead age, infection, anatomy, and procedural risk together (Andreas et al., 2024).

CASE 3 • The pacemaker lead is not the whole diagnosis

Aisha, 72, develops edema after several years of transvenous pacing. A new three-dimensional study shows restricted septal leaflet motion where the lead crosses the valve, but also marked annular dilation. She has no fever, the device pocket is normal, and there are no current clinical findings of infection.

These observations support a mechanical lead contribution, not an assumption of endocarditis. The referral should include the device type, pacing dependence, lead history, previous echocardiograms, and the imaging evidence of interference. The RN should not promise that lead removal will cure the regurgitation. An electrophysiology–valve discussion is needed because extraction can injure tissue and may not correct established remodeling. New fever, bacteremia, or pocket changes would create a separate urgent infection pathway (Andreas et al., 2024; Delgado et al., 2023).

Systemic diseases can scar, restrict, or infiltrate the valve

Rheumatic disease can thicken leaflets, shorten chordae, and fuse commissures, often producing mixed stenosis and regurgitation with other valve involvement. Degenerative or connective-tissue disease may produce redundant tissue, prolapse, or flail leaflets. Trauma, endomyocardial biopsy, and intracardiac instrumentation can disrupt the apparatus. In systemic lupus erythematosus or antiphospholipid syndrome, sterile vegetations or thrombotic lesions may resemble infection; microbiologic and systemic assessment are needed before choosing treatment (Shah et al., 2016).

Carcinoid heart disease is a fibrotic valvulopathy caused by circulating vasoactive products, prominently serotonin, rather than simply metastatic tumor deposited on a leaflet. The serotonin metabolite 5-HIAA is a disease-activity marker, not the substance that should be described as directly activating the fibrotic receptor pathway. Thickened, retracted, poorly mobile right-sided leaflets may cause regurgitation, stenosis, or both. The pulmonary valve also requires assessment. Flushing or diarrhea in a patient with a neuroendocrine tumor should prompt attention to cardiac symptoms and an appropriate surveillance pathway (Grozinsky-Glasberg et al., 2022).

Control of hormone secretion and treatment of established valve failure address different problems. Improvement in diarrhea does not prove that fixed valvular fibrosis has reversed. Decisions about surgery integrate symptoms, right ventricular condition, nutrition, tumor prognosis, and the risk of a carcinoid crisis. Bioprosthetic valves are commonly favored in this setting; the possibility of recurrent plaque does not justify a blanket preference for a mechanical tricuspid valve. Periprocedural planning with neuroendocrine and anesthesia specialists is important (Grozinsky-Glasberg et al., 2022).

Amyloidosis, sarcoidosis, radiation-associated injury, and other infiltrative or inflammatory conditions can involve the valve apparatus or the myocardium that supports it. Drug exposure history also matters because some serotonergic and ergot-derived agents have been associated with restrictive valvulopathy. These are prompts for targeted evaluation, not reasons to stop a prescribed medication without review. The treatment plan must separate reversible contributors from established structural damage and avoid attributing every abnormal leaflet to the most familiar diagnosis (Grozinsky-Glasberg et al., 2022; Shah et al., 2016).

Medical management: relieve congestion without losing the referral window

Medical treatment addresses congestion and the underlying contributors to regurgitation. Loop diuretics are central when fluid overload is present. Therapy for left-sided heart failure, atrial fibrillation, pulmonary disease, and other conditions should be optimized according to their own indications. Mineralocorticoid receptor antagonists, sodium–glucose cotransporter 2 inhibitors, or additional diuretic strategies may be appropriate for the associated heart-failure phenotype

and clinical circumstances. These therapies should not be presented as proven repairs of a damaged tricuspid valve (Praz et al., 2025).

Nursing monitoring should connect the intended diuretic effect with actual response. Review weight, edema, abdominal symptoms, urine output, blood pressure, orthostasis, kidney function, sodium, potassium, and magnesium. A rise in creatinine has to be interpreted alongside perfusion and congestion rather than triggering an automatic conclusion of overdiuresis. Persistent venous congestion can itself impair kidney function. Conversely, hypotension, dizziness, poor intake, or falling urine output may signal excessive volume removal or deteriorating forward flow. Communicate the pattern and timing, not just an isolated laboratory value (Praz et al., 2025).

Sequential nephron blockade can increase fluid removal but also increases the need for electrolyte and renal surveillance. Potassium supplementation should be reconciled when a mineralocorticoid antagonist or other potassium-altering therapy is changed. Medication reconciliation must include nonprescription anti-inflammatory drugs, supplements, and the patient's actual dosing behavior. A patient who skips a diuretic because of difficulty accessing a toilet needs a practical plan, not merely a higher prescribed dose (Praz et al., 2025).

Rate and rhythm management can improve hemodynamics in selected patients, but neither successful rate control nor sinus rhythm restoration guarantees reversal of longstanding annular disease. Anticoagulation for atrial fibrillation follows the patient's thromboembolic risk and valve/prosthesis context, not the presence of regurgitation alone. Severe tricuspid regurgitation is not by itself a universal indication for anticoagulation. Mechanical prostheses require a vitamin K antagonist; a direct oral anticoagulant is not a substitute for that indication (Otto et al., 2021; Praz et al., 2025).

Pulmonary hypertension requires classification. A high estimated pulmonary pressure in a patient with left-sided valve disease does not automatically justify pulmonary arterial hypertension drugs. The mechanism may be postcapillary disease, lung disease, thromboembolic disease, or mixed physiology. Empirical vasodilator treatment can be inappropriate. When classification affects intervention eligibility or treatment, specialist assessment and invasive hemodynamics may be needed (Mukherjee et al., 2025).

Clinical improvement after decongestion is useful but should not erase the referral question. Recurrent admissions, increasing medication burden, exercise decline, progressive right-heart enlargement, and new organ dysfunction are signals to reassess the structural problem. Medical optimization and referral should usually proceed in parallel. When intervention is unlikely to provide meaningful benefit, symptom-directed and palliative care remain active treatment, with goals discussed openly and revisited as the clinical situation changes (Praz et al., 2025).

Referral, risk, and the decision to intervene

A multidisciplinary valve team evaluates more than operative mortality. The discussion should address the valve mechanism and severity, right ventricular reserve, pulmonary vascular disease, left-sided lesions, frailty, kidney and liver dysfunction, nutritional status, infection, venous access, device leads, life expectancy, and the outcomes that matter to the patient. A person seeking relief from disabling ascites may value improved daily function even when a survival advantage is uncertain. Shared decisions require realistic benefit and risk estimates rather than a promise that a smaller jet will solve every symptom (Praz et al., 2025).

Risk assessment has progressed beyond historical case-series mortality. The TRI-SCORE was developed specifically for in-hospital mortality after isolated tricuspid surgery, and the Society of Thoracic Surgeons offers an isolated tricuspid surgery risk calculator. These tools support discussion and case selection; they are not interchangeable, and neither should be used outside its intended population as a solitary futility rule. Estimates from surgical populations do not automatically predict transcatheter outcomes (Dreyfus et al., 2022; Society of Thoracic Surgeons [STS], n.d.).

Hepatic and renal findings are particularly important because they can reflect the consequences of venous congestion as well as independent chronic disease. Bilirubin elevation, low albumin, ascites, and reduced renal filtration should prompt careful assessment. The conventional MELD score incorporates the international normalized ratio; warfarin can therefore confound interpretation as a measure of intrinsic liver disease. An INR-independent approach may be informative in selected settings, but no liver score replaces integrated cardiac, hepatic, and renal assessment (Dreyfus et al., 2022; Praz et al., 2025).

“Too well for referral” and “too sick for treatment” can become successive stages of the same delayed pathway. Referral does not commit a patient to a procedure. It creates an opportunity to clarify anatomy, establish a baseline, optimize reversible factors, and monitor for a changing indication. Severe symptoms or organ dysfunction should not be required before a valve team is asked to consider a patient with progressive right ventricular enlargement or declining function (Otto et al., 2021; Praz et al., 2025).

Surgery: timing and technique are separate decisions

Concomitant treatment during left-sided valve surgery

The 2025 ESC/EACTS recommendations distinguish severe, moderate, and mild disease rather than using annular diameter as an automatic instruction for everyone. When left-sided valve surgery is already planned, concomitant tricuspid surgery is recommended for severe primary or secondary regurgitation. Repair should be considered for moderate primary or secondary regurgitation. For selected patients with mild secondary regurgitation and annular dilation greater than 40 mm or greater than 21 mm/m², repair may be considered. These correspond to Classes I, IIa, and IIb, respectively (Praz et al., 2025).

The difference between “should be considered” and “may be considered” matters. Annular dilation identifies risk, but the balance also includes right ventricular condition, expected progression, repair durability, conduction injury, and the additional procedure. The randomized concomitant-repair trial in degenerative mitral regurgitation found less tricuspid progression with annuloplasty but more permanent pacemaker implantation. This is a genuine tradeoff, not proof that all additional repair is either harmless or inappropriate (Gammie et al., 2022).

CASE 4 • Moderate regurgitation before mitral repair

Robert, 66, is scheduled for repair of severe degenerative mitral regurgitation. His tricuspid regurgitation is moderate, and the annulus measures 43 mm. He is told that the right-sided valve will probably improve once the mitral valve is repaired.

Improvement is possible, but persistence or progression is also possible. The findings warrant a specific preoperative discussion about concomitant tricuspid repair. The team should explain both the rationale for preventing later regurgitation and the risk of conduction disturbance or pacemaker implantation. The RN checks that the consent discussion addresses the planned procedures and expected postoperative monitoring. This is not the same decision as prophylactic repair in every patient with mild regurgitation; severity, anatomy, and patient preferences must be distinguished (Gammie et al., 2022; Praz et al., 2025).

Isolated surgery and repair versus replacement

Symptomatic severe primary regurgitation is an established surgical indication in an appropriate candidate without severe right ventricular dysfunction or severe pulmonary hypertension. Severe secondary regurgitation may also warrant isolated surgery when symptoms or progressive right ventricular changes are present and advanced ventricular or pulmonary vascular disease does not make the risk disproportionate. U.S. ACC/AHA guidance and the newer European recommendations should be interpreted within their publication dates and regional practice. Neither supports postponing all isolated secondary disease until irreversible organ failure (Otto et al., 2021; Praz et al., 2025).

Annuloplasty reduces annular size and restores a more favorable coaptation geometry. A prosthetic ring or band generally provides more durable annular support than a suture-only reduction, although outcomes also depend on the mechanism and surgical technique. Marked leaflet tethering, deficient tissue, severe destruction, or a previous failed repair can require leaflet reconstruction or valve replacement. Repair is preferred when a durable result is achievable; replacement is not simply a punishment for late referral and repair is not automatically durable because a ring was implanted (Dahou et al., 2019; Praz et al., 2025).

Bioprosthetic and mechanical replacement have different tradeoffs. Bioprostheses can degenerate but may facilitate future valve-in-valve treatment in selected patients. Mechanical prostheses are durable but carry a substantial thrombosis and lifelong anticoagulation burden in the low-pressure right-sided circulation. Choice incorporates age, other prostheses, bleeding risk, reliable access to anticoagulation

monitoring, pregnancy plans, expected longevity, and possible future interventions. Avoid promising a fixed lifespan for any prosthesis (Otto et al., 2021; Praz et al., 2025).

After surgery, monitor for low output, residual regurgitation or obstruction, conduction abnormalities, bleeding, right coronary compromise, renal injury, and infection. A new requirement for pacing may reflect proximity to the conduction system. The postoperative imaging baseline should document the repair or prosthesis and the hemodynamic conditions under which measurements were obtained. Later changes are easier to interpret when a reliable baseline is available (Zoghbi et al., 2024).

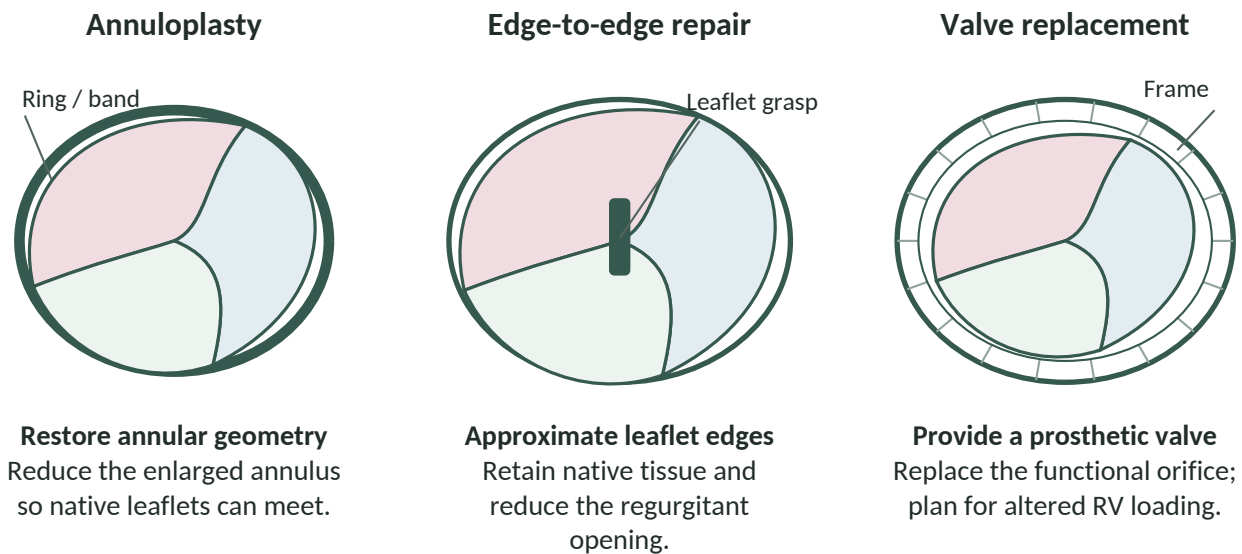
Transcatheter treatment: repair is not replacement

Transcatheter edge-to-edge repair approximates selected leaflet edges to improve coaptation while retaining the native valve. It depends on usable leaflet tissue, adequate visualization, a treatable jet location, and anatomy that permits secure capture without causing important stenosis. Large coaptation gaps, marked tethering, poor image quality, leaflet destruction, or a lead in the intended grasping zone can limit feasibility. A numerical gap threshold should not be treated as a universal rule independent of device, operator experience, and imaging (Abbott Medical, 2024; Praz et al., 2025).

Orthotopic transcatheter replacement places a prosthesis in the native tricuspid position. It can achieve more complete regurgitation elimination in suitable anatomy, including some patients poorly suited for leaflet approximation, but requires CT-based sizing, secure fixation, an acceptable relationship to nearby structures and leads, and an executable antithrombotic plan. The consequences of abruptly removing the low-pressure regurgitant route are especially relevant when right ventricular reserve is limited. Figure 4 compares leaflet approximation, annular reduction, and replacement (Edwards Lifesciences, 2024; Hahn et al., 2025).

The U.S. Food and Drug Administration approved the EVOQUE replacement system on February 1, 2024, and the TriClip G4 repair system on April 1, 2024. The TriClip indication includes symptomatic severe regurgitation despite optimal medical treatment, intermediate or greater surgical risk, and an expectation that repair can reduce regurgitation to moderate or less. EVOQUE is indicated to improve health status in

Different interventions solve different anatomical problems



Mechanism, imaging, ventricular reserve, leads, thrombosis risk, and patient goals determine suitability. These schematics do not select a device.

Figure 4. Annular reduction, leaflet approximation, and prosthetic replacement have different mechanisms and safety implications. Original conceptual diagrams; no commercial device geometry is depicted. Content: Praz et al. (2025); Abbott Medical (2024); Edwards Lifesciences (2024).

patients with symptomatic severe regurgitation despite optimal medical treatment when replacement is considered appropriate by a Heart Team. These are distinct product indications, not interchangeable eligibility checklists (Abbott Medical, 2024; Edwards Lifesciences, 2024; FDA, 2024a, 2024b).

Active infection and inability to tolerate the required antithrombotic treatment are major contraindications. Relevant material hypersensitivity must also be assessed. Before a procedure, the team should review the current product instructions, not only an older trial protocol. The nursing preparation includes confirming medication interruption or continuation instructions, renal and blood-count results, infection screening, access-site assessment, allergies, baseline rhythm, and a plan for pacing dependence. Patient education should identify whether the procedure repairs leaflets or implants a replacement prosthesis (Abbott Medical, 2024; Edwards Lifesciences, 2024).

Other approaches include transcatheter annuloplasty and heterotopic caval valves. Caval implantation aims to reduce venous backflow and congestion without directly correcting the native tricuspid lesion. It is

therefore not physiologically equivalent to orthotopic replacement. Availability, approval, evidence, and reimbursement differ across countries and individual systems. Early feasibility reports for historical spacers, annular devices, or experimental valves must not be presented as evidence that they are routinely available or suitable for all high-risk patients (Hausleiter et al., 2026; Praz et al., 2025).

What randomized evidence now supports

An improvement in a hierarchical composite endpoint does not mean that every component improved. A win ratio compares prioritized outcomes between patient pairs; it is neither a relative mortality reduction nor an event rate. Quality-of-life improvements are clinically important, but they should be named accurately. Compare each trial's population, treatment strategy, follow-up, crossover rules, endpoints, and adverse events before translating results into counseling.

In the full randomized TRILUMINATE Pivotal cohort of 572 patients, edge-to-edge repair improved the hierarchical primary outcome and health status, while mortality or tricuspid surgery and heart-failure hospitalization were not reduced at one year. This

supports a meaningful symptomatic benefit in selected patients without converting that one-year result into a claim of proven survival benefit. The reduction in residual regurgitation also provides a mechanistic explanation for why patients may feel better (Tang et al., 2025).

TRISCEND II randomized 400 patients to EVOQUE replacement plus medical therapy or medical therapy alone. The hierarchical outcome favored replacement, with a win ratio of 2.02, driven primarily by symptom and quality-of-life improvements. Severe bleeding occurred in 15.4% versus 5.3%, and new permanent pacemaker implantation in 17.4% versus 2.3%. These percentages refer to the reported trial groups, not specifically a pacemaker-naïve subgroup. The results require counseling about both health-status benefit and the greater procedural and conduction-related burden (Hahn et al., 2025).

TRIC-I-HF, published in August 2026, extends the evidence beyond the earlier predominantly health-status findings. Among 360 patients with symptomatic severe regurgitation and increased heart-failure risk, a transcatheter repair strategy plus medical treatment reduced the coprimary composite of death or heart-failure hospitalization compared with medical treatment alone. The hazard ratio was 0.40 (95% confidence interval, 0.29–0.55). Estimated freedom from that composite through three years was 52.4% versus 21.0% (Hausleiter et al., 2026).

Interpret those findings with their limits: this was an open-label strategy trial at experienced German centers, different repair systems were used, average follow-up was about 1.6 years, and fewer patients had reached three years. Individual mortality analyses were secondary and not adjusted for multiple comparisons. The result does not establish superiority of one repair device, guarantee survival benefit for every candidate, or demonstrate the same effect for replacement. It does make a blanket statement that tricuspid intervention only improves quality of life outdated (Hausleiter et al., 2026).

The practical lesson is to discuss the outcome actually supported for a patient resembling the trial population. Neither severe end-stage right ventricular failure nor very different congenital anatomy should be assumed to have the same benefit. A favorable trial is a reason to improve referral and selection, not to bypass assessment. The 2025 guideline framework also predates this 2026 trial; new trial evidence should be

distinguished from a formal change in guideline recommendation or product labeling (Hausleiter et al., 2026; Praz et al., 2025).

Tricuspid stenosis and the failing prosthesis

Tricuspid stenosis obstructs diastolic filling. Rheumatic disease is an important cause, often with associated mitral disease and tricuspid regurgitation. Other causes include congenital abnormalities, carcinoid fibrosis, obstructing masses, and prosthetic dysfunction. Congestion may be prominent even when the right ventricle is not markedly enlarged. Doppler assessment integrates leaflet morphology, valve opening, inflow velocity, mean gradient, heart rate, and flow state (Praz et al., 2025; Shah et al., 2016).

A mean diastolic gradient greater than 5 mmHg at a normal heart rate supports severe native stenosis; a valve area around or below 1.0 cm² can provide additional evidence. The gradient is flow-dependent. Tachycardia shortens filling time, while high flow increases velocity, so an elevated value during rapid atrial fibrillation should not be interpreted without reassessment. Conversely, low forward flow can conceal important obstruction. Native-valve thresholds should not be applied mechanically to every prosthetic model (Praz et al., 2025; Zoghbi et al., 2024).

Severe symptomatic stenosis generally requires specialist consideration of intervention. Balloon valvuloplasty may be appropriate in selected isolated stenosis with suitable commissural anatomy and no important regurgitation. It separates fused tissue; it does not reverse the underlying fibrotic disease. Mixed stenosis and regurgitation, marked tissue distortion, or other operative indications may favor surgery. The risk of worsening regurgitation must be considered before dilating either a native or a prosthetic valve (Praz et al., 2025).

A rising gradient across a bioprosthesis requires a differential diagnosis rather than immediate labeling as degeneration. Thrombosis, pannus, structural deterioration, endocarditis, patient–prosthesis mismatch, tachycardia, and increased flow can produce abnormal findings. Compare with the baseline study and the expected performance of the specific model and size. Review anticoagulation, infection symptoms, new regurgitation, leaflet motion, and hemodynamics; transesophageal imaging or CT may clarify the mechanism (Zoghbi et al., 2024).

Valve-in-valve treatment places a transcatheter valve inside a failed surgical bioprosthesis. The true internal diameter, not merely the labeled surgical valve size, is needed for planning. Valve-in-ring treatment is a different and often more difficult problem because a surgical ring may be incomplete, noncircular, or poorly suited to sealing. Neither procedure should be confused with replacement of a native valve. Product-specific labeling and local authorization must be verified because off-label use may be involved (Praz et al., 2025; Zoghbi et al., 2024).

Balloon dilation of a stenotic tricuspid bioprosthesis has been described as a bridge or palliative strategy in selected high-risk patients, but durability is uncertain and evidence is much weaker than for established structural treatment pathways. Exclude potentially treatable thrombosis and active infection before accepting mechanical dilation as the solution. A technically lower gradient immediately after a procedure does not establish sustained clinical benefit (Praz et al., 2025; Zoghbi et al., 2024).

Periprocedural nursing surveillance and medication safety

The procedure handoff should identify the exact repair or replacement, access route, residual regurgitation, inflow gradient, right ventricular performance, hemodynamic support, rhythm changes, and any complication. Include the most recent antithrombotic doses, reversal or transfusion, renal status, contrast exposure, and the next laboratory and imaging checks. “Valve procedure completed” is insufficient information for the receiving nurse to anticipate device-specific risks.

Hypotension after tricuspid intervention has a differential diagnosis: access-site or internal bleeding, tamponade or perforation, new conduction disease, arrhythmia, right ventricular failure after altered loading, pulmonary complications, anesthesia effects, or another cause. Assess perfusion, rhythm, oxygenation, access sites, hemoglobin trends, and urine output while obtaining urgent team review. The absence of visible groin bleeding does not exclude significant internal bleeding. A good-looking valve result does not exclude a life-threatening complication. Figure 5 organizes early recognition and escalation (Abbott Medical, 2024; Edwards Lifesciences, 2024; Zoghbi et al., 2024).

CASE 5 • A successful implant with an unstable patient

Two hours after transcatheter tricuspid replacement, Helen, 81, becomes lightheaded. Blood pressure falls from 116/64 to 82/48 mmHg, heart rate is 42/min, and urine output has declined. The procedure report described minimal residual regurgitation.

The small residual jet is not reassurance against current instability. The RN activates urgent review, assesses airway, oxygenation and perfusion, obtains rhythm documentation, checks access sites and ordered laboratory results, and prepares for the team’s pacing and imaging plan. New conduction disease is a prominent concern, but bleeding, tamponade, and right ventricular failure must also be evaluated. Large empiric fluid boluses or additional diuretics are not automatically appropriate. Management depends on the cause of hypotension and the patient’s loading conditions. The handoff must state the actual deterioration, not only the successful implant result (Edwards Lifesciences, 2024; Hahn et al., 2025).

After edge-to-edge repair, look for recurrent or residual regurgitation, single-leaflet device attachment, leaflet injury, or an excessive inflow gradient. After replacement, surveillance additionally emphasizes prosthetic function, thrombosis, paravalvular regurgitation, conduction disturbance, and right ventricular adaptation. Both procedures can cause vascular, bleeding, embolic, renal, and infectious complications. Monitoring intensity and discharge timing should follow clinical stability, the procedure, and local protocols rather than a generic same-day expectation (Abbott Medical, 2024; Edwards Lifesciences, 2024).

Antithrombotic treatment must match the intervention

A patient with a mechanical tricuspid prosthesis requires lifelong vitamin K antagonist treatment with an individualized target and a reliable monitoring system. Do not substitute a direct oral anticoagulant or stop treatment because the valve is right-sided. For a newly implanted surgical tricuspid bioprosthesis without another anticoagulation indication, the 2025 ESC/EACTS guideline advises considering a vitamin K antagonist for the first three months. Repair and

Early deterioration: assess the patient, not just the implant

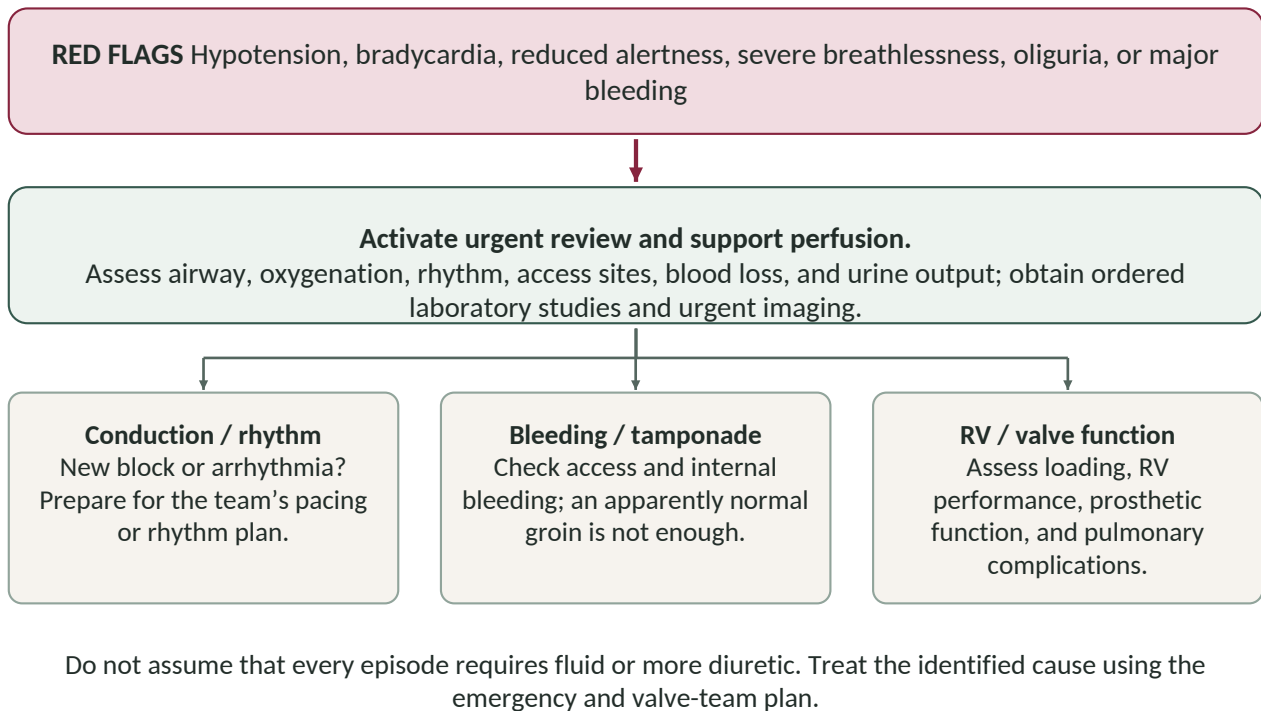


Figure 5. An early-warning framework after tricuspid intervention. Clinical stabilization and evaluation occur in parallel, guided by the treating team and local protocols. Original algorithm. Content: Abbott Medical (2024); Edwards Lifesciences (2024); Zoghbi et al. (2024).

transcatheter replacement require their own plans; these are not identical to aortic transcatheter valve protocols (Otto et al., 2021; Praz et al., 2025).

After transcatheter tricuspid replacement, anticoagulation is commonly used for a longer early period and may continue indefinitely for another indication, but the optimum regimen and duration are not established by one universal randomized protocol. Follow the current device instructions and individualized specialist plan. Inability to take the required treatment is part of preprocedure eligibility, not merely a discharge inconvenience. Do not describe lifelong anticoagulation as mandatory for every biological or caval device regardless of product and patient context (Edwards Lifesciences, 2024; Praz et al., 2025).

Before discharge, reconcile the drug, dose, timing, duration, indication, interacting medications, renal considerations, and any required blood testing. Explicitly resolve whether an antiplatelet agent is still needed when oral anticoagulation is prescribed; unnecessary combination treatment can increase bleeding. Teach the patient what to do after a missed dose using the instructions for that medication, which

symptoms require urgent care, and who authorizes interruption for dental work or another procedure. A plan is not executable until the patient can obtain the medication and monitoring.

Follow-up, education, and meaningful outcomes

Follow-up should assess function and congestion as well as the valve. Ask about walking distance, stairs, sleep, appetite, abdominal fullness, edema, fatigue, and the ability to perform usual activities. Record medication burden and hospitalizations. A symptom questionnaire can help track health status but does not replace examination. Severe residual regurgitation, progressive chamber enlargement, increasing gradients, or a worsening clinical trajectory should trigger reassessment even when a previous procedure was considered technically successful (Praz et al., 2025; Zoghbi et al., 2024).

The 2025 ESC/EACTS guideline supports at least six-monthly clinical and echocardiographic follow-up for moderate or severe regurgitation. New symptoms or rapid change require earlier review. Postprocedure surveillance is individualized to the implant, baseline examination, clinical findings, and local valve program.

Make the next appointment and testing plan explicit, including who will review the results. A referral placed without a completed visit is not continuity of care (Praz et al., 2025).

Teach a practical daily weight and symptom routine where appropriate, with the patient-specific threshold and contact number supplied by the treating team. Patients should not independently make large diuretic or anticoagulant changes based solely on this article. Sodium and fluid advice should reflect congestion, blood pressure, renal function, nutrition, and the prescribed plan. Overly restrictive advice that worsens intake can be counterproductive in a patient with early satiety or frailty. Assess access to a scale, transportation, medications, laboratory testing, and caregiver support.

Urgent symptoms include syncope, severe breathlessness, chest pain, major bleeding, new neurologic symptoms, marked reduction in urine output, or signs of circulatory instability. Fever or chills in a patient with a prosthetic valve, repair material, or implanted cardiac device deserve prompt evaluation for infection. Encourage dental care and good oral hygiene. Antibiotic prophylaxis for selected dental procedures applies to specified high-risk groups, including prosthetic valves and prosthetic repair material; uncomplicated native regurgitation alone is not a universal indication (Delgado et al., 2023).

Use teach-back to test the plan rather than asking only whether instructions were understood. A patient might explain which medicine prevents prosthetic thrombosis, when the next blood test occurs, and which new symptom requires an urgent call. Include interpreter support, accessible written instructions, and a caregiver when appropriate. Patients with congenital heart disease need their specific anatomy and prior procedures documented in a form that can accompany them to unfamiliar services.

For some people, another intervention will not match their goals or offer sufficient likely benefit. Discuss symptom control, practical support, and palliative care without framing these as abandonment. Reassess decisions when function, comorbidity, or preferences change. For others, earlier referral and a carefully selected intervention may substantially improve daily life and reduce heart-failure events. The role of nursing is to make the patient's course visible and to ensure that each decision leads to an actionable next step.

Clinical synthesis

The tricuspid valve is no longer a structure to ignore until treatment becomes hazardous. Its disease must be understood through anatomy, mechanism, congestion, forward flow, ventricular reserve, and the patient's goals. Use integrated imaging, distinguish device presence from device causation, recognize special congenital and systemic conditions, and refer before progressive organ dysfunction closes useful options. Surgery, edge-to-edge repair, and replacement solve different anatomic problems and carry different risks. Current evidence supports more than symptom improvement in selected repair populations, but careful interpretation remains essential. Safe care continues after the procedure through rhythm surveillance, medication reconciliation, imaging follow-up, and patient education.

References

- Abbott Medical. (2024). *TriClip G4 System: Instructions for use*. https://www.accessdata.fda.gov/cdrh_docs/pdf23/P230007C.pdf
- Andreas, M., Burri, H., Praz, F., Soliman, O., Badano, L., Barreiro, M., Cavalcante, J. L., de Potter, T., Doenst, T., Friedrichs, K., Hausleiter, J., Karam, N., Kodali, S., Latib, A., Marijon, E., Mittal, S., Nickenig, G., Rinaldi, A., Rudzinski, P. N., ... Leclercq, C. (2024). Tricuspid valve disease and cardiac implantable electronic devices. *European Heart Journal*, 45(5), 346–365. <https://doi.org/10.1093/eurheartj/ehad783>
- Dahou, A., Levin, D., Reisman, M., & Hahn, R. T. (2019). Anatomy and physiology of the tricuspid valve. *JACC: Cardiovascular Imaging*, 12(3), 458–468. <https://doi.org/10.1016/j.jcmg.2018.07.032>
- Delgado, V., Ajmone Marsan, N., de Waha, S., Bonaros, N., Brida, M., Burri, H., Caselli, S., Doenst, T., Ederhy, S., Erba, P. A., Foldager, D., Fosbøl, E. L., Kovac, J., Mestres, C. A., Miller, O. I., Miro, J. M., Pazdernik, M., Pizzi, M. N., Quintana, E., ... ESC Scientific Document Group. (2023). 2023 ESC guidelines for the management of endocarditis. *European Heart Journal*, 44(39), 3948–4042. <https://doi.org/10.1093/eurheartj/ehad193>
- Dreyfus, J., Audureau, E., Bohbot, Y., Coisne, A., Lavie-Badie, Y., Bouchery, M., Flagiello, M., Bazire, B., Eggenspieler, F., Viau, F., Riant, E., Mbaki, Y., Eyharts, D., Senage, T., Modine, T., Nicol, M., Doguet, F., Nguyen, V., Le Tourneau, T., ... Messika-Zeitoun, D. (2022). TRI-SCORE: A new risk score for in-hospital mortality prediction after isolated tricuspid

- valve surgery. *European Heart Journal*, 43(7), 654–662. <https://doi.org/10.1093/eurheartj/ehab679>
- Edwards Lifesciences. (2024). *Edwards EVOQUE tricuspid valve replacement system: Instructions for use*. https://www.accessdata.fda.gov/cdrh_docs/pdf23/P230013C.pdf
- Gammie, J. S., Chu, M. W. A., Falk, V., Overbey, J. R., Moskowitz, A. J., Gillinov, M., Mack, M. J., Voisine, P., Krane, M., Yerokun, B., Bowdish, M. E., Conradi, L., Bolling, S. F., Miller, M. A., Taddei-Peters, W. C., Jeffries, N. O., Parides, M. K., Weisel, R., Jessup, M., ... Ailawadi, G. (2022). Concomitant tricuspid repair in patients with degenerative mitral regurgitation. *New England Journal of Medicine*, 386(4), 327–339. <https://doi.org/10.1056/NEJMoa2115961>
- Grozinsky-Glasberg, S., Davar, J., Hofland, J., Dobson, R., Prasad, V., Pascher, A., Denecke, T., Tesselaar, M. E. T., Panzuto, F., Albåge, A., Connolly, H. M., Obadia, J.-F., Riechelmann, R., & Toumpanakis, C. (2022). European Neuroendocrine Tumor Society (ENETS) 2022 guidance paper for carcinoid syndrome and carcinoid heart disease. *Journal of Neuroendocrinology*, 34(7), e13146. <https://doi.org/10.1111/jne.13146>
- Gurvitz, M., Krieger, E. V., Fuller, S., Davis, L. L., Kittleson, M. M., Aboulhosn, J. A., Bradley, E. A., Buber, J., Daniels, C. J., Dimopoulos, K., Egbe, A., Geoffrion, T. R., John, A., Khairy, P., Kim, Y. Y., Kreutzer, J., Lewis, M. J., Menachem, J. N., Moore, J. P., ... Wald, R. M. (2026). 2025 ACC/AHA/HRS/ISACHD/SCAI guideline for the management of adults with congenital heart disease: A report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *Circulation*, 153(8), e115–e251. <https://doi.org/10.1161/CIR.0000000000001402>
- Hahn, R. T., Makkar, R., Thourani, V. H., Makar, M., Sharma, R. P., Haeffele, C., Davidson, C. J., Narang, A., O'Neill, B., Lee, J., Yadav, P., Zahr, F., Chadderdon, S., Eleid, M., Pislari, S., Smith, R., Szerlip, M., Whisenant, B., Sekaran, N. K., ... Kodali, S. K. (2025). Transcatheter valve replacement in severe tricuspid regurgitation. *New England Journal of Medicine*, 392(2), 115–126. <https://doi.org/10.1056/NEJMoa2401918>
- Hausleiter, J., Stocker, T. J., Geisler, T., Lurz, P., Rottbauer, W., Lubos, E., Schofer, N., Frerker, C., Frank, J., Thiele, H., Osteresch, R., Rudolph, V., Lauten, A., Xhepa, E., Rothe, J., Rassaf, T., Baldus, S., Nickenig, G., Leistner, D. M., ... Massberg, S. (2026). Tricuspid-valve intervention in heart failure. *New England Journal of Medicine*. Advance online publication. <https://doi.org/10.1056/NEJMoa2606934>
- Mukherjee, M., Rudski, L. G., Addetia, K., Afilalo, J., D'Alto, M., Freed, B. H., Friend, L. B., Gargani, L., Grapsa, J., Hassoun, P. M., Hua, L., Kim, J., Mercurio, V., Saggar, R., & Vonk-Noordegraaf, A. (2025). Guidelines for the echocardiographic assessment of the right heart in adults and special considerations in pulmonary hypertension: Recommendations from the American Society of Echocardiography. *Journal of the American Society of Echocardiography*, 38(3), 141–186. <https://doi.org/10.1016/j.echo.2025.01.006>
- Otto, C. M., Nishimura, R. A., Bonow, R. O., Carabello, B. A., Erwin, J. P., III, Gentile, F., Jneid, H., Krieger, E. V., Mack, M., McLeod, C., O'Gara, P. T., Rigolin, V. H., Sundt, T. M., III, Thompson, A., & Toly, C. (2021). 2020 ACC/AHA guideline for the management of patients with valvular heart disease: A report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *Circulation*, 143(5), e72–e227. <https://doi.org/10.1161/CIR.0000000000000923>
- Praz, F., Borger, M. A., Lanz, J., Marin-Cuartas, M., Abreu, A., Adamo, M., Ajmone Marsan, N., Barili, F., Bonaros, N., Cosyns, B., De Paulis, R., Gamra, H., Jahangiri, M., Jeppsson, A., Klautz, R. J. M., Mores, B., Pérez-David, E., Pöss, J., Prendergast, B. D., ... ESC/EACTS Scientific Document Group. (2025). 2025 ESC/EACTS guidelines for the management of valvular heart disease. *European Journal of Cardio-Thoracic Surgery*, 67(8), eza276. <https://doi.org/10.1093/ejcts/ezaf276>
- Shah, S., Jenkins, T., Markowitz, A., Gilkeson, R., & Rajiah, P. (2016). Multimodal imaging of the tricuspid valve: Normal appearance and pathological entities. *Insights into Imaging*, 7(5), 649–667. <https://doi.org/10.1007/s13244-016-0504-7>
- Society of Thoracic Surgeons. (n.d.). *Isolated tricuspid valve surgery risk calculator*. Retrieved October 5, 2026, from <https://www.sts.org/resources/isolated-tricuspid-valve-surgery-risk-calculator>
- Tang, G. H. L., Hahn, R. T., Whisenant, B. K., Hamid, N., Naik, H., Makkar, R. R., Tadros, P., Price, M. J., Singh, G. D., Fam, N. P., Kar, S., Mehta, S. R., Bae, R., Sekaran, N. K., Warner, T., Makar, M., Zorn, G., Benza, R., Jorde, U. P., ... TRILUMINATE Pivotal Investigators. (2025). Tricuspid transcatheter edge-to-edge repair for severe tricuspid regurgitation: 1-year outcomes from the TRILUMINATE randomized cohort. *Journal of the American College of Cardiology*, 85(3), 235–246. <https://doi.org/10.1016/j.jacc.2024.10.086>
- U.S. Food and Drug Administration. (2024a). *Edwards EVOQUE tricuspid valve replacement*

- system-P230013. <https://www.fda.gov/medical-devices/recently-approved-devices/edwards-evoque-tricuspid-valve-replacement-system-p230013>
- U.S. Food and Drug Administration. (2024b). *TriClip G4 system-P230007*. <https://www.fda.gov/medical-devices/recently-approved-devices/triclip-g4-system-p230007>
- Zoghbi, W. A., Adams, D., Bonow, R. O., Enriquez-Sarano, M., Foster, E., Grayburn, P. A., Hahn, R. T., Han, Y., Hung, J., Lang, R. M., Little, S. H., Shah, D. J., Shernan, S., Thavendiranathan, P., Thomas, J. D., & Weissman, N. J. (2017). Recommendations for noninvasive evaluation of native valvular regurgitation: A report from the American Society of Echocardiography developed in collaboration with the Society for Cardiovascular Magnetic Resonance. *Journal of the American Society of Echocardiography*, 30(4), 303–371. <https://doi.org/10.1016/j.echo.2017.01.007>
- Zoghbi, W. A., Jone, P.-N., Chamsi-Pasha, M. A., Chen, T., Collins, K. A., Desai, M. Y., Grayburn, P., Groves, D. W., Hahn, R. T., Little, S. H., Kruse, E., Sanborn, D., Shah, S. B., Sugeng, L., Swaminathan, M., Thaden, J., Thavendiranathan, P., Tsang, W., Weir-McCall, J. R., & Gill, E. (2024). Guidelines for the evaluation of prosthetic valve function with cardiovascular imaging: A report from the American Society of Echocardiography developed in collaboration with the Society for Cardiovascular Magnetic Resonance and the Society of Cardiovascular Computed Tomography. *Journal of the American Society of Echocardiography*, 37(1), 2–63. <https://doi.org/10.1016/j.echo.2023.10.004>