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# Catheter-Based Interventions for Mitral Regurgitation: Nursing Care and Clinical Considerations

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## Abstract

**Background:** Mitral regurgitation (MR) is the second most prevalent valvular heart disease globally, with clinical presentations ranging from asymptomatic to life-threatening. Traditional surgical repair remains the gold standard, but high-risk patients often require less invasive alternatives.

**Aim:** To review catheter-based interventions for MR, focusing on nursing care, anatomical considerations, procedural techniques, and clinical outcomes.

**Methods:** This narrative review synthesizes evidence from pivotal trials (EVEREST I & II, COAPT) and current guidelines (ACC/AHA, ESC/EACTS), emphasizing anatomical prerequisites, device selection, procedural workflow, and complication management.

**Results:** Transcatheter edge-to-edge repair (TEER), modeled on the Alfieri stitch, has emerged as the most validated percutaneous approach. Favorable anatomy includes adequate leaflet length, central jet location, and mitral valve area >4 cm². Technological advances (MitraClip G4, PASCAL Precision) enable treatment of complex anatomies such as flail segments and commissural jets. TEER demonstrates significant symptomatic improvement and reduced hospitalization in high-risk cohorts, with low rates of major complications (SLDA 1.5–5%, leaflet injury ≤2%, device embolization ≤0.7%). Nursing roles encompass preprocedural preparation, intraoperative monitoring, anticoagulation management, and postprocedural surveillance for complications such as tamponade, vascular injury, and residual MR.

**Conclusion:** TEER represents a transformative option for patients unsuitable for surgery, offering durable MR reduction and improved quality of life when applied with rigorous anatomical assessment and multidisciplinary collaboration.

**Keywords:** Mitral regurgitation, transcatheter edge-to-edge repair, MitraClip, PASCAL, nursing care, structural heart intervention

## Introduction

Mitral regurgitation (MR) constitutes a highly prevalent form of valvular heart disease and is widely recognized as one of the most frequently encountered valvular abnormalities in clinical practice, ranking second only to aortic valve stenosis in overall occurrence.[1][2] The clinical course of MR is heterogeneous, ranging from incidental, clinically silent findings to rapidly progressive, life-threatening hemodynamic compromise. Consequently, therapeutic decision-making is fundamentally anchored in a precise appreciation of the temporal profile of disease onset, the underlying etiologic mechanism, and, most critically, the severity of regurgitation and its physiological consequences. Contemporary management strategies therefore emphasize timely recognition of high-risk phenotypes, careful assessment of ventricular and atrial remodeling, and individualized selection of medical, surgical, or transcatheter interventions. Acute severe MR represents a distinct and particularly unstable clinical entity, most commonly arising from catastrophic structural disruption of the mitral valve apparatus, such as papillary muscle rupture following myocardial infarction or leaflet perforation associated with infective endocarditis.[3] In these settings, the abrupt development of significant regurgitant flow precipitates a sudden rise in left atrial pressure, leading to pulmonary congestion and profound reductions in forward cardiac output. The left ventricle and left atrium have insufficient time to undergo compensatory


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remodeling, and the resulting acute volume overload rapidly culminates in respiratory distress, cardiogenic shock, and marked hemodynamic instability. Because the pathophysiology is dominated by mechanical failure and immediate circulatory deterioration, definitive management typically requires urgent surgical correction rather than prolonged stabilization, underscoring the time-sensitive nature of intervention in acute severe presentations.[3] In contrast, chronic MR generally evolves more gradually and can be broadly classified into primary and secondary forms, a distinction that has major implications for both prognosis and treatment selection. Primary MR is defined by intrinsic pathology affecting one or more elements of the mitral valve complex, including the leaflets, chordae tendineae, papillary muscles, or annulus, and is frequently associated with degenerative processes that alter leaflet integrity and coaptation.[3][4] By comparison, secondary MR is principally functional in origin and reflects perturbations in left ventricular or left atrial geometry and performance that secondarily distort the mitral apparatus, even when leaflet tissue itself is structurally normal.[3][4] This mechanistic framework is clinically essential because it clarifies whether intervention should primarily address valve structure directly or focus on the underlying myocardial disease and chamber remodeling that generate regurgitation.

For patients with chronic MR who remain minimally symptomatic or asymptomatic and demonstrate only mild regurgitation, conservative management with longitudinal surveillance is often appropriate, provided that cardiac structure and function remain preserved.[3] Medical therapy in such cases is typically directed toward comorbid conditions and risk-factor optimization, while periodic reassessment aims to identify early signs of adverse remodeling or the emergence of symptoms. However, once chronic MR becomes symptomatic, the balance of risk and benefit shifts substantially, and patients should undergo comprehensive evaluation to determine suitability for surgical intervention, including repair or replacement strategies depending on valve anatomy and institutional expertise.[3][4] Importantly, the presence of symptoms often signals that compensatory mechanisms are failing and that regurgitation is exerting clinically meaningful effects on functional capacity and cardiopulmonary reserve, making timely escalation of care essential. Even in the absence of overt symptoms, selected individuals with chronic MR may warrant consideration for intervention when objective markers indicate evolving cardiac compromise or increased risk of irreversible deterioration. Specifically, surgical evaluation becomes particularly relevant when there is evidence of left ventricular systolic dysfunction or progressive chamber dilatation, as these changes may reflect the transition from compensated volume overload to decompensation.[5][6] Additionally, the development of atrial fibrillation or pulmonary hypertension in the context of chronic MR may represent important thresholds suggesting rising left atrial pressures, atrial remodeling, and pulmonary vascular consequences of longstanding regurgitant burden.[5][6] These features are clinically significant because outcomes can worsen if intervention is delayed until advanced remodeling has occurred, and they reinforce the importance of integrating imaging and hemodynamic indicators into decision-making rather than relying on symptom status alone. Accurate characterization of MR severity and mechanism is therefore central to contemporary management, and transthoracic echocardiography (TTE) is widely regarded as the initial imaging modality of choice for screening and diagnostic evaluation.[3] TTE provides essential information regarding mitral valve morphology and motion, helps identify the mechanistic basis of regurgitation, and supports estimation of disease severity while simultaneously assessing left ventricular function, left ventricular size, and left atrial dimensions.[3] In routine clinical practice, MR evaluation relies on an integrated approach that synthesizes qualitative and quantitative indices, including two-dimensional assessment of leaflet characteristics and coaptation, evaluation of regurgitant jet features relative to left atrial area, measurement of vena contracta, calculation of effective regurgitant orifice area and regurgitant volume, and appraisal of ventricular systolic performance through left ventricular ejection fraction and end-diastolic size parameters. This multiparametric strategy is crucial because no single measurement fully captures the complexity of MR, particularly when loading conditions vary or when regurgitation is dynamic.

Nevertheless, TTE may be limited by suboptimal acoustic windows, complex valve anatomy, or the need for more detailed visualization of leaflet pathology and scallop-specific involvement. In such circumstances, transesophageal

echocardiography (TEE) offers enhanced spatial resolution and a more comprehensive depiction of the mitral valve apparatus.[7][8] The emergence of three-dimensional TEE has further refined diagnostic capability by providing an "enface" perspective of the mitral valve that closely approximates the operative view, thereby facilitating clearer communication among multidisciplinary teams and supporting precise procedural planning.[7][8] When TEE is contraindicated or not feasible, cardiac magnetic resonance imaging may serve as an alternative modality, offering robust quantification of regurgitant severity and highly accurate evaluation of left ventricular volumes and dimensions, which can be particularly valuable when echocardiographic measurements are equivocal. In parallel with advances in imaging and risk stratification, the therapeutic landscape for MR has expanded considerably, particularly for patients deemed at elevated risk for conventional surgery. Findings from more recent investigations have supported percutaneous mitral valve repair as an important alternative for high-surgical-risk individuals with severe symptomatic MR, demonstrating favorable procedural safety profiles and comparatively low rates of morbidity and mortality in appropriately selected patients.[9] This evolution is clinically meaningful because a substantial subset of patients with advanced age, frailty, or multiple comorbidities may be ineligible for surgical repair despite significant symptom burden and adverse prognosis if left untreated. As such, transcatheter options have become integral to modern heart-team discussions, emphasizing patient-centered selection criteria, procedural feasibility, and alignment of therapeutic intensity with expected clinical benefit. The evidentiary foundation for catheter-based edge-to-edge repair was substantially shaped by pivotal trials that established both feasibility and long-term performance. The Endovascular Valve Edge-to-Edge Repair Study Trial (EVEREST) 1 provided critical early validation by demonstrating the safety and procedural practicality of an edge-to-edge repair approach in the percutaneous setting. The subsequent EVEREST 2 randomized controlled trial compared percutaneous edge-to-edge repair with surgical mitral valve repair or replacement, suggesting greater efficacy of surgery in achieving more complete MR reduction while simultaneously affirming the long-term safety of the transcatheter device and its sustained capacity to lessen regurgitation in selected cohorts.[10][11] Collectively, these data informed clinical adoption patterns by clarifying the relative strengths of each strategy, supporting the role of percutaneous repair in patients for whom surgical risk is prohibitive, and underscoring the importance of careful anatomical and clinical selection to optimize outcomes.

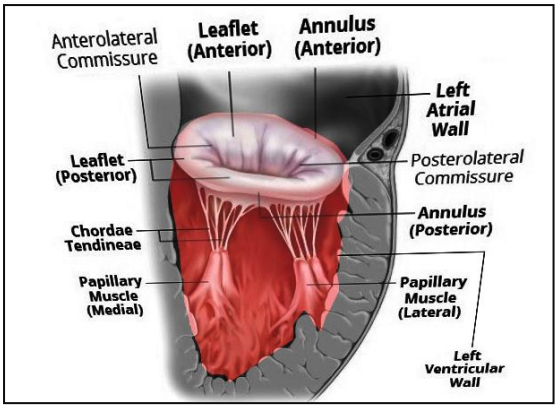


**Fig. 1**: Mitral valve anatomy.

Conceptually, edge-to-edge leaflet repair is grounded in the surgical "Alfieri stitch," a technique developed by Dr. Ottavio Alfieri that approximates the mitral leaflets at the site of regurgitation to enhance coaptation and reduce backward flow.[12][13] Translating this principle into a minimally invasive, catheter-based therapy enables mechanical leaflet approximation without the need for open-heart surgery, typically resulting in the formation of a double-orifice configuration analogous to the surgical construct.[12][13] The procedural objective is not merely to reduce the visual appearance of regurgitant jets but to achieve hemodynamically meaningful reduction of regurgitant volume, thereby alleviating left atrial pressure elevation, improving pulmonary congestion, and supporting ventricular-atrial coupling under physiologic loading conditions. Beyond edge-to-edge repair, the contemporary percutaneous repertoire includes multiple strategies designed to address diverse mechanisms of MR in patients with substantial comorbidity burdens and heightened operative risk.[14] These catheter-based therapies are often categorized according to the anatomical target within the mitral apparatus, reflecting the recognition that MR may arise from leaflet malcoaptation, annular dilatation, chordal disruption, or adverse ventricular remodeling.[14][15][16] Accordingly, available approaches encompass leaflet-focused devices that approximate or stabilize leaflet coaptation, annuloplasty techniques that reshape or reduce annular dimensions through direct or indirect means, chordal-based strategies involving neo-chord

implantation or percutaneous chordal repair concepts, and ventricular remodeling interventions aimed at modifying left ventricular geometry to mitigate tethering forces and functional regurgitation.[14][15][16] The growth of these options underscores a broader paradigm shift in MR care toward mechanism-specific, anatomy-driven therapy, supported by sophisticated imaging, multidisciplinary evaluation, and the goal of achieving consistent, durable improvements in symptoms and clinical outcomes across an increasingly complex patient population.

**Anatomy and Physiology**

The anatomical understanding of the mitral valve has evolved alongside the broader history of cardiac science, and even its nomenclature reflects an early attempt to link structure with recognizable form. Andreas Vesalius was the first to propose the term “mitral” for the left-sided atrioventricular valve because of its perceived resemblance to a bishop’s miter, a comparison that has remained enduring in medical terminology and continues to provide a memorable reference point for learners and clinicians alike.[17][18] While the name itself is historically rooted, contemporary clinical practice emphasizes that the mitral valve is not a simple flap-like structure but rather a highly integrated apparatus whose components function in synchrony to preserve unidirectional blood flow from the left atrium to the left ventricle. From an anatomical perspective, the mitral valve apparatus is best conceptualized as a dynamic system composed of multiple interdependent elements: the anterior and posterior mitral leaflets, the mitral annulus, the subvalvular apparatus that includes the chordae tendineae and papillary muscles, and the left ventricle, which provides the geometric and functional environment necessary for normal valve operation (see Images. Mitral Valve Anatomy and Mitral Valve, Transverse View). The mitral leaflets are specialized fibrous tissues designed to withstand repetitive mechanical stress while forming a competent seal during systole. The anterior leaflet is typically larger and occupies a greater portion of the annular circumference, whereas the posterior leaflet, though smaller, is often described as having multiple scallops that contribute to nuanced coaptation and allow a high degree of adaptability to ventricular motion. The annulus, serving as the fibrous hinge line for leaflet attachment, provides a structural foundation while simultaneously undergoing cyclical deformation throughout the cardiac cycle. The mitral annulus itself is not a flat ring; rather, it is a saddle-shaped structure whose three-dimensional configuration is critical to effective leaflet stress distribution and maintenance of valve competence.[2][18][19] This saddle geometry reduces leaflet strain and supports efficient closure by optimizing the spatial relationship between leaflet edges and chordal insertions. Annular contraction during systole contributes to effective reduction of the annular area, thereby promoting leaflet coaptation. Conversely, annular dilatation or flattening—whether due to chronic volume overload, atrial enlargement, or ventricular remodeling—can disrupt these relationships, increasing the likelihood of incomplete closure and regurgitant flow.

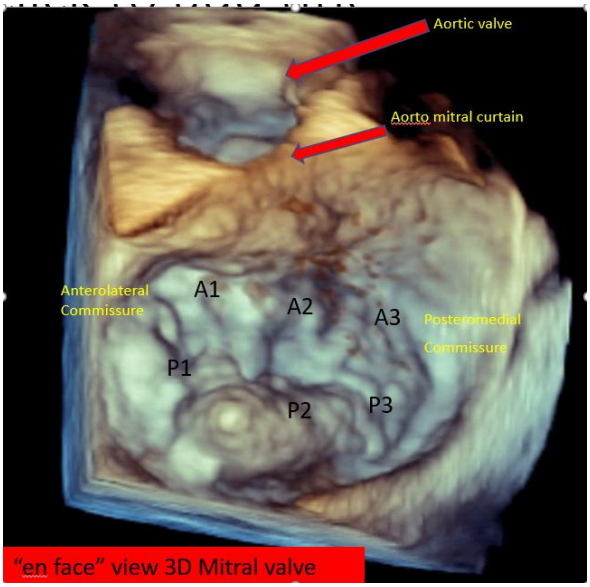


**Fig. 2:** Mitral valve.

Physiologically, the central requirement for preventing mitral regurgitation is reliable leaflet coaptation during systole. In normal function, as the left ventricle contracts and intraventricular pressure rises, the mitral leaflets are pushed toward closure. The chordae tendineae, tethered to papillary muscles, counterbalance this pressure by preventing prolapse of the leaflets into the left atrium. This coordinated mechanism depends on appropriate tensioning of the chordae, synchronized papillary muscle contraction, and preserved ventricular geometry. Thus, the mitral valve’s competence is not determined solely by leaflet integrity, but by the coordinated interaction among all components of the mitral apparatus, each of which contributes to stable coaptation and controlled distribution of mechanical forces.[2][18][19] The subvalvular apparatus is particularly vital in maintaining this stability. The chordae tendineae function as fibrous cords

connecting the leaflet free edges to the papillary muscles, and they are organized into primary, secondary, and tertiary groups with distinct insertion patterns and mechanical roles. The papillary muscles, arising from the ventricular myocardium, contract with the ventricle and maintain appropriate chordal tension during systole, thereby preventing leaflet displacement and ensuring that coaptation occurs in the correct plane. If papillary muscle function is compromised—such as in ischemia or infarction—this balance can fail, resulting in malcoaptation, prolapse, or tethering depending on the nature of the injury. These relationships highlight why the left ventricle itself is considered part of the mitral valve apparatus: ventricular size, shape, contractility, and synchrony determine the spatial arrangement of papillary muscles and the vector forces applied to the chordae, directly influencing leaflet closure dynamics. Because mitral competence relies on precise integration, dysfunction in any component can culminate in mitral regurgitation. Structural abnormalities of leaflets, chordae, or papillary muscles can impair coaptation through prolapse, flail segments, or restricted movement, while abnormalities of the annulus can lead to insufficient leaflet overlap and central regurgitation. Likewise, ventricular dilatation or remodeling can displace papillary muscles apically and laterally, increasing tethering forces and preventing adequate leaflet approximation. For this reason, MR is not best understood as a single lesion, but as a final common pathway resulting from diverse structural and functional disturbances affecting the mitral apparatus.[20]

Within this framework, MR is typically categorized into two broad types: primary and secondary. Primary MR is fundamentally a degenerative or structural valve disorder in which the initiating pathology arises from intrinsic abnormalities of the valve tissue or its supporting structures. Such disease processes directly compromise the leaflets or chordae and often produce regurgitation through prolapse or flail motion. In contrast, secondary MR is more characteristically a functional myocardial condition, wherein the mitral leaflets may remain structurally intact but are rendered incompetent because of geometric and functional changes in the left ventricle—commonly described as ventricular remodeling.[20] In secondary MR, alterations in ventricular size or shape distort the annulus and subvalvular apparatus, tethering the leaflets and preventing effective systolic closure. This distinction is clinically consequential because it reinforces that restoring competence in primary MR often requires addressing the valve structure itself, whereas improving secondary MR may depend more heavily on correcting the underlying ventricular pathology and the forces that disrupt leaflet coaptation.

**Etiology of Mitral Regurgitation**

Mitral regurgitation (MR) arises from a heterogeneous set of pathological processes that ultimately converge on a common mechanical outcome: incomplete coaptation of the mitral leaflets during systole, allowing retrograde flow from the left ventricle into the left atrium. Although the clinical phenotype of MR is often described through the lens of severity and symptom burden, a mechanistic understanding begins with etiology, because the underlying cause strongly influences disease trajectory, response to medical therapy, and the relative suitability of surgical versus transcatheter interventions. Contemporary classification distinguishes primary MR, in which the initiating lesion is intrinsic to the mitral valve apparatus itself, from secondary MR, in which the valve is structurally normal or only mildly abnormal but becomes incompetent due to adverse remodeling of the left ventricle or left atrium that distorts the geometry and closing forces required for valve competence. Primary MR encompasses disorders that directly affect one or more components of the mitral valve complex, including the leaflets, chordae tendineae, papillary muscles, or annulus. A major contributor is mitral valve prolapse associated with myxomatous degeneration, a spectrum of connective tissue changes that can weaken leaflet tissue and alter its architecture, producing excessive leaflet motion into the left atrium during systole. Within this myxomatous process, clinically meaningful regurgitation may develop when chordae become elongated, attenuated, or ruptured, or when a flail segment emerges because the leaflet edge loses its tethering support, thereby preventing effective coaptation and generating eccentric, often severe regurgitant jets. Degenerative MR also includes conditions characterized by progressive leaflet thickening, fibrosis, and calcification; these changes may reduce leaflet mobility, alter the coaptation line, and increase annular rigidity, all of which can compromise sealing during systole. Infectious causes represent another important primary category, particularly infective endocarditis, where vegetations may interfere with leaflet closure and destructive

complications such as leaflet perforation can abruptly create substantial regurgitant orifices. Inflammatory etiologies, including rheumatic heart disease and collagen vascular disorders, can produce leaflet restriction and commissural fusion or chordal shortening, leading to malcoaptation through reduced leaflet excursion and altered leaflet geometry. In addition, primary MR may be precipitated or exacerbated by exogenous injury to valve tissue, such as drug-induced valvulopathy or radiation-associated heart disease, both of which can provoke fibrotic thickening and retraction of leaflets and subvalvular structures. Finally, congenital abnormalities—such as parachute mitral valve or a cleft mitral leaflet—can predispose to MR by disrupting normal leaflet formation, chordal distribution, or the symmetry of coaptation. Annular dilation may also function as a primary driver when it reflects intrinsic alterations of the annulus or atrial enlargement that enlarges the valvular orifice beyond what the leaflets can seal, even when leaflet tissue is otherwise preserved [3].

Secondary MR, by contrast, is most often the valvular expression of myocardial disease and altered ventricular mechanics. Ischemic secondary MR typically occurs in the setting of coronary artery disease, where regional wall motion abnormalities and papillary muscle displacement change the tethering forces on the leaflets and prevent complete closure. This mechanism may develop after myocardial infarction or chronic ischemia and is frequently characterized by leaflet tethering rather than prolapse, with regurgitation severity fluctuating with loading conditions and contractility. Nonischemic cardiomyopathy constitutes another major substrate for secondary MR; progressive ventricular dilation and spherical remodeling can enlarge the mitral annulus and displace papillary muscles laterally and apically, thereby increasing leaflet tenting and reducing the effective coaptation surface. In these functional forms, the valve leaflets often remain anatomically intact, yet the closing forces become insufficient to overcome tethering and geometric distortion, emphasizing why correction of the underlying myocardial process and restoration of coordinated ventricular function are central considerations in management. Given the spectrum of etiologies and clinical presentations, both primary and secondary MR are further stratified into staged categories that reflect progression from risk states to advanced symptomatic disease. These stages—commonly designated A through D—serve to integrate anatomical features, hemodynamic severity, downstream chamber consequences, and clinical symptom status, thereby supporting a structured approach to surveillance and intervention planning. In clinical practice, such staging is not merely descriptive; it guides timing of referral, intensity of imaging follow-up, and thresholds for procedural evaluation, particularly in patients who remain asymptomatic despite physiologically severe regurgitation [21].

Within primary MR, Stage A corresponds to individuals “at risk” for regurgitation, in whom early valve abnormalities are present but do not yet produce significant hemodynamic disturbance. This stage may include mild mitral valve prolapse with preserved and effective coaptation, or mild leaflet thickening and restriction that does not translate into a meaningful regurgitant jet. Doppler echocardiography at this stage may show no detectable MR or only a small central jet occupying less than one-fifth of the left atrial area, and the vena contracta is typically small, reflecting a very limited regurgitant orifice. Importantly, there are no major hemodynamic sequelae, and patients are generally asymptomatic. Stage B represents progressive MR, wherein structural abnormalities have become more pronounced and measurable regurgitation is present, yet the disease has not reached the threshold of severe hemodynamic burden. Patients in this category may show moderate to severe mitral valve prolapse while maintaining effective coaptation, or rheumatic-related leaflet restriction accompanied by partial loss of central coaptation. A history of infective endocarditis may also place patients in this stage if residual anatomical disruption contributes to regurgitation without meeting severe criteria. Echocardiographic evaluation commonly demonstrates a regurgitant jet of intermediate magnitude—such as a central jet occupying approximately one-fifth to two-fifths of the left atrial area, or an eccentric jet that appears late systolic—alongside parameters consistent with nonsevere regurgitation, including a vena contracta below severe thresholds, regurgitant volume under 60 mL, regurgitant fraction below 50%, and an effective regurgitant orifice area below 0.40 $cm^2$. Chamber remodeling is typically limited: mild left atrial enlargement may be present, while left ventricular size and pulmonary pressures are often still within normal range, and symptoms are generally absent. Stage C denotes asymptomatic severe primary MR

and captures a clinically important population in whom regurgitation has reached severe hemodynamic criteria despite the absence of overt dyspnea or exercise limitation. Anatomically, this stage may include severe prolapse with clear loss of coaptation, a flail leaflet, advanced rheumatic restriction with central malcoaptation, or structural sequelae after endocarditis. Radiation-associated thickening and retraction of leaflets may also generate severe regurgitation in this category. Hemodynamically, severe MR is reflected by findings such as a central regurgitant jet exceeding two-fifths of the left atrial area, a holosystolic eccentric jet, a vena contracta at or above severe cutoffs, regurgitant volume at or above 60 mL, regurgitant fraction at or above 50%, and an effective regurgitant orifice area at or above 0.4 $cm^2$, often accompanied by high-grade angiographic regurgitation. Although symptoms are absent by definition, the physiological impact is evident through moderate to severe left atrial enlargement and left ventricular enlargement, and pulmonary hypertension may be present either at rest or provoked by exercise. Stage C is further refined into subcategories based on left ventricular systolic function and dimensions, reflecting the prognostic importance of early ventricular decompensation; preserved ejection fraction and smaller end-systolic dimension indicate a more compensated state, whereas reductions in ejection fraction and/or increases in end-systolic dimension suggest early impairment [21].

Stage D describes symptomatic severe primary MR and reflects the point at which the regurgitant lesion and its consequences manifest clinically, most commonly as exertional dyspnea and reduced exercise tolerance. Anatomical and hemodynamic criteria mirror those of Stage C in terms of severe structural disruption and quantitative severity indices, but the distinction is the presence of symptoms attributable to MR. At this stage, left atrial and left ventricular enlargement are typically evident, pulmonary hypertension is more likely to be present, and the overall clinical urgency of intervention increases because persistent severe regurgitation can accelerate heart failure progression and promote irreversible myocardial remodeling if not corrected. The staging framework is also relevant to therapeutic eligibility and procedural planning, particularly as transcatheter strategies continue to expand. While multiple interventions—both surgical and nonsurgical—are used in the treatment of severe MR, patients with severe MR who are deemed high risk or prohibitive risk for surgery are currently the primary subgroup for whom catheter-based management is recommended. This reflects both the historical evidence base and a risk-benefit calculus that prioritizes less invasive therapies when operative risk is excessive. The clinical stakes of appropriate recognition and timely treatment are substantial, because severe MR, when left untreated, can culminate in progressive cardiac dilation, functional decline, recurrent hospitalizations, and ultimately fatal outcomes, including heart failure.[21] It is essential to recognize that although several echocardiographic hemodynamic criteria are commonly cited for defining MR severity, the full set of parameters within any given category is not necessarily present in every patient, and measurements may vary with technical image quality and physiological loading conditions. Therefore, categorizing MR as mild, moderate, or severe requires careful integration of qualitative observations, quantitative indices, and the broader clinical context, rather than reliance on a single measurement in isolation.[3] This integrative principle is particularly important when evaluating patients for intervention, where misclassification can lead either to premature procedural risk or to harmful delay in the setting of truly severe, progressive disease.[3]

**Stages of Secondary Mitral Regurgitation**

Secondary mitral regurgitation (MR), often termed functional MR, is best understood as a valvular consequence of myocardial disease rather than a primary disorder of the leaflets themselves. In this setting, the mitral valve (MV) apparatus is frequently structurally preserved, yet it becomes incompetent because left ventricular (LV) remodeling and altered contractile mechanics distort the geometric relationships that normally ensure effective leaflet coaptation. The staging framework for secondary MR, organized from Stage A through Stage D, offers a clinically meaningful structure for describing the continuum of risk, progressive hemodynamic deterioration, and eventual symptomatic decompensation. This approach integrates valve anatomy, quantitative hemodynamic indices, associated cardiac remodeling, and patient-centered symptom status, thereby supporting surveillance strategies and therapeutic decision-making that are aligned with both pathophysiology and clinical risk. Stage A represents the “at risk” state for secondary MR. In this early phase, the MV leaflets, chordae tendineae, and annulus are

essentially normal, and any regurgitation is absent or trivial. The clinical relevance of Stage A lies in the presence of the underlying myocardial substrate—most commonly coronary artery disease or cardiomyopathy—which creates the conditions for future MR by promoting LV dilation, regional wall motion abnormalities, or global systolic dysfunction. Echocardiographic evaluation at this stage may demonstrate no MR jet or only a small central color Doppler jet occupying less than 20% of the left atrial area, alongside a very small vena contracta, typically under 0.30 cm. Although the valve itself appears competent, the associated cardiac findings begin to reflect the initiating myocardial process: the LV may be normal in size or only mildly dilated, but it often demonstrates either fixed regional dysfunction due to infarction or inducible ischemia with stress. In cardiomyopathic states, early LV dilation and reduced systolic function may be evident even before MR becomes clinically significant. Symptomatically, patients in Stage A may report manifestations related to ischemia or heart failure; however, these complaints are generally attributable to the primary cardiac disease and may improve with revascularization where appropriate and with guideline-directed medical therapy [22].

Stage B describes progressive secondary MR and reflects the point at which the geometric and functional consequences of myocardial disease begin to translate into measurable regurgitation. Anatomically, this stage is characterized by regional wall motion abnormalities with mild tethering of the mitral leaflets and/or annular dilation accompanied by partial loss of central coaptation. The regurgitation remains nonsevere by quantitative standards, commonly reflected by an effective regurgitant orifice (ERO) area below 0.40 $cm^2$, a regurgitant volume under 60 mL, and a regurgitant fraction below 50%. These hemodynamic findings occur in parallel with progressive LV abnormalities, including reduced systolic function associated with ischemic injury or primary myocardial disease, as well as varying degrees of LV dilation. Clinically, symptoms may still be dominated by the underlying coronary ischemia or heart failure syndrome, and they may continue to respond to revascularization and optimization of medical therapy. Nevertheless, Stage B is significant because it indicates the emergence of valve incompetence as a consequence of LV remodeling, and it serves as a marker of advancing disease with potential implications for prognosis, serial follow-up, and the need for more detailed imaging assessments when clinical status changes. Stage C denotes asymptomatic severe secondary MR and represents a pivotal juncture in the disease trajectory, as hemodynamic severity becomes substantial even if the patient does not report clear MR-attributable symptoms. The defining anatomical pattern in this stage is severe leaflet tethering driven by regional wall motion abnormalities and/or LV dilation, together with pronounced annular dilation and severe loss of central coaptation. Quantitatively, severe MR is typically indicated by an ERO of at least 0.40 $cm^2$, regurgitant volume of at least 60 mL, and regurgitant fraction of at least 50%. Associated cardiac findings commonly include marked LV dilation and systolic dysfunction due to the primary myocardial disorder, with regional wall motion abnormalities often persisting in ischemic etiologies. Although the stage is labeled “asymptomatic,” it is clinically important to recognize that patients may still experience symptoms linked to ischemia or heart failure that can appear responsive to revascularization and medical optimization, potentially obscuring the contribution of MR. This stage therefore demands careful clinical correlation, as the absence of classic symptoms does not imply physiologic triviality; rather, it underscores the need to interpret symptoms within the broader context of myocardial disease, functional capacity, and objective evidence of MR severity [22].

Stage D corresponds to symptomatic severe secondary MR and reflects persistent clinical compromise in which heart failure symptoms attributable to MR remain despite correction of reversible ischemia and optimization of medical therapy. The anatomical and hemodynamic profile resembles Stage C, including severe tethering, major annular dilation, and substantial loss of leaflet coaptation, together with severe quantitative indices such as ERO at or above 0.40 $cm^2$, regurgitant volume at or above 60 mL, and regurgitant fraction at or above 50%. What distinguishes Stage D is the presence of ongoing symptomatic limitation—commonly reduced exercise tolerance and exertional dyspnea—where MR is no longer merely an epiphenomenon of myocardial disease but a contributor to elevated left atrial pressures, pulmonary congestion, and diminished forward cardiac output. This stage carries particular management significance because it identifies patients in whom addressing MR may offer

incremental symptomatic and prognostic benefit beyond what can be achieved with revascularization and medical therapy alone. Although these stages provide structured thresholds, MR assessment in secondary disease requires nuanced interpretation, because not all echocardiographic criteria will be simultaneously present in every patient, and severity categorization depends on the quality of data acquisition and the integrated synthesis of multiple parameters alongside other clinical evidence.[3] This principle is especially salient in secondary MR, where regurgitation can be dynamic, varying with preload, afterload, and contractile state. Additionally, the geometry of the regurgitant orifice and the flow convergence region can differ from the more circular assumptions embedded in certain quantitative techniques. Notably, measurement of the proximal isovelocity surface area (PISA) by two-dimensional transthoracic echocardiography (2D TTE) in secondary MR may underestimate the true ERO because the proximal convergence often adopts a crescentic rather than hemispheric shape.[3] This limitation reinforces why secondary MR evaluation should avoid overreliance on a single metric and instead employ a comprehensive approach that reconciles quantitative measures with qualitative imaging features and physiologic plausibility [22].

Echocardiography remains the principal tool for assessing MV structure and function, evaluating systolic competence, and confirming the absence of clinically relevant obstruction during diastole. Beyond measuring regurgitant severity, echocardiography also characterizes leaflet motion patterns that can reveal the dominant mechanism of valve dysfunction (see Table 4. Mitral Valve Pathology Based on Echocardiography).[22] In broad mechanistic terms, Type I motion reflects essentially normal leaflet mobility, where MR arises not from restricted or excessive leaflet movement but from issues such as annular dilation without leaflet tethering, congenital clefts or indentations, or leaflet perforation. Type II motion describes excessive leaflet movement and includes billowing, prolapse, and flail leaflet configurations, patterns more typical of primary MR but still relevant to differential diagnosis when mixed pathology exists. Type III motion encompasses restricted leaflet movement and may be expressed as systolic restriction, either symmetric or asymmetric, as well as combined systolic and diastolic restriction. Symmetric systolic restriction is commonly associated with dilated or ischemic cardiomyopathy and leaflet tethering, whereas asymmetric systolic restriction often reflects segmental ischemia with localized tethering. Combined systolic and diastolic restriction is classically linked to rheumatic disease. Type IV refers to systolic anterior motion, a phenomenon associated with hypertrophic cardiomyopathy or occasionally observed following MV repair. Type V designates mixed conditions, such as prolapse of one leaflet with restriction of another, emphasizing that real-world patients may demonstrate overlapping mechanisms that require individualized interpretation.[22] For the practical echocardiographic description of severe MR, color flow Doppler may demonstrate a jet that is central and large—often exceeding 6 $cm^2$ or more than 30% of the left atrial area—or, alternatively, an eccentric jet that may appear smaller in area yet courses along and may partially encircle the left atrial wall. Additional supportive findings can include pulmonary vein flow abnormalities such as systolic blunting or frank systolic flow reversal, reflecting elevated left atrial pressures and substantial regurgitant burden. Quantitative parameters may include a vena contracta width of at least 0.5 cm when measured in the parasternal long-axis view, regurgitant volume of at least 45 mL per beat, regurgitant fraction of at least 40%, and/or a regurgitant orifice area of at least 0.30 $cm^2$, in accordance with criteria associated with the American College of Cardiology and American Heart Association.[23] While these thresholds provide clinically useful anchors, their interpretation in secondary MR should remain integrative and mechanism-aware, particularly given the potential underestimation issues with 2D PISA and the dependence of MR severity on loading conditions.[3] Overall, staging secondary MR from A through D provides a coherent clinical narrative: myocardial disease establishes risk, remodeling generates progressive tethering and annular dilation, hemodynamic severity increases, and ultimately symptoms persist despite optimal management of the underlying substrate. When applied thoughtfully and supported by comprehensive echocardiographic assessment—including careful attention to leaflet motion patterns and the limitations of certain quantitative methods—this framework enables clinicians to communicate severity consistently, monitor progression systematically, and align therapeutic strategies with both anatomical feasibility and patient-centered outcomes.[3][22][23]

**Preprocedural Anatomical Considerations**

Successful transcatheter edge-to-edge repair (TEER) of the mitral valve is fundamentally contingent on meticulous anatomical assessment and careful patient selection, because the procedure relies on the predictable mechanical capture and sustained approximation of the mitral leaflets. Unlike surgical repair, which permits direct visualization and broad reconstructive options, catheter-based leaflet repair is constrained by device geometry, echocardiographic guidance, and the need to achieve a stable grasp within a moving, three-dimensional structure exposed to high systolic forces. For this reason, preprocedural anatomical considerations are not ancillary details but rather central determinants of procedural feasibility, safety, and durability. A thorough preprocedural evaluation seeks to ensure that leaflet tissue quality, valve geometry, and inflow conditions will allow effective leaflet capture without causing clinically meaningful iatrogenic stenosis, while also identifying anatomic variants that may increase technical complexity or reduce the likelihood of a durable reduction in mitral regurgitation (MR). At the most basic level, effective TEER requires that the target leaflet segments be suitable for mechanical grasping. The leaflets must be sufficiently pliable at the intended grasping site, and the tissue should not be heavily calcified, because rigid calcified regions reduce the ability of the device to capture and retain leaflet tissue and can predispose to suboptimal clip position, leaflet injury, or early device failure. Similarly, the presence of significant leaflet clefts or perforations is problematic because these discontinuities can prevent adequate coaptation even if the device is properly deployed, and they may also interfere with secure leaflet insertion within the device arms. These constraints illustrate why TEER is best conceptualized as a “coaptation-enhancing” intervention: it does not replace missing tissue or reconstruct destroyed leaflet architecture, but rather improves the seal by bringing existing leaflet tissue into stable apposition. Leaflet length, particularly of the posterior leaflet, is another critical prerequisite because adequate tissue insertion is required to prevent detachment or residual regurgitation. Device design influences the minimum tissue requirements. The shorter MitraClip configurations, NT and NTW, require a minimal posterior leaflet length of 6 mm, whereas the longer clip designs, XT and XTW, require a posterior leaflet length of at least 9 mm.[24] This distinction is clinically important, because the selection of device type must align not only with the severity and mechanism of MR, but also with the available leaflet tissue. Longer devices can be advantageous for capturing more tissue and addressing larger coaptation defects, yet they demand longer leaflet length and may increase the risk of chordal interaction in anatomically crowded regions. The interplay between device length and leaflet anatomy therefore becomes a central theme in preprocedural planning: a device may be theoretically optimal for regurgitation reduction but practically infeasible if leaflet length is insufficient [21][22][23].

Equally important is the need to preserve adequate diastolic inflow after repair. Because TEER creates a double-orifice configuration and can reduce the effective mitral valve area, it may increase diastolic transmitral gradients, particularly when multiple devices are required. Consequently, a transmitral gradient below 5 mm Hg and a mitral valve area of at least 4 $cm^2$ are generally desirable to reduce the risk of clinically significant mitral stenosis after the procedure. When the mitral valve area is 3 $cm^2$ or less, TEER is considered contraindicated, reflecting the heightened likelihood that postprocedural obstruction will outweigh the hemodynamic benefit of regurgitation reduction. In cases that fall near these thresholds, proceeding can be individualized based on the location and severity of MR and the anticipated number of devices needed, because the incremental reduction in valve area may vary depending on clip size, number, and placement strategy. Accurate measurement of mitral valve area is therefore essential, and three-dimensional (3D) multiplanar reformatting is emphasized to minimize overestimation errors that can arise from two-dimensional assumptions and oblique imaging planes. Anatomical complexity is especially evident in degenerative MR with flail leaflet pathology, where a segment of leaflet has lost chordal restraint and moves freely, creating a gap that can be challenging to bridge. The initial TEER clinical trials, including EVEREST 2, excluded patients with extensive flail segments, specifically those with a flail width of 15 mm or greater or a flail gap of 10 mm or greater.[10] This exclusion reflected early concerns about feasibility and durability in extreme anatomies, given that the device must capture two leaflets and eliminate a substantial regurgitant orifice. However, degenerative disease with flail segments remains one of the most clinically relevant applications of TEER, particularly because severe degenerative MR in older populations can confer a

higher mortality risk when untreated or when surgery is not feasible.[25] Importantly, the presence of a flail segment has been associated with a greater acute improvement in mean left atrial pressure after TEER, and reductions in left atrial pressure have been linked to improved functional status.[26] These observations support the concept that effectively treating severe regurgitation in flail pathology can yield immediate hemodynamic benefits, even in patients with advanced age or comorbidity, provided that adequate leaflet capture and stable device positioning can be achieved.

Technological evolution has expanded the anatomical range that TEER can address. The availability of longer and wider TEER devices has facilitated treatment of wider flail segments and larger flail gaps by allowing greater tissue capture and improved coaptation enhancement. Moreover, the development of independent leaflet grasping technology has further advanced feasibility in complex degenerative anatomies. This feature, available in systems such as MitraClip G4 and PASCAL, enables operators to capture the flail leaflet segment first and then maneuver to engage the opposing nonflail leaflet, thereby ensuring both leaflets are inserted securely.[24] By decoupling leaflet capture, independent grasping reduces the procedural vulnerability that arises when one leaflet is highly mobile and difficult to engage simultaneously with the other. In practical terms, this innovation can stabilize the repair construct and broaden the range of treatable flail morphologies, though careful imaging and operator expertise remain essential. The anatomic location of the regurgitant jet also strongly influences suitability and complexity. In EVEREST 2, enrollment was restricted to patients with a primary regurgitant jet arising from the central A2–P2 segments.[10] This selection facilitated standardization and aligned with a zone that is relatively accessible and often offers favorable tissue characteristics for grasping. However, the consequence was systematic exclusion of many patients with noncentral MR, leaving a substantial proportion without access to TEER. Noncentral MR, often originating from commissural regions and extending toward leaflet edges, accounts for nearly one-third of significant MR cases.[27][28] These commissural lesions pose distinctive technical challenges. The commissures contain a dense, complex chordal network, and the proximity of chordae increases the risk that device arms may become entangled or that chordae may be disrupted during delivery and positioning. Additionally, prolapsing or flail pathology near the medial or lateral commissures may be more difficult to visualize and orient relative to the coaptation line, increasing the likelihood of malalignment and residual MR. In these noncentral scenarios, device selection and procedural strategy are often adapted to mitigate chordal risk. Some operators prefer smaller or shorter TEER devices because longer arms can increase the chance of entanglement within the crowded commissural chordal architecture. Notably, the posterior leaflet length in commissural regions is often shorter, which can make short device arms adequate for tissue grasping, particularly when leaflet length is below 9 mm and would not support longer-arm devices.[29] The imaging demands also increase substantially. Extensive use of 3D transthoracic echocardiography and unconventional imaging planes can be invaluable for delineating the full extent of commissural pathology, clarifying leaflet scallop involvement, guiding device orientation, and supporting informed selection between device types.[29] The central message is that commissural MR can be treated with TEER, but success depends on specialized imaging strategies and careful procedural planning to avoid chordal complications and ensure a stable grasp.

Another degenerative phenotype that heightens procedural complexity is severe leaflet prolapse associated with Barlow disease. Patients with Barlow anatomy were excluded from the EVEREST trials due to the challenge of obtaining a stable TEER grasp in hypermobile, redundant leaflets.[30] In Barlow disease, the leaflets are often thickened, elongated, and excessively mobile, and multisegment prolapse can create broad regions of malcoaptation rather than a single discrete lesion. These features make leaflet capture technically demanding and can necessitate implantation of multiple large TEER devices to reduce leaflet height and achieve durable regurgitation reduction.[30] However, each additional device can further reduce mitral valve area and increase transmitral gradients, thereby raising the risk of postprocedural stenosis. Thus, Barlow disease exemplifies the trade-off between adequate regurgitation reduction and preservation of diastolic inflow, and it highlights the importance of preprocedural valve area measurement and gradient assessment when multidevice strategies are anticipated. When planning TEER for secondary MR, anatomical considerations extend beyond leaflet characteristics to include the mechanistic substrate of

regurgitation. It is particularly important to distinguish patients with preserved LV function and predominant annular dilation—often described as atrial functional MR—from those with LV dysfunction and leaflet tethering driven by ventricular remodeling. This distinction matters because the feasibility and expected benefit of TEER may differ depending on whether the dominant abnormality is annular enlargement with relatively preserved leaflet mobility or severe tethering with a deep coaptation point. A subgroup analysis of the 2018 Cardiovascular Outcomes Assessment of the MitraClip Percutaneous Therapy for Heart Failure Patients with Functional Mitral Regurgitation trial indicated that patients with atrial fibrillation who underwent TEER maintained clinical benefit, yet their prognosis was worse than that of patients without atrial fibrillation.[31] This observation underscores the concept that rhythm disorders and atrial pathology are not merely comorbidities but can reflect advanced structural disease and hemodynamic burden that influence long-term outcomes even when MR reduction is achieved. Mitral annular calcification introduces another layer of anatomical and procedural complexity. As a degenerative process primarily affecting the mitral annulus, it is frequently associated with MR and can simultaneously reduce annular compliance and impair leaflet motion.[32] In these patients, leaflets may be thickened and stiff, making secure grasping more difficult and increasing the risk of suboptimal insertion. Moreover, mitral annular calcification often coexists with a reduced baseline valve area, which increases susceptibility to high postprocedural diastolic gradients when the valve orifice is further partitioned by TEER devices. Despite these challenges, emerging evidence suggests that in carefully selected patients with annular calcification and severe MR, TEER can be safe and feasible with midterm outcomes that are comparable to those seen in other treated populations.[33] This finding emphasizes that annular calcification is not an absolute barrier but rather a condition that requires heightened attention to valve area, gradient, leaflet quality, and anticipated device number.

A further preprocedural scenario of growing relevance is MR recurrence after prior surgical mitral valve repair. Even at high-volume centers, up to 35% of patients may develop moderate to severe MR a decade after initial surgical repair, reflecting the long-term vulnerability of repaired valves to progressive degenerative change, annular dynamics, or recurrent functional remodeling.[34][35][36] Reoperative sternotomy in these patients often carries substantial morbidity and mortality risks, particularly in older individuals or those with complex comorbidities.[37] In this context, TEER has emerged as a potentially attractive less invasive alternative, and multiple studies have reported feasibility and procedural safety, although definitive evaluation of efficacy and long-term durability remains an active area for further research.[38][39][40] Importantly, prior surgical repair alters anatomy in ways that can directly affect TEER planning, making detailed preprocedural imaging even more essential than in native valves. One key issue in previously repaired valves is the presence of an annuloplasty ring, which can reduce the effective mitral valve area even before any transcatheter device is implanted. Because more than one TEER device is frequently necessary to achieve meaningful MR reduction, procedural teams must carefully anticipate and monitor diastolic inflow gradients to avoid creating clinically significant obstruction. Additionally, postsurgical repairs often involve posterior leaflet resection, leaving a shorter posterior leaflet remnant that can be difficult to grasp securely during TEER. In such cases, an alternative strategy may involve grasping portions of the anterior and posterior sections of the annuloplasty ring when posterior leaflet tissue is insufficient, although experience with this approach remains limited and requires high operator expertise. Imaging challenges also intensify after surgical repair: annuloplasty rings can reduce visualization of the posterior leaflet and shadow important regions on echocardiography, complicating real-time assessment of leaflet insertion and stability. The risk of device entanglement may also be increased, particularly when artificial chords are present, because the device and delivery system may interact unpredictably with prosthetic materials.[41] These considerations highlight that “post-repair” TEER should not be approached as a routine extension of native-valve intervention but rather as a distinct anatomical category requiring tailored imaging protocols and procedural caution.

Because of the central role of anatomy in determining feasibility, preprocedural transesophageal echocardiography (TEE) is frequently used not only to define mechanism and quantify severity, but also to predict the technical difficulty of TEER and to identify features that may represent relative contraindications (see Table 5. Echocardiography Predictors of Transcatheter Edge-

to-Edge Repair Difficulties).[8][42][43] Ideal anatomy typically includes a regurgitant jet localized to the central A2–P2 region and an absence of perforation, cleft, or severe leaflet calcification, whereas involvement of commissural segments or lateral and medial regions such as A1–P1 or A3–P3 is generally more challenging and may require advanced imaging and device strategy adjustments.[8][42][43] Mitral valve area and transmitral gradient are also prominent predictors: a valve area greater than 4 $cm^2$ and gradient below 4 mm Hg are favorable, while borderline values increase concern for postprocedural stenosis, and more restrictive values can function as relative contraindications depending on the balance between anticipated MR reduction and obstruction risk.[8][42][43] Leaflet grasping length is similarly influential, with longer available insertion lengths being ideal and shorter lengths increasing the probability of unstable capture or residual MR, particularly when multidevice implantation is required.[8][42][43] In secondary MR, coaptation depth serves as a practical marker of tethering severity and procedural complexity. Shallow coaptation depths are generally more favorable because they imply less severe leaflet tethering and a coaptation point closer to the annular plane, conditions that facilitate stable grasping and meaningful coaptation enhancement. By contrast, deeper coaptation reflects significant tethering and apical displacement of the coaptation zone, which can limit the ability of the device to approximate leaflets sufficiently and can increase the likelihood of residual MR after implantation.[8][42][43] In primary MR, flail dimensions remain central: smaller flail widths and gaps are more favorable for stable capture, whereas very large flail segments may be feasible only when valve area is sufficiently large and device technology allows secure, independent grasping strategies.[8][42][43] Barlow disease remains an especially challenging anatomy due to multisegment involvement and hypermobility, often requiring multiple devices and heightened vigilance for induced gradients.[30]

Taken together, preprocedural anatomical considerations for mitral TEER can be understood as a balancing act between achieving meaningful regurgitation reduction and preserving adequate diastolic inflow, while minimizing procedural hazards such as chordal entanglement, leaflet injury, or iatrogenic stenosis. This balance is shaped by leaflet tissue quality, leaflet length, flail dimensions, jet location, annular geometry, baseline valve area, transmitral gradient, and the broader context of ventricular and atrial remodeling. Technological advances—particularly longer and wider devices and independent leaflet grasping—have broadened eligibility and improved feasibility in complex anatomies, yet they have not eliminated the foundational requirement for precise imaging and disciplined patient selection.[24] Ultimately, a comprehensive preprocedural evaluation, grounded in detailed echocardiography and informed by established predictors of difficulty, is essential to optimizing outcomes, reducing complications, and ensuring that TEER is applied to patients most likely to benefit.[8][42][43]

**Indications**

Transcatheter edge-to-edge repair (TEER) has become a central catheter-based strategy for the treatment of mitral regurgitation (MR), particularly for patients who are not optimal candidates for conventional surgery. Among currently established catheter therapies, the edge-to-edge leaflet repair approach remains the only widely recommended transcatheter intervention with a substantial evidence base supporting its safety and clinical utility across carefully selected patient populations. At the same time, the field of transcatheter mitral intervention is rapidly evolving, and multiple innovative platforms are under active investigation or early clinical adoption, including—but not limited to—devices designed to implant neo-chords, a variety of transcatheter mitral valve repair concepts, and ring-based technologies intended to modify annular geometry. Within this expanding therapeutic landscape, the indications for TEER reflect a convergence of clinical severity, procedural risk stratification, anatomical suitability, and a realistic expectation of patient-centered benefit, including symptom improvement and meaningful functional recovery. In contemporary practice, TEER is considered in patients with moderate-to-severe primary MR and in those with moderate-to-severe secondary MR, provided that the clinical scenario aligns with guideline-informed thresholds for intervention and that the patient's symptoms and physiologic status suggest that MR is materially contributing to clinical deterioration. The overarching logic behind this indication is that significant MR imposes a chronic volume overload on the left atrium and left ventricle, promotes progressive chamber remodeling, and can precipitate pulmonary congestion and functional limitation. TEER is

therefore contemplated most often in patients who manifest symptomatic heart failure, typically evidenced by exertional dyspnea, reduced exercise tolerance, or recurrent decompensation requiring escalation of diuretic therapy or hospitalization. Symptom burden is especially relevant because TEER is a corrective, procedure-based intervention with inherent procedural risks and resource requirements; thus, the anticipated benefit should be clinically tangible and aligned with the patient's goals of care.

A defining aspect of TEER candidacy is surgical risk. Patients at high or prohibitive risk for surgical mitral valve repair or replacement represent the principal group for whom TEER is most strongly justified, because the transcatheter approach offers a less invasive alternative with the potential to reduce MR and improve symptoms without the physiologic stress of sternotomy and cardiopulmonary bypass. Risk determination is typically multifactorial and incorporates age, frailty, comorbidity burden, prior cardiac surgery, pulmonary disease, renal dysfunction, and other factors that may increase perioperative mortality or morbidity. Importantly, risk assessment is not purely numerical; it is also contextual, integrating clinician judgment, institutional experience, and patient preferences. In addition to high surgical risk, TEER candidates should have favorable anatomy, meaning that the mitral valve structure and lesion characteristics permit stable device implantation, effective leaflet capture, and a clinically meaningful reduction in MR without producing a prohibitive transmitral gradient. Finally, because TEER is intended to provide functional improvement and to reduce adverse sequelae of severe MR, patients are generally expected to have a life expectancy exceeding one year, ensuring that the procedural benefit is not eclipsed by advanced noncardiac disease or terminal comorbid conditions.[44][45] Collectively, these elements form a coherent indication profile: significant MR severity, symptomatic status, excessive surgical risk, anatomical feasibility, and sufficient life expectancy to derive meaningful benefit.[44][45] While the clinical rationale for TEER is compelling in appropriately selected patients, careful attention to contraindications is essential because certain conditions either amplify procedural risk beyond acceptable limits or undermine the likelihood of durable success. A fundamental contraindication is inability to tolerate anticoagulation, given that periprocedural and postprocedural thromboembolic risk management may require anticoagulant therapy depending on patient characteristics, device implantation context, and concomitant indications. Active infective endocarditis of the mitral valve is another critical contraindication, as ongoing infection and tissue destruction can prevent secure device anchoring, heighten the risk of embolic complications, and compromise outcomes. Rheumatic mitral valve disease may also preclude TEER in many cases because leaflet thickening, calcification, and restricted motion can limit adequate grasping and increase the risk of iatrogenic stenosis. Similarly, the presence of intracardiac thrombus, or thrombus in the inferior vena cava or femoral venous system, poses an unacceptable embolic hazard during catheter manipulation and transseptal access. Severe mitral annular calcification involving the leaflets may limit tissue pliability and increase the risk of leaflet injury or incomplete capture, while significant clefts or perforations in the leaflets can prevent effective coaptation despite device implantation. Finally, mitral valve stenosis constitutes a major contraindication because TEER typically reduces effective orifice area and can worsen transmitral gradients, potentially precipitating symptomatic obstruction.[46][47][48] These contraindications collectively emphasize a central principle: TEER is not merely a technically achievable procedure, but one that must be applied where leaflet tissue quality, valvular geometry, and procedural safety parameters align to support a net clinical benefit.[46][47][48]

The logistical and technical demands of TEER require comprehensive procedural equipment that supports safe vascular access, precise transseptal puncture, real-time imaging guidance, hemodynamic monitoring, and readiness for emergent rescue. The edge-to-edge leaflet repair device is the central therapeutic tool, but it is deployed within a broader platform that includes a transseptal puncture kit with catheters, needles, and, in many centers, a radiofrequency wire to facilitate controlled septal crossing in challenging anatomy. Fluoroscopy is essential for device navigation, spatial orientation, and confirmation of catheter positioning, while transesophageal echocardiography—preferably with three-dimensional capability—is indispensable for defining the lesion, guiding device trajectory, optimizing leaflet capture, and verifying reduction of MR before final device release. A code cart with

defibrillation capability is required to address arrhythmias or hemodynamic instability, and invasive hemodynamic monitoring transducers and equipment support real-time assessment of left atrial pressure responses and procedural physiology. The procedural environment also necessitates standard sterile preparation supplies, including sterile gowns and drapes, as well as anesthetic resources, most commonly general anesthesia. Because rare but severe complications such as device embolization or the need for emergent surgical conversion can occur, the availability of a perfusionist and a heart-lung machine is often recommended to ensure prompt initiation of cardiopulmonary bypass if required. Among TEER platforms, the MitraClip system has historically held a foundational role and remains a leading technology with regulatory approvals for both primary and secondary MR. It was the first transcatheter mitral technology to obtain approval from both the U.S. Food and Drug Administration and the Conformité Européenne, reflecting early evidence supporting safety, feasibility, and clinical benefit in selected cohorts.[49][50] By 2020, the fourth-generation MitraClip platform had expanded to offer four implant sizes across two widths and two arm lengths, reflecting an effort to tailor device geometry to lesion complexity and leaflet anatomy.[51] The platform includes a “traditional” 4 mm width as well as a newer 6 mm option, with both widths available in NT configurations featuring a 9 mm arm length and XT configurations featuring a 12 mm arm length. This diversification is clinically meaningful because it enables operators to match device dimensions to leaflet length, coaptation gaps, and flail morphologies, thereby expanding the spectrum of treatable anatomy.

Structurally, MitraClip devices comprise two rigid cobalt–chromium arms and flexible nitinol-based grippers that facilitate leaflet capture. The grippers contain small hooks—often referred to as frictional elements—arranged longitudinally, with four hooks in the NT/NTW variants and six hooks in the XT/XTW variants. The longer-arm XT/XTW devices have particular relevance because they extend beyond the strict anatomical and morphological constraints originally employed in the EVEREST trials, thereby enabling treatment of larger coaptation gaps and more extensive leaflet flails that were previously considered borderline or unsuitable for transcatheter repair.[52] However, expanding TEER to more complex anatomy also raises legitimate mechanistic concerns. Longer and stiffer devices, coupled with active locking mechanisms, can increase leaflet tension when larger amounts of tissue are captured, which may elevate the risk of leaflet injury and single-leaflet device attachment—an adverse outcome in which only one leaflet remains secured after implantation. Such tension-related risks may become more pronounced in anatomies with calcified or fragile leaflets, where tissue resilience is reduced and stress distribution is less forgiving.[52] These considerations highlight the necessity of aligning device choice not merely with the size of the coaptation defect, but with tissue quality and predicted stress responses. Importantly, registry-level evidence has helped contextualize these concerns. A comprehensive analysis of the EXPAND registry did not demonstrate higher rates of adverse leaflet events associated with the long-arm XTR system compared with the smaller NTR device, suggesting that, in real-world practice and with appropriate selection and technique, longer-arm devices can be used without an inevitable increase in leaflet complications.[24] Furthermore, the fourth-generation MitraClip platform provides an advantage through autonomous and controlled gripper actuation, allowing operators to confirm and refine leaflet gripping before final release, while enabling continuous left atrial pressure monitoring through the guiding catheter. These features support procedural precision by combining mechanical control with physiologic feedback, which is particularly valuable in complex MR where incremental reductions can meaningfully change left atrial pressures and pulmonary congestion. Another prominent TEER technology is the PASCAL transcatheter mitral valve repair system, first introduced in 2016 and initially evaluated in a compassionate-use cohort of 23 patients characterized by anatomies considered challenging for conventional edge-to-edge repair.[53] The system has continued to evolve, and its second version integrates three catheters: a 22 Fr steerable guide sheath, a maneuverable catheter, and an implant catheter with the device preattached at the distal end. This configuration is designed to enhance steerability and expand the range of motion within the left atrium, which is especially relevant when navigating complex jet locations, broad prolapse, or commissural pathology.

The PASCAL P10 implant is constructed from nitinol and incorporates a central spacer flanked by two curved, spring-loaded paddles. When opened to 180 degrees, it offers a gripping length of 26 mm, along with two clasps measuring 10 mm each. The

central spacer is a distinctive design element intended to occupy the coaptation defect in the region of the primary MR jet, thereby reducing the degree of leaflet stress required to achieve an effective seal. This stress-reducing rationale is particularly pertinent in degenerative anatomy, where leaflets may be redundant but delicate, or in functional MR, where tethering forces oppose closure. The nitinol clasps contain a horizontal line of small hooks—referred to as retention elements—near the distal end, and, crucially, these clasps can be adjusted independently, enabling either simultaneous or staged leaflet capture. A smaller device, the PASCAL Ace, preserves a comparable gripping breadth while narrowing paddle width to 6 mm, making it potentially advantageous in smaller anatomies and allowing multiple implant techniques. Both PASCAL variants support separate leaflet gripping, permitting "leaflet optimization" strategies or staged capture, which can be valuable when one leaflet is more mobile, restricted, or otherwise difficult to engage. In August 2022, the second-generation PASCAL Precision platform was introduced with refinements to the catheter system intended to improve device stability and steerability, underscoring the iterative nature of device development in response to procedural experience.[54] Because TEER is a complex structural heart intervention, procedural success depends not only on devices and imaging but also on coordinated multidisciplinary personnel. Core procedural staff typically include an interventional cardiologist as the primary operator, an echocardiographer who may be a cardiac anesthesiologist or cardiologist with advanced structural imaging expertise, and a cardiac anesthesiologist to manage general anesthesia and ensure stable conditions for high-quality transesophageal echocardiography. A first assistant supports procedural execution, while nursing and technical staff manage sterile preparation, equipment handling, imaging coordination, and hemodynamic monitoring. A cardiac surgeon and operating room team should be available on standby to address rare but critical complications requiring emergent surgical intervention, and a perfusionist may be necessary if cardiopulmonary bypass is required. This team-based structure reflects the high-stakes nature of structural interventions and the principle that rapid response capacity improves patient safety.

Preparation for TEER typically occurs in a catheterization laboratory or a hybrid operating room equipped with fluoroscopy, and the procedure relies on real-time echocardiographic guidance, most prominently transesophageal echocardiography (TEE). TEE is pivotal not only for confirming the underlying pathology but also for guiding transseptal puncture, steering the delivery system, aligning the device perpendicular to the line of coaptation, ensuring adequate leaflet insertion, and confirming reduction of MR prior to device release. Given the need for uninterrupted imaging quality and avoidance of patient movement, TEER is most commonly performed under general anesthesia, which facilitates stable TEE imaging and reduces procedural hazard associated with sudden motion. Before catheter manipulation begins, a preoperative TEE examination is performed to define lesion anatomy, quantify MR, and assess the feasibility of repair; in select cases, additional cardiac imaging may be required and should be conducted by clinicians with specialized expertise in cardiovascular intervention or imaging to ensure optimal interpretation and procedural relevance. A dedicated anesthetic evaluation is essential to optimize the patient for general anesthesia and to minimize perioperative complications. Because TEER requires close coordination among multiple disciplines, structured collaboration within a structural heart team is critical, encompassing the interventional cardiologist, cardiac anesthesiologist, echocardiography personnel, operating room or cath-lab staff, and nursing team. Effective preparation also demands verification that all devices, backup equipment, and emergency resources are immediately available. Accordingly, many programs incorporate a formal preprocedural time-out to confirm patient identity, procedural plan, imaging strategy, anticipated device selection, anticoagulation plan, and availability of rescue resources, thereby reducing communication failures and improving procedural reliability. Finally, sterility is an indispensable component of TEER preparation, reflecting the procedure's invasive nature and the significant consequences of bloodstream infection or device-associated endocarditis. A sterile field must be established according to catheterization standards, and the vascular access site is prepared with thorough antisepsis consistent with institutional protocols. Personnel operating near the sterile field are expected to adhere strictly to aseptic technique, including appropriate surgical scrubbing and the use of full sterile attire—gowns, masks, hats, and gloves. The procedural area is cleaned and draped to maintain an

aseptic environment throughout catheter insertion, transseptal access, and device deployment, thereby protecting patient safety and aligning with best practices for infection prevention.[55] In sum, the indications for TEER encompass a carefully delineated clinical profile centered on significant MR severity, symptomatic burden, high or prohibitive surgical risk, favorable anatomy, and sufficient life expectancy to benefit from intervention.[44][45] Contraindications emphasize scenarios where infection, thrombosis, stenosis risk, or severe structural leaflet abnormalities undermine either procedural safety or likelihood of success.[46][47][48] The evolving TEER device ecosystem—exemplified by iterative advances in MitraClip and the development of PASCAL platforms—reflects a broader trend toward tailored catheter solutions capable of addressing increasingly complex anatomy, while underscoring the ongoing need for robust imaging, multidisciplinary expertise, and rigorous procedural preparation to ensure safe and durable outcomes.[24][49][50][51][52][53][54][55]

**Device Selection**

Device selection for transcatheter edge-to-edge repair (TEER) is a technically consequential step that integrates anatomical feasibility, hemodynamic safety, and the anticipated repair strategy into a coherent procedural plan. With the expanding availability of TEER platforms and implant geometries, contemporary selection has moved beyond a simple preference for one system over another and instead emphasizes a tailored match between device characteristics and the patient's mitral valve morphology. When three-dimensional echocardiography (3DE) is employed—particularly three-dimensional transesophageal imaging—it becomes possible to evaluate the mitral valve in a manner that closely reflects its true spatial complexity. In this context, careful preprocedural appraisal of MR etiology, baseline mitral valve area (MVA), mean transmitral gradient, and anatomical complexity is essential before choosing a specific implant. The overarching objective is to achieve durable reduction in MR while preserving adequate diastolic inflow and minimizing complications such as single leaflet device attachment, leaflet injury, chordal interaction, or iatrogenic mitral stenosis. Because TEER modifies the valve orifice by creating a double-orifice configuration and by drawing leaflet tissue toward the coaptation line, device selection must be made with explicit awareness of how implant geometry and placement will influence both regurgitant reduction and valve area. A central anatomical variable is the length of the leaflet grasping zone, which defines how much leaflet tissue can be safely captured and retained. Devices with shorter arms are generally favored when the available leaflet length is limited, because they can achieve stable capture without requiring excessive insertion depth. As reflected in commonly applied selection criteria (see Table 6. Mitral Valve Criteria for Device Selection), when the leaflet grasping zone is less than 9 mm, shorter-arm options such as NT and NTW are typically considered suitable, and PASCAL P10 and PASCAL ACE can also be appropriate given their ability to accommodate variable leaflet capture strategies. By contrast, when the grasping zone exceeds 9 mm, longer-arm implants such as XT and XTW become more feasible and may offer advantages in bridging broader coaptation defects; PASCAL devices also remain applicable in this setting. These relationships highlight that selection is not merely a function of device availability but of tissue geometry and the mechanical requirements for stable coaptation enhancement.

Certain degenerative phenotypes, particularly Barlow disease, introduce additional selection pressures. Barlow anatomy often features redundant, hypermobile leaflets with multisegment involvement, and achieving meaningful height reduction and durable MR control may require implants with extended reach and robust coaptation engagement. In many such cases, longer-arm MitraClip configurations (XT, XTW) and PASCAL devices are considered more appropriate because they are better suited to address broad prolapse and large coaptation gaps, and they can engage larger volumes of leaflet tissue. Conversely, in valves with thin leaflet structures, there is a premium on minimizing tissue stress while maintaining stable capture, a balance that can support the use of NT and NTW devices as well as PASCAL implants. In practice, thin leaflet tissue demands careful imaging confirmation of leaflet insertion and tension distribution, because excessive traction can predispose to leaflet injury or destabilization even when initial grasp appears satisfactory. Another determinant of device choice is the breadth of the regurgitant gap and the anticipated need for stabilization across a wide prolapse or significant flail. In anatomies characterized by broad gap size, selection criteria commonly favor wider devices, such as NTW or XTW, and may also favor PASCAL P10 when its design characteristics are

advantageous for filling the coaptation defect. The logic is that a broader device footprint can improve the probability of capturing sufficient tissue to reduce MR effectively, particularly when the regurgitant orifice is large or when leaflet malcoaptation spans a wide region. At the same time, a broader device may increase the likelihood of mitral orifice reduction and a rise in transmitral gradients, especially when multiple implants are used. Thus, broad-gap anatomies require a careful balance between efficacy and inflow preservation, and they often elevate the importance of baseline MVA quantification. Commissural jets represent a distinct anatomical scenario with specific selection considerations. Commissural regions contain dense chordal architecture and may provide shorter posterior leaflet lengths, both of which influence device choice. Selection criteria frequently favor smaller-arm devices such as NT and NTW for commissural lesions, and in some settings the PASCAL ACE may be preferred due to its suitability for smaller anatomies and its implant maneuverability. The objective of overarching in commissural MR is to reduce the risk of chordal entanglement and to maintain precise orientation and steering in a challenging region. Longer-arm devices may increase the chance of interaction with chordae tendineae in the commissures, thereby raising procedural risk. Consequently, the combination of smaller arm length and dependable steering capability is commonly prioritized when the regurgitant jet is localized to commissural segments.

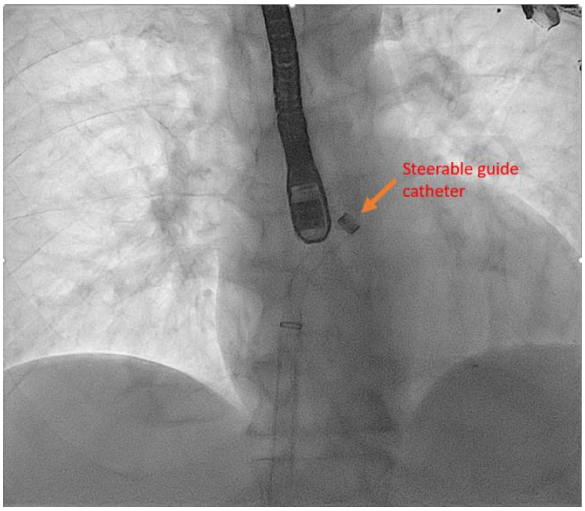


**Fig. 3:** MitraClip and Steerable Guide Catheter.

Baseline mitral valve area is a recurring theme because TEER inherently reduces effective valve orifice size. Accurate MVA quantification ideally relies on multiplanar reconstruction from high-resolution 3D volumes, rather than two-dimensional estimates that may over- or underestimate the true orifice due to nonplanar geometry. Reductions in MVA after implantation can be substantial, and device choice should incorporate this expected change. Observational data suggest that deploying a PASCAL P10 device can reduce MVA by approximately 47%, while NTR and XTR implants have been associated with reductions of about 52% and 57%, respectively.[56] These values emphasize that device geometry and stiffness can have meaningful hemodynamic consequences, and they underscore the necessity of selecting an implant that achieves sufficient MR reduction without crossing a threshold into clinically significant mitral stenosis. Importantly, the magnitude of MVA reduction is not determined solely by device type; it is also influenced by device location along the line of coaptation. Placement at A2/P2 has been associated with the most pronounced reduction in MVA, whereas commissural placement tends to produce the least reduction.[56] This anatomic dependence reinforces that device selection cannot be separated from implantation strategy: the same implant may be tolerable in one position and problematic in another when baseline valve area is borderline. Two additional considerations that strongly influence device selection in transcatheter mitral valve repair are the overall treatment strategy and the localization of the regurgitant jet. For example, when MR presents as discrete jets and the anticipated strategy involves implanting two spatially separated clips, the baseline MVA must typically be larger to preserve adequate residual orifice area after repair. In this context, a baseline MVA around 6 $cm^2$ has been suggested to help prevent the development of clinically significant mitral stenosis when two distant implants are planned.[56] This principle highlights the cumulative effect of multiple devices: even if each implant is individually well positioned and reduces MR effectively, the combined impact on diastolic inflow can become clinically limiting if the baseline orifice is not sufficiently large. In anatomies with large flail gaps or wide prolapse, especially when multiple implants are required to stabilize leaflet motion and achieve durable reduction in regurgitation, devices with extended arms—such as XTW, XT, or PASCAL platforms—have demonstrated increased effectiveness in reducing MR.[57] The mechanistic rationale is straightforward: extended arms and larger grasping

surfaces can capture more leaflet tissue, bridge broader coaptation defects, and create a more stable double-orifice configuration in the presence of significant malcoaptation. However, device design may constrain multi-implant strategies. When a multiple-clip approach is anticipated, PASCAL P10 is generally not recommended because the concave design of its paddles can complicate the precise alignment of two implants, potentially increasing the risk of malorientation or interference between devices.[57] This limitation does not negate the value of PASCAL P10 in appropriate anatomies, but it illustrates why selection must consider not only the first implant but the entire procedural plan, including whether sequential implantation is likely.

For isolated commissural lesions, the procedural emphasis typically shifts toward maximizing steerability and minimizing the risk of chordal interaction. In such cases, implants with smaller arms—NT or NTW—are often preferred, particularly when their steering characteristics facilitate precise alignment perpendicular to the coaptation line in a confined region.[57] This approach aligns with the practical reality that commissural pathology often provides limited leaflet length and increased chordal density; therefore, smaller, more maneuverable devices can be safer while still achieving adequate tissue grasp. The selection choice in commissural MR thus reflects a risk-mitigation framework rather than solely an efficacy framework, aiming to reduce procedural hazards while still accomplishing meaningful MR reduction. Beyond gross anatomical measures, careful evaluation of leaflet tissue thickness and length is indispensable. Leaflets that are short, thin, or tethered—particularly in secondary MR—can be vulnerable to excessive tension and injury when longer-arm, more rigid devices are used. When annular calcification with leaflet infiltration is identified, it may predict higher transmitral gradients after TEER, and this finding can favor selection of smaller and more flexible devices to reduce the likelihood of excessive obstruction and to improve conformability at the grasp site.[26][58] Similarly, in secondary MR where the posterior leaflet may be short and tethered, avoiding extended-arm MitraClip devices such as XT and XTW can be prudent to reduce the risk of single leaflet device attachment or leaflet injury, events that can occur when leaflet capture is marginal and stress concentrations are high. In such settings, PASCAL devices are frequently preferred because their nitinol construction confers flexibility and their horizontal gripping orientation can concentrate capture forces closer to the leaflet base, often described as the “hinge point” near the mitral annulus.[54] This base-oriented gripping may be advantageous when leaflet free-edge mobility is restricted or when insertion length is limited, enabling a stable capture without requiring excessive traction on fragile or tethered leaflet tissue. Overall, device selection in TEER is an integrative exercise that depends on high-quality 3DE characterization of valve anatomy and hemodynamics, careful prediction of postprocedural gradients and orifice area reduction, and alignment of implant geometry with both lesion localization and the intended treatment strategy. The evidence that different implants can produce substantial MVA reductions, modulated by device position along the coaptation line, underscores the need for precise anatomical planning and individualized decision-making.[56] Similarly, the recognition that extended-arm devices may be more effective for large flail gaps yet may be less suitable in short posterior leaflet tethering highlights how selection must be mechanism-specific, not merely device-driven.[54][57] By integrating leaflet grasping zone length, tissue quality, jet location, baseline MVA, and anticipated number and placement of implants, clinicians can optimize the balance between MR reduction and preservation of diastolic inflow, thereby maximizing the likelihood of a safe, durable, and clinically meaningful repair.[26][54][56][57][58]

**Technique or Treatment**

**The Edge-to-Edge Leaflet Repair Device**

Transcatheter mitral valve repair using an edge-to-edge leaflet repair device has evolved into a highly standardized structural heart procedure that depends on coordinated multidisciplinary expertise, advanced imaging, and precise catheter manipulation within the left atrium and left ventricle. The intervention is typically performed by a structural heart team that integrates complementary competencies, most notably those of the interventional cardiologist, cardiac surgeon, cardiac anesthesiologist, and operating room nurse, with additional support from echocardiography specialists and technical staff. This team-based model is not merely organizational; it reflects the procedural reality that success requires simultaneous interpretation of hemodynamic signals, real-time transesophageal echocardiography (TEE) guidance, and fluoroscopic spatial orientation, all while maintaining readiness for rare but serious

complications that may require emergent surgical backup. Procedural suites are therefore purpose-built environments, generally configured as catheterization laboratories or hybrid operating rooms equipped with high-quality fluoroscopy and the ability to support continuous TEE imaging before, during, and after device implantation.[59] Because patient immobility is critical to procedural safety and imaging fidelity, the procedure is usually performed under general anesthesia, which supports airway control, minimizes motion artifact, and enables consistent TEE probe positioning across all phases of the intervention. The procedural workflow can be conceptualized as a sequence of interdependent steps, each guided by a set of echocardiographic windows and each associated with distinct technical objectives and potential complications. Although the steps are commonly taught in a linear fashion, in practice they are iterative: operators repeatedly reassess leaflet anatomy, device alignment, and hemodynamics to confirm that the evolving repair remains both effective and physiologically tolerable. The initial phase is preprocedural cardiac assessment and detailed mitral valve evaluation by TEE, which functions as the definitive confirmation of pathology and as the principal determinant of procedural feasibility.[60] At this stage, the team must validate the MR mechanism, quantify baseline severity, and exclude contraindications such as intracardiac thrombus, which could pose catastrophic embolic risk if disturbed during transseptal access or left atrial catheter manipulation. A comprehensive TEE examination typically includes standard midesophageal four-chamber and two-chamber views, modified bicaval views, midesophageal long-axis imaging, and left atrial appendage–focused views. These views are combined with color Doppler and pulsed-wave Doppler interrogation of the left atrial appendage to support thrombus exclusion and to characterize appendage flow. In addition, the interatrial septum is evaluated for anatomical suitability for transseptal puncture, including assessment of septal thickness, mobility, and the location of the fossa ovalis. The baseline evaluation also incorporates Doppler gradients across the mitral valve, measurement of the mitral valve area (MVA), assessment for pericardial effusion, and analysis of pulmonary vein Doppler profiles, which can provide supportive evidence of severe MR through systolic blunting or reversal.[61] Echocardiographic features associated with favorable procedural conditions include adequate coaptation length and limited coaptation depth, as well as flail dimensions below common thresholds, such as a flail gap under 10 mm and flail width under 15 mm.[9] Importantly, documenting any pre-existing pericardial effusion and quantifying it before instrumentation is critical, because new or increasing effusion after transseptal puncture may signal perforation or evolving tamponade, necessitating immediate recognition and response.

Following confirmation of feasibility and safety prerequisites, vascular access is established, most commonly through femoral venous cannulation. This step is fundamental because the entire transseptal and left-sided catheter course depends on reliable venous access capable of accommodating large-bore sheaths and delivery systems. Ultrasound guidance is commonly used to optimize puncture location, reduce inadvertent arterial access, and limit access-related bleeding. Wire advancement is continuously tracked under fluoroscopy to prevent kinking, malposition, or unintended vessel injury, and assessment of vessel caliber is essential to ensure compatibility with the chosen sheath and guide catheter system. Venous compressibility and color Doppler imaging can help exclude femoral venous thrombus before large devices are introduced, thereby reducing embolic risk. Some operators employ a micropuncture technique followed by progressive upsizing and the use of percutaneous suture-mediated closure devices to support hemostasis at the conclusion of the procedure. Echocardiographically, transgastric inferior vena cava (IVC) views in short and long axis may help visualize the wire as it advances into the IVC and right atrium, complementing fluoroscopy in confirming safe intravascular positioning. The access phase is associated with complications that are largely vascular, including bleeding at the puncture site, retroperitoneal hemorrhage from high puncture, femoral arterial injury, and injury to surrounding structures, underscoring the importance of careful access technique and surveillance. The transseptal puncture is one of the most technically and physiologically consequential phases of the TEER procedure because it establishes the trajectory through which the steerable guide and device delivery systems will operate. The puncture is typically performed in the posterior-superior portion of the interatrial septum to provide an optimal working height and maneuverability within the left

atrium. Under TEE, the transseptal needle tip often appears as tenting or indentation of the septum, and operators must confirm appropriate anterior–posterior and superior–inferior orientation as well as the vertical height above the mitral annular plane before crossing. A commonly targeted puncture height is approximately 4 to 5 cm above the annulus, reflecting a balance between sufficient working space and the ability to direct the device below the leaflets for capture. A puncture that is too low may compromise maneuverability and may force operators to work closer to the ventricle, increasing the risk of chordal entanglement; conversely, a puncture that is too high can restrict the ability to pass beneath the leaflets and can make grasping more difficult. Height selection can also be adapted to jet location, with higher puncture heights often favored for medial jets and lower heights used for lateral jets.[62] Throughout this phase, de-airing of the delivery system is essential to reduce the risk of air embolism, and a radiofrequency transseptal needle may be considered when the septum is unusually thick, fibrotic, lipomatous, or excessively mobile. Real-time TEE monitoring is indispensable to ensure that the needle is not directed toward the aorta or the posterior left atrial wall, both of which could result in catastrophic injury. Orthogonal plane imaging and wide-sector full-volume views can further enhance spatial awareness. Systemic anticoagulation is typically instituted with a target activated clotting time (ACT) above 250 seconds, with serial monitoring every 15 to 30 minutes to mitigate thrombus formation risk on large-bore catheters in the left atrium. Potential complications at this stage include aortic root or aortic valve injury from an overly anterior puncture, cardiac tamponade from perforation, and air embolism, making this step a focal point for vigilance and coordinated team communication.

After successful septal crossing, the steerable guide catheter (SGC) is introduced into the left atrium, creating the stable conduit through which the clip delivery system will be advanced. In many workflows, an extra-stiff guidewire such as an Amplatz wire is positioned in the left upper pulmonary vein under combined TEE and fluoroscopic guidance to provide support. The SGC and dilator are then advanced over this wire, with echocardiographic identification aided by recognizing the cone-shaped dilator tip and the radiopaque double-ring signature of the guide catheter. Once the SGC is positioned appropriately in the left atrium, the wire and dilator are withdrawn, leaving the guide catheter as the primary working channel. Real-time three-dimensional imaging—often from aortic valve short-axis or mitral commissural perspectives—helps the team assess the spatial relationship of the guide catheter and delivery system within the left atrium, ensuring that the trajectory is compatible with subsequent alignment over the mitral valve and that atrial wall contact is minimized. The introduction of the clip delivery system into the left atrium and subsequent positioning above the mitral valve represents the transition from access mechanics to precision valve therapy. The delivery system is advanced through the SGC under continuous TEE and fluoroscopic guidance.[63] Achieving the desired position typically requires coordinated maneuvers that include posterior torque of the SGC, medial deflection of the delivery system, and controlled retraction of the entire system to center it above the valve. Throughout this process, alignment adjustments are made in both medial–lateral and anterior–posterior planes, with the device tip directed toward the largest regurgitant region. Real-time 3D imaging is used to verify that the delivery system is appropriately located within the left atrium and is approaching the mitral valve along a trajectory that will allow perpendicular crossing of the coaptation line. This phase carries risks such as atrial wall injury, arrhythmias, tamponade, damage to the mitral apparatus, and air embolism, especially if catheter motion is excessive or if the system contacts delicate atrial structures. Axial alignment of the clip is a critical determinant of procedural success because effective leaflet capture requires that the device arms be oriented perpendicular to the mitral coaptation line. Achieving this orientation depends on both fluoroscopic markers and echocardiographic visualization. The midesophageal mitral commissural view is commonly used to define medial and lateral orientation, while the midesophageal long-axis view clarifies anterior–posterior alignment. Three-dimensional en face visualization of the mitral valve further assists by providing a surgical-like view of the valve plane and the MR jet origin, enabling iterative fine-tuning of device position. Real-time 3D en face imaging is particularly valuable for guiding the transition from the left atrial side through the mitral orifice into the left ventricle, as it helps operators maintain perpendicularity and avoid rotational misalignment that could yield partial leaflet capture or residual MR.

Advancement of the device into the left ventricle and the leaflet grasping process constitute

the most delicate phase of the intervention, because the device must pass below the leaflets without injuring the subvalvular apparatus and must capture both the anterior and posterior leaflets securely. Continuous visualization of the device tip is emphasized to reduce the risk of entanglement or traumatic contact. The device should remain perpendicular to both the regurgitant orifice and the coaptation line, and its position relative to the MR jet and leaflet edges must be confirmed as it crosses into the ventricle. Ensuring that no entanglement exists within the chordae tendineae is crucial, because chordal disruption can worsen MR and complicate rescue strategies. In select cases, rapid pacing or brief ventilatory holds may be employed to reduce cardiac motion and improve precision during leaflet capture. Imaging frequently relies on 3D en face views from both atrial and ventricular perspectives, alongside biplane imaging that combines commissural and long-axis views with and without color Doppler. Fluoroscopy complements echocardiography by confirming arm orientation and device stability. Complications at this stage include arrhythmias, direct injury to the mitral valve, and device or catheter entanglement within the subvalvular apparatus. Once leaflet capture is achieved, assessment of capture adequacy and device deployment requires integration of echocardiographic and fluoroscopic data to confirm sufficient leaflet insertion, absence of excessive restriction, and meaningful MR reduction. Excessive manipulation at this stage is avoided because repeated re-crossing and rotation can increase the risk of entanglement and subvalvular injury. In situations where the coaptation defect is broad, strategies such as “zip and clip” may be considered, in which the first device is deployed immediately adjacent to the dominant defect to facilitate subsequent leaflet grasping across a wider region. Rapid ventricular pacing or controlled ventilation holds may again be used to reduce motion and improve stability during final grasp optimization.[43] In parallel, the team monitors for spontaneous echo contrast and maintains attention to ACT values to prevent thrombus formation in the left atrium. With simultaneous two-dimensional commissural and long-axis imaging, operators can estimate leaflet length within the device arms and assess whether leaflet motion has become excessively restricted. Fluoroscopy confirms device position and stability before release. Adverse outcomes at this stage include device detachment, injury to the mitral apparatus, leaflet injury producing severe MR not amenable to further percutaneous repair, and the development of mitral stenosis due to excessive reduction in orifice area.

After deployment, a structured post-deployment assessment is performed to confirm both effectiveness and safety. The team evaluates complications, verifies device stability and durable leaflet capture, and quantifies residual MR and any increase in transmitral gradients. A transmitral which mean gradient below 5 mm Hg is commonly targeted to reduce the risk of clinically relevant iatrogenic mitral stenosis. Imaging typically combines simultaneous two-dimensional commissural and long-axis views with three-dimensional en face visualization from both left atrial and left ventricular perspectives, and Doppler assessment is used to quantify gradients and residual regurgitation. If significant residual MR persists, an additional device may be required, and this decision again must balance regurgitation reduction against the risk of increased gradients and stenosis. The final procedural phase involves withdrawal of the delivery system and the SGC and completion of vascular hemostasis. At this time, evaluation for an iatrogenic atrial septal defect (ASD) becomes important, as transseptal access can leave a persistent septal communication. Heparin is commonly reversed with protamine to support hemostasis, while monitoring protamine reaction. Femoral venous access sites are closely observed for bleeding, and a complete postprocedure echocardiographic assessment is performed to document repair success and rule out complications. Standard views include midesophageal four-chamber, commissural, and long-axis imaging with and without color Doppler, pulmonary venous flow assessment to corroborate MR reduction, continuous-wave Doppler to evaluate for stenosis, and three-dimensional en face imaging to confirm device stability and leaflet grip. Three-dimensional reconstruction and multiplanar analysis can be used to assess the area of the repaired double-orifice valve, while bicaval and 3D imaging of the interatrial septum aid in assessing iatrogenic ASD.[64][65][66] Potential complications during withdrawal include tamponade, iatrogenic ASD, injury to the IVC or femoral veins, and reactions to protamine. Postprocedural monitoring typically occurs in a post-anesthesia care unit or intensive care setting, where surveillance focuses on respiratory complications, postoperative nausea and vomiting, hemodynamic instability, tamponade, and

bleeding from the femoral site. This monitoring phase is essential because certain complications may evolve after the procedure, and early recognition facilitates prompt intervention. Across the entire procedural arc, the consistent theme is that TEER is an imaging-driven therapy in which success depends on the integration of TEE and fluoroscopy, disciplined attention to anticoagulation and sterility, and coordinated team performance within a specialized procedural environment.[59]

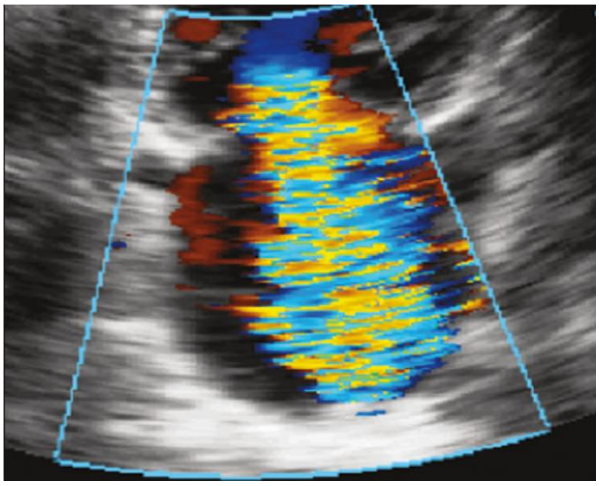

**Fig. 4:** Mitral Valve, Echocardiograph.

**Complications**

Transcatheter edge-to-edge repair (TEER) has become widely established as a comparatively safe and effective method for treating mitral regurgitation (MR), particularly in patients who frequently present with advanced age, frailty, and multiple comorbid conditions that increase the risk of conventional surgical intervention. Notwithstanding this high-risk clinical backdrop, contemporary experience demonstrates that TEER is associated with a relatively low probability of major adverse events, reflecting both maturation of procedural technique and improvements in device design and imaging guidance. Nevertheless, the procedure is not without risk, and a comprehensive understanding of potential complications is essential for appropriate patient selection, informed consent, procedural preparedness, and postprocedural surveillance. Complications may arise from device–leaflet interaction, unintended alteration of mitral valve hemodynamics, vascular access and transseptal manipulation, embolic phenomena, and the broader physiological consequences of abruptly reducing a chronic regurgitant lesion. Among device-related complications, single leaflet device attachment (SLDA) is one of the most recognized adverse events, occurring in approximately 1.5% to 5.1% of cases.[11][67] SLDA typically refers to loss of sustained attachment to one leaflet after initial deployment, which can result in recurrent or residual MR and may precipitate hemodynamic deterioration depending on the severity of regurgitation and the patient's baseline reserve. Mechanistically, SLDA can reflect inadequate leaflet insertion at deployment, excessive leaflet tension, leaflet fragility, or progressive tissue injury. The risk may be accentuated in anatomies where leaflet quality is compromised, including long-standing secondary MR in which chronic tethering and altered stress distribution may render leaflet tissue susceptible to tearing. This concern becomes even more salient when calcification is present, because stiffened or infiltrated leaflets may not conform well to device grasping and may concentrate mechanical forces at localized points, increasing the propensity for detachment or tear.

Leaflet injury, encompassing perforation or tearing, has been reported at rates ranging from 0% to 2%.[51][52] Although numerically uncommon, this complication is clinically consequential because it can convert a treatable lesion into severe MR that is not readily amenable to additional percutaneous repair, thereby necessitating urgent surgical consultation or leaving the patient with persistent hemodynamic compromise if surgery is not feasible. The relationship between device choice and leaflet injury has been a subject of concern, particularly with longer or stiffer implants that may increase leaflet tension. As procedural practice has expanded to include more complex anatomies, careful intraprocedural imaging confirmation of insertion depth, leaflet mobility, and stress response has become central to minimizing these events. Device embolization is a rare but potentially catastrophic complication, occurring in roughly 0.05% to 0.7% of procedures.[11][68] Embolization may result from failure of stable leaflet capture, device malposition, or late detachment, and it carries risks related to obstruction, end-organ ischemia, or the need for urgent retrieval. Percutaneous retrieval of embolized devices can be technically challenging, particularly when larger clip configurations are involved, because retrieval requires precise snaring and controlled extraction without causing vascular or intracardiac injury.[70] The potential difficulty of retrieval underscores why procedural teams must maintain readiness for emergent escalation, including surgical backup capability, and why meticulous attention to leaflet insertion and device stability prior to release is

considered non-negotiable. From a hemodynamic standpoint, an elevated transmitral gradient following TEER represents a clinically important adverse outcome because it reflects iatrogenic narrowing of the mitral orifice, potentially leading to symptomatic mitral stenosis. A postprocedural transmitral gradient exceeding 5 mmHg has been reported in up to 15% of cases.[52] This phenomenon is strongly influenced by baseline mitral valve area, annular and leaflet characteristics, and the number and position of implanted devices. It is particularly relevant when multiple implants are required to control MR, as cumulative reduction in the effective orifice area can compromise diastolic filling and raise left atrial pressures. Consequently, careful preprocedural measurement of mitral valve area and intraprocedural gradient monitoring are pivotal in balancing MR reduction against stenosis risk.

Residual MR is another common outcome with important clinical implications, typically defined as more than moderate regurgitation after intervention. Rates of residual MR greater than 2+ have ranged from approximately 3.4% to 17.0%.[51][52] Residual MR may arise from incomplete coverage of the regurgitant orifice, suboptimal device position, complex multi-jet anatomy, or progressive ventricular remodeling in secondary MR that continues to distort the valve apparatus even after initial repair. Clinically, persistent MR may blunt the symptomatic benefit of TEER and may be associated with worse long-term outcomes compared with more complete reduction, particularly in patients with limited physiologic reserve. Residual MR also introduces the potential need for additional interventions, including placement of another device, redo TEER, or, in selected cases, surgical reintervention. Complications related to cardiac perforation and pericardial fluid accumulation, including tamponade, are infrequent in contemporary series, with pericardial effusion or tamponade reported at rates of 0% to 0.5%.[69] When these events occur, they often relate to transseptal puncture, catheter manipulation within the atrium, or accidental injury to cardiac structures. Because tamponade can evolve rapidly and become life-threatening, procedural and postoperative monitoring must include vigilance for hemodynamic instability, rising pericardial effusion on imaging, and clinical deterioration. Rapid recognition and pericardiocentesis capability are therefore essential elements of institutional readiness for TEER programs. Vascular access remains a nontrivial source of morbidity, particularly given the large-bore venous sheaths used for TEER. Major vascular complications have been reported in approximately 1.4% to 4.0% of cases.[69] These complications may include access-site bleeding, hematoma, pseudoaneurysm formation, arteriovenous fistula, or, less commonly, retroperitoneal hemorrhage from high puncture or vessel injury. Severe bleeding requiring blood transfusion has been described across a broad range, from 0% to 17%.[69] Variability in bleeding rates may reflect differences in patient baseline risk, anticoagulation management, access technique, and institutional definitions of “major bleeding.” Regardless, bleeding risk highlights the importance of ultrasound-guided access, careful anticoagulation monitoring, appropriate reversal when indicated, and systematic postprocedural site surveillance.

Thromboembolic and ischemic complications, while uncommon, remain critical because of their potential to cause permanent disability or death. Stroke rates have been reported between 0% and 1%.[69] Potential mechanisms include embolization of thrombus formed on catheters or within the left atrium, dislodgement of pre-existing thrombus, or air embolism. Myocardial infarction rates ranging from 0% to 3% have been reported, potentially related to hemodynamic instability, coronary embolization, or stress-induced ischemia in patients with advanced coronary disease.[69] These events reinforce the rationale for strict attention to anticoagulation targets during left atrial catheter dwell time, meticulous de-airing of systems, and comprehensive baseline assessment for intracardiac thrombus. Beyond these more commonly tabulated procedural complications, TEER can precipitate physiological responses that require careful interpretation and management. Afterload mismatch is a recognized phenomenon, particularly in patients with reduced LV function. In chronic severe MR, the regurgitant orifice provides a low-impedance pathway during systole, effectively reducing LV afterload. When MR is suddenly reduced, the ventricle may confront a higher effective afterload, potentially revealing limited contractile reserve and causing transient declines in forward output. Although afterload mismatch is generally infrequent and often transient—frequently managed with inotropic support and not requiring mechanical circulatory assistance—it may signal advanced heart failure physiology and has been suggested to

adversely affect longer-term outcomes in some patients, reflecting a late stage of ventricular disease.[71] In a subset of patients with severely impaired LV function, thrombus formation within the left atrium or ventricle may occur, potentially reflecting stasis, altered flow patterns, and reduced contractility; in such cases, early and intensified anticoagulation may be considered to mitigate thromboembolic risk.[71]

Management decisions become particularly complex when residual or recurrent MR is identified after TEER. In these circumstances, the multidisciplinary team must reassess whether the patient should undergo surgery, repeat transcatheter intervention, or optimized medical therapy, with the decision informed by MR severity, symptom burden, ventricular function, and procedural feasibility. Repeat TEE is typically warranted to clarify the mechanism of failure, characterize residual leaflet anatomy that may support additional device implantation, and evaluate the risk of creating clinically significant mitral stenosis if another implant is placed. In selected case series where safety data are limited, alternative catheter-based approaches have been explored for substantial para-clip or inter-clip residual MR. Examples include occlusion using an Amplatzer vascular plug and the use of an expanded polytetrafluoroethylene double-disk occluder originally developed for atrial septal defect closure.[38][72] While such approaches are not broadly standardized, their existence underscores the need for creative problem-solving in complex failures and the importance of careful anatomical assessment before proceeding. Registry data provide additional context regarding the prognostic significance of device-related failure. In a large multicenter registry, implant failure due to leaflet perforation, tear, or loss occurred in approximately 3.5% of patients and was associated with increased in-hospital and long-term mortality.[73] This association does not necessarily imply direct causality in all cases, as implant failure may also mark a subgroup with more complex anatomy, more advanced disease, or more limited physiological reserve. Nonetheless, the finding emphasizes that procedural complications are not merely acute events but can have sustained implications for survival and long-term outcomes. Within this framework, redo TEER has emerged as a viable strategy and may be preferable to surgery in anatomically suitable patients with primary or secondary MR, particularly when surgical outcomes are predicted to be suboptimal or when surgical risk remains prohibitive.[74] The feasibility of redo TEER reinforces the principle that careful imaging-driven assessment can identify opportunities for iterative transcatheter optimization, although the risk of increased transmitral gradients and complex device interactions must be weighed carefully.

The clinical significance of catheter-based MR management extends beyond complication profiles, as TEER represents a major innovation that has expanded corrective options for patients with severe MR who previously had limited therapeutic alternatives due to surgical risk. Some contemporary studies suggest that catheter management may, in specific circumstances, compare favorably with surgical intervention.[20] From a physiological perspective, following successful edge-to-edge repair, LV contractility and cardiac output may remain stable, while total ejection fraction and global strain can decrease. This apparent paradox is often interpreted as a reflection of reduced regurgitant volume after repair, which lowers LV end-diastolic volume and thereby diminishes the contribution of regurgitant flow to measured ejection fraction. Importantly, this reduction in volume load can lower myocardial oxygen demand and has been associated with improvement in New York Heart Association functional class within several months after the procedure.[75] These observations highlight that post-TEER metrics must be interpreted in the context of altered loading conditions and should be aligned with clinical improvement rather than isolated reliance on ejection fraction changes. Optimizing outcomes and minimizing complications in TEER depends heavily on interprofessional team performance. Catheter management of MR requires coordinated, patient-centered care spanning preprocedural evaluation, intraprocedural execution, and longitudinal follow-up. Physicians—particularly cardiovascular interventionalists—direct the procedural strategy and integrate imaging and hemodynamic data, while advanced practitioners support comprehensive assessment, patient counseling, and continuity of care. Nurses with cardiology expertise play a central role in patient preparation, intraoperative monitoring, early recognition of complications, postprocedural surveillance, patient education, and coordination of follow-up visits and imaging. Cardiovascular imaging specialists, structural heart professionals, and anesthesiologists contribute specialized expertise that enhances procedural precision, optimizes

cardiopulmonary stability, and ensures safe delivery of anesthesia in often fragile patients. Pharmacists provide essential consultation regarding anticoagulation strategies, antiplatelet considerations, pain control, antiemetics, and medication reconciliation, which can be particularly complex in heart failure populations with polypharmacy. Effective communication among these professionals is not incidental; it is a key safety mechanism that reduces preventable errors, improves response to complications, and supports consistent application of evidence-based practices across the procedural pathway.

In summary, while TEER is generally associated with low rates of major complications despite being performed in high-risk populations, the procedure carries a spectrum of device-related, hemodynamic, vascular, and thromboembolic risks that demand rigorous preparation and vigilant monitoring.[11][51][52][67][68][69] Recognizing patient-specific vulnerabilities—such as calcified leaflets in long-standing secondary MR, reduced LV function predisposing to afterload mismatch, or the complexity of managing residual MR—supports more precise selection and tailored procedural strategies.[70][71] When complications occur, outcomes are optimized through multidisciplinary reassessment, repeat imaging, and individualized selection among reintervention options, including redo TEER when anatomically feasible.[73][74] Ultimately, the safe delivery of TEER and the maximization of its clinical benefits depend on both technical excellence and interprofessional collaboration across the continuum of care.[20][75]

**Conclusion:**

Transcatheter edge-to-edge repair has revolutionized the management of severe MR in patients at high or prohibitive surgical risk. By leveraging advanced imaging, device innovation, and multidisciplinary expertise, TEER provides a safe and effective alternative to conventional surgery. Its success hinges on meticulous patient selection, guided by anatomical feasibility and hemodynamic thresholds, as well as comprehensive preprocedural planning to mitigate risks such as iatrogenic stenosis and leaflet injury. Nursing professionals play a pivotal role throughout the care continuum—ensuring optimal preparation, maintaining procedural sterility, monitoring anticoagulation, and detecting early complications. Postprocedural care emphasizes hemodynamic stability, vascular site management, and patient education to support recovery and adherence to follow-up protocols. While TEER demonstrates favorable outcomes in terms of symptom relief and reduced hospitalization, challenges remain, including residual MR, device-related complications, and anatomical limitations in complex cases such as Barlow disease or severe annular calcification. Future directions will likely involve expanded device platforms, improved imaging modalities, and refined patient selection algorithms to enhance durability and broaden applicability. Ultimately, TEER exemplifies the paradigm shift toward minimally invasive, patient-centered interventions in structural heart disease, underscoring the critical interplay between technology, clinical judgment, and collaborative care.

**References:**

1. Iung B, Baron G, Butchart EG, Delahaye F, Gohlke-Bärwolf C, Levang OW, Tornos P, Vanoverschelde JL, Vermeer F, Boersma E, Ravaud P, Vahanian A. A prospective survey of patients with valvular heart disease in Europe: The Euro Heart Survey on Valvular Heart Disease. European heart journal. 2003 Jul:24(13):1231-43
2. Quader N, Rigolin VH. Two and three dimensional echocardiography for pre-operative assessment of mitral valve regurgitation. Cardiovascular ultrasound. 2014 Oct 25:12():42. doi: 10.1186/1476-7120-12-42.
3. Writing Committee Members, Otto CM, Nishimura RA, Bonow RO, Carabello BA, Erwin JP 3rd, Gentile F, Jneid H, Krieger EV, Mack M, McLeod C, O'Gara PT, Rigolin VH, Sundt TM 3rd, Thompson A, Toly C, ACC/AHA Joint Committee Members, O'Gara PT, Beckman JA, Levine GN, Al-Khatib SM, Armbruster A, Birtcher KK, Ciggaroa J, Deswal A, Dixon DL, Fleisher LA, de Las Fuentes L, Gentile F, Goldberger ZD, Gorenek B, Haynes N, Hernandez AF, Hlatky MA, Joglar JA, Jones WS, Marine JE, Mark D, Palaniappan L, Piano MR, Spatz ES, Tamis-Holland J, Wijeysundera DN, Woo YJ. 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. The Journal of thoracic and cardiovascular surgery. 2021 Aug:162(2):e183-e353. doi: 10.1016/j.jtcvs.2021.04.002.

4. Guarracino F, Baldassarri R, Ferro B, Giannini C, Bertini P, Petronio AS, Di Bello V, Landoni G, Alfieri O. Transesophageal echocardiography during MitraClip® procedure. Anesthesia and analgesia. 2014 Jun:118(6):1188-96. doi: 10.1213/ANE.0000000000000215.
5. Vahanian A, Beyersdorf F, Praz F, Milojevic M, Baldus S, Bauersachs J, Capodanno D, Conradi L, De Bonis M, De Paulis R, Delgado V, Freemantle N, Gilard M, Haugaa KH, Jeppsson A, Jüni P, Pierard L, Prendergast BD, Sádaba JR, Tribouilloy C, Wojakowski W, ESC/EACTS Scientific Document Group. 2021 ESC/EACTS Guidelines for the management of valvular heart disease: Developed by the Task Force for the management of valvular heart disease of the European Society of Cardiology (ESC) and the European Association for Cardio-Thoracic Surgery (EACTS). Revista espanola de cardiologia (English ed.). 2022 Jun:75(6):524. doi: 10.1016/j.rec.2022.05.006.
6. Kang DH, Kim JH, Rim JH, Kim MJ, Yun SC, Song JM, Song H, Choi KJ, Song JK, Lee JW. Comparison of early surgery versus conventional treatment in asymptomatic severe mitral regurgitation. Circulation. 2009 Feb 17:119(6):797-804. doi: 10.1161/CIRCULATIONAHA.108.802314.
7. Sidebotham DA, Allen SJ, Gerber IL, Fayers T. Intraoperative transesophageal echocardiography for surgical repair of mitral regurgitation. Journal of the American Society of Echocardiography : official publication of the American Society of Echocardiography. 2014 Apr:27(4):345-66. doi: 10.1016/j.echo.2014.01.005.
8. Bonow RO, O'Gara PT, Adams DH, Badhwar V, Bavaria JE, Elmariah S, Hung JW, Lindenfeld J, Morris AA, Satpathy R, Whisenant B, Woo YJ. 2020 Focused Update of the 2017 ACC Expert Consensus Decision Pathway on the Management of Mitral Regurgitation: A Report of the American College of Cardiology Solution Set Oversight Committee. Journal of the American College of Cardiology. 2020 May 5:75(17):2236-2270. doi: 10.1016/j.jacc.2020.02.005.
9. Feldman T, Kar S, Rinaldi M, Fail P, Hermiller J, Smalling R, Whitlow PL, Gray W, Low R, Herrmann HC, Lim S, Foster E, Glower D, EVEREST Investigators. Percutaneous mitral repair with the MitraClip system: safety and midterm durability in the initial EVEREST (Endovascular Valve Edge-to-Edge REpair Study) cohort. Journal of the American College of Cardiology. 2009 Aug 18:54(8):686-94. doi: 10.1016/j.jacc.2009.03.077.
10. Feldman T, Kar S, Elmariah S, Smart SC, Trento A, Siegel RJ, Apruzzese P, Fail P, Rinaldi MJ, Smalling RW, Hermiller JB, Heimansohn D, Gray WA, Grayburn PA, Mack MJ, Lim DS, Ailawadi G, Herrmann HC, Acker MA, Silvestry FE, Foster E, Wang A, Glower DD, Mauri L, EVEREST II Investigators. Randomized Comparison of Percutaneous Repair and Surgery for Mitral Regurgitation: 5-Year Results of EVEREST II. Journal of the American College of Cardiology. 2015 Dec 29:66(25):2844-2854. doi: 10.1016/j.jacc.2015.10.018.
11. Feldman T, Foster E, Glower DD, Kar S, Rinaldi MJ, Fail PS, Smalling RW, Siegel R, Rose GA, Engeron E, Loghin C, Trento A, Skipper ER, Fudge T, Letsou GV, Massaro JM, Mauri L, EVEREST II Investigators. Percutaneous repair or surgery for mitral regurgitation. The New England journal of medicine. 2011 Apr 14:364(15):1395-406. doi: 10.1056/NEJMoa1009355.
12. Alfieri O, Maisano F, De Bonis M, Stefano PL, Torracca L, Oppizzi M, La Canna G. The double-orifice technique in mitral valve repair: a simple solution for complex problems. The Journal of thoracic and cardiovascular surgery. 2001 Oct:122(4):674-81
13. Mack MJ, Lindenfeld J, Abraham WT, Kar S, Lim DS, Mishell JM, Whisenant BK, Grayburn PA, Rinaldi MJ, Kapadia SR, Rajagopal V, Sarembock IJ, Brieke A, Rogers JH, Marx SO, Cohen DJ, Weissman NJ, Stone GW, COAPT Investigators. 3-Year Outcomes of Transcatheter Mitral Valve Repair in Patients With Heart Failure. Journal of the American College of Cardiology. 2021 Mar 2:77(8):1029-1040. doi: 10.1016/j.jacc.2020.12.047.
14. Khatib D, Neuburger PJ, Pan S, Rong LQ. Transcatheter Mitral Valve Interventions for Mitral Regurgitation: A Review of Mitral Annuloplasty, Valve Replacement, and Chordal Repair Devices. Journal of cardiothoracic and vascular anesthesia. 2022 Oct:36(10):3887-3903. doi: 10.1053/j.jvca.2022.05.005.
15. De Backer O, Piazza N, Banai S, Lutter G, Maisano F, Herrmann HC, Franzen OW,

Søndergaard L. Percutaneous transcatheter mitral valve replacement: an overview of devices in preclinical and early clinical evaluation. Circulation. Cardiovascular interventions. 2014 Jun:7(3):400-9. doi: 10.1161/CIRCINTERVENTIONS.114.001607.

16. Ooms JF, Van Mieghem NM. Transcatheter Repair and Replacement Technologies for Mitral Regurgitation: a European Perspective. Current cardiology reports. 2021 Jul 16:23(9):125. doi: 10.1007/s11886-021-01556-6.
17. Condado JA, Vélez-Gimón M. Catheter-based approach to mitral regurgitation. Journal of interventional cardiology. 2003 Dec:16(6):523-34
18. Perloff JK, Roberts WC. The mitral apparatus. Functional anatomy of mitral regurgitation. Circulation. 1972 Aug:46(2):227
19. Blanke P, Naoum C, Webb J, Dvir D, Hahn RT, Grayburn P, Moss RR, Reisman M, Piazza N, Leipsic J. Multimodality Imaging in the Context of Transcatheter Mitral Valve Replacement: Establishing Consensus Among Modalities and Disciplines. JACC. Cardiovascular imaging. 2015 Oct:8(10):1191-1208. doi: 10.1016/j.jcmg.2015.08.004.
20. El Sabbagh A, Reddy YNV, Nishimura RA. Mitral Valve Regurgitation in the Contemporary Era: Insights Into Diagnosis, Management, and Future Directions. JACC. Cardiovascular imaging. 2018 Apr:11(4):628-643. doi: 10.1016/j.jcmg.2018.01.009.
21. Dziadzko V, Clavel MA, Dziadzko M, Medina-Inojosa JR, Michelena H, Maalouf J, Nkomo V, Thapa P, Enriquez-Sarano M. Outcome and undertreatment of mitral regurgitation: a community cohort study. Lancet (London, England). 2018 Mar 10:391(10124):960-969. doi: 10.1016/S0140-6736(18)30473-2.
22. Shah PM, Raney AA. Echocardiography in mitral regurgitation with relevance to valve surgery. Journal of the American Society of Echocardiography : official publication of the American Society of Echocardiography. 2011 Oct:24(10):1086-91. doi: 10.1016/j.echo.2011.08.017.
23. Shiota T. Role of echocardiography for catheter-based management of valvular heart disease. Journal of cardiology. 2017 Jan:69(1):66-73. doi: 10.1016/j.jjcc.2016.09.015.
24. Asch FM, Little SH, Mackensen GB, Grayburn PA, Sorajja P, Rinaldi MJ, Maisano F, Kar S. Incidence and standardised definitions of mitral valve leaflet adverse events after transcatheter mitral valve repair: the EXPAND study. EuroIntervention : journal of EuroPCR in collaboration with the Working Group on Interventional Cardiology of the European Society of Cardiology. 2021 Dec 3:17(11):e932-e941. doi: 10.4244/EIJ-D-21-00012.
25. Avierinos JF, Tribouilloy C, Grigioni F, Suri R, Barbieri A, Michelena HI, Ionico T, Rusinaru D, Ansaldi S, Habib G, Szymanski C, Giorgi R, Mahoney DW, Enriquez-Sarano M, Mitral regurgitation International DAtabase (MIDA) Investigators. Impact of ageing on presentation and outcome of mitral regurgitation due to flail leaflet: a multicentre international study. European heart journal. 2013 Sep:34(33):2600-9. doi: 10.1093/eurheartj/eht250.
26. Thaden JJ, Malouf JF, Nkomo VT, Pislaru SV, Holmes DR Jr, Reeder GS, Rihal CS, Eleid MF. Mitral Valve Anatomic Predictors of Hemodynamic Success With Transcatheter Mitral Valve Repair. Journal of the American Heart Association. 2018 Jan 13:7(2):. doi: 10.1161/JAHA.117.007315.
27. Estévez-Loureiro R, Franzen O, Winter R, Sondergaard L, Jacobsen P, Cheung G, Moat N, Ihlemann N, Ghione M, Price S, Duncan A, Streit Rosenberg T, Barker S, Di Mario C, Settergren M. Echocardiographic and clinical outcomes of central versus noncentral percutaneous edge-to-edge repair of degenerative mitral regurgitation. Journal of the American College of Cardiology. 2013 Dec 24:62(25):2370-2377. doi: 10.1016/j.jacc.2013.05.093.
28. Lapenna E, De Bonis M, Sorrentino F, La Canna G, Grimaldi A, Torracca L, Maisano F, Alfieri O. Commissural closure for the treatment of commissural mitral valve prolapse or flail. The Journal of heart valve disease. 2008 May:17(3):261-6
29. Katz WE, Conrad Smith AJ, Crock FW, Cavalcante JL. Echocardiographic evaluation and guidance for MitraClip procedure. Cardiovascular diagnosis and therapy. 2017 Dec:7(6):616-632. doi: 10.21037/cdt.2017.07.04.
30. Khan F, Winkel M, Ong G, Brugger N, Pilgrim T, Windecker S, Praz F, Fam N. Percutaneous

Mitral Edge-to-Edge Repair: State of the Art and a Glimpse to the Future. Frontiers in cardiovascular medicine. 2019:6():122. doi: 10.3389/fcvm.2019.00122.

31. Gertz ZM, Herrmann HC, Lim DS, Kar S, Kapadia SR, Reed GW, Puri R, Krishnaswamy A, Gersh BJ, Weissman NJ, Asch FM, Grayburn PA, Kosmidou I, Redfors B, Zhang Z, Abraham WT, Lindenfeld J, Stone GW, Mack MJ. Implications of Atrial Fibrillation on the Mechanisms of Mitral Regurgitation and Response to MitraClip in the COAPT Trial. Circulation. Cardiovascular interventions. 2021 Apr:14(4):e010300. doi: 10.1161/CIRCINTERVENTIONS.120.010300.
32. Abramowitz Y, Jilaihawi H, Chakravarty T, Mack MJ, Makkar RR. Mitral Annulus Calcification. Journal of the American College of Cardiology. 2015 Oct 27:66(17):1934-41. doi: 10.1016/j.jacc.2015.08.872.
33. Fernández-Peregrina E, Pascual I, Freixa X, Tirado-Conte G, Estévez-Loureiro R, Carrasco-Chinchilla F, Benito-González T, Asmarats L, Sanchís L, Jiménez-Quevedo P, Avanzas P, Caneiro-Queija B, Molina-Ramos AI, Fernández-Vázquez F, Li CH, Flores-Umanzor E, Sans-Roselló J, Nombela-Franco L, Arzamendi D. Transcatheter edge-to-edge mitral valve repair in patients with mitral annulus calcification. EuroIntervention : journal of EuroPCR in collaboration with the Working Group on Interventional Cardiology of the European Society of Cardiology. 2022 Mar 18:17(16):1300-1309. doi: 10.4244/EIJ-D-21-00205.
34. Flint N, Raschpichler M, Rader F, Shmueli H, Siegel RJ. Asymptomatic Degenerative Mitral Regurgitation: A Review. JAMA cardiology. 2020 Mar 1:5(3):346-355. doi: 10.1001/jamacardio.2019.5466.
35. Flameng W, Herijgers P, Bogaerts K. Recurrence of mitral valve regurgitation after mitral valve repair in degenerative valve disease. Circulation. 2003 Apr 1:107(12):1609-13
36. Kim JH, Lee SH, Joo HC, Youn YN, Yoo KJ, Chang BC, Lee S. Effect of Recurrent Mitral Regurgitation After Mitral Valve Repair in Patients With Degenerative Mitral Regurgitation. Circulation journal : official journal of the Japanese Circulation Society. 2017 Dec 25:82(1):93-101. doi: 10.1253/circj.CJ-17-0380.
37. Mehaffey HJ, Hawkins RB, Schubert S, Fonner C, Yarboro LT, Quader M, Speir A, Rich J, Kron IL, Ailawadi G. Contemporary outcomes in reoperative mitral valve surgery. Heart (British Cardiac Society). 2018 Apr:104(8):652-656. doi: 10.1136/heartjnl-2017-312047.
38. Niikura H, Bae R, Gössl M, Lin D, Jay D, Sorajja P. Transcatheter therapy for residual mitral regurgitation after MitraClip therapy. EuroIntervention : journal of EuroPCR in collaboration with the Working Group on Interventional Cardiology of the European Society of Cardiology. 2019 Aug 9:15(6):e491-e499. doi: 10.4244/EIJ-D-18-01148.
39. Braun D, Frerker C, Körber MI, Gaemperli O, Patzelt J, Schaefer U, Hammerstingl C, Boekstegers P, Ott I, Ince H, Thiele H, Hausleiter J. Percutaneous Edge-to-Edge Repair of Recurrent Severe Mitral Regurgitation After Surgical Mitral Valve Repair. Journal of the American College of Cardiology. 2017 Jul 25:70(4):504-505. doi: 10.1016/j.jacc.2017.05.045.
40. Grasso C, Ohno Y, Attizzani GF, Cannata S, Immè S, Barbanti M, Pistritto AM, Ministeri M, Caggegi A, Chiarandà M, Dipasqua F, Ronsivalle G, Mangiafico S, Scandura S, Capranzano P, Capodanno D, Tamburino C. Percutaneous mitral valve repair with the MitraClip system for severe mitral regurgitation in patients with surgical mitral valve repair failure. Journal of the American College of Cardiology. 2014 Mar 4:63(8):836-8. doi: 10.1016/j.jacc.2013.09.045.
41. Kanda BS, Jay D, Farivar RS, Sorajja P. Leaflet-to-Annuloplasty Ring Clipping for Severe Mitral Regurgitation. JACC. Cardiovascular interventions. 2016 Apr 11:9(7):e63-4. doi: 10.1016/j.jcin.2015.12.272.
42. Wu IY, Barajas MB, Hahn RT. The MitraClip Procedure-A Comprehensive Review for the Cardiac Anesthesiologist. Journal of cardiothoracic and vascular anesthesia. 2018 Dec:32(6):2746-2759. doi: 10.1053/j.jvca.2018.05.020.
43. Hahn RT. Transcathether Valve Replacement and Valve Repair: Review of Procedures and Intraprocedural Echocardiographic Imaging. Circulation research. 2016 Jul 8:119(2):341-56. doi: 10.1161/CIRCRESAHA.116.307972.
44. Hahn RT, Chan V, Adams DH. Current Indications for Transcatheter Edge-to-Edge

Repair in a Patient With Primary Mitral Regurgitation. Circulation. 2022 Oct 25:146(17):1263-1265. doi: 10.1161/CIRCULATIONAHA.122.061495.

45. Lee G, Chikwe J, Milojevic M, Wijeysundera HC, Biondi-Zoccai G, Flather M, Gaudino MFL, Fremes SE, Tam DY. ESC/EACTS vs. ACC/AHA guidelines for the management of severe aortic stenosis. European heart journal. 2023 Mar 7:44(10):796-812. doi: 10.1093/eurheartj/ehac803.
46. Russell EA, Walsh WF, Costello B, McLellan AJA, Brown A, Reid CM, Tran L, Maguire GP. Medical Management of Rheumatic Heart Disease: A Systematic Review of the Evidence. Cardiology in review. 2018 Jul/Aug:26(4):187-195. doi: 10.1097/CRD.0000000000000185.
47. Van Praet KM, Stamm C, Sündermann SH, Meyer A, Unbehaun A, Montagner M, Nazari Shafti TZ, Jacobs S, Falk V, Kempfert J. Minimally Invasive Surgical Mitral Valve Repair: State of the Art Review. Interventional cardiology (London, England). 2018 Jan:13(1):14-19. doi: 10.15420/icr.2017:30:1.
48. Taramasso M, Gaemperli O, Maisano F. Treatment of degenerative mitral regurgitation in elderly patients. Nature reviews. Cardiology. 2015 Mar:12(3):177-83. doi: 10.1038/nrcardio.2014.210.
49. Asch FM, Grayburn PA, Siegel RJ, Kar S, Lim DS, Zaroff JG, Mishell JM, Whisenant B, Mack MJ, Lindenfeld J, Abraham WT, Stone GW, Weissman NJ, COAPT Investigators. Echocardiographic Outcomes After Transcatheter Leaflet Approximation in Patients With Secondary Mitral Regurgitation: The COAPT Trial. Journal of the American College of Cardiology. 2019 Dec 17:74(24):2969-2979. doi: 10.1016/j.jacc.2019.09.017.
50. Nishimura RA, Vahanian A, Eleid MF, Mack MJ. Mitral valve disease--current management and future challenges. Lancet (London, England). 2016 Mar 26:387(10025):1324-34. doi: 10.1016/S0140-6736(16)00558-4.
51. Chakravarty T, Makar M, Patel D, Oakley L, Yoon SH, Stegic J, Singh S, Skaf S, Nakamura M, Makkar RR. Transcatheter Edge-to-Edge Mitral Valve Repair With the MitraClip G4 System. JACC. Cardiovascular interventions. 2020 Oct 26:13(20):2402-2414. doi: 10.1016/j.jcin.2020.06.053.
52. Praz F, Braun D, Unterhuber M, Spirito A, Orban M, Brugger N, Brinkmann I, Spring K, Moschovitis A, Nabauer M, Blazek S, Pilgrim T, Thiele H, Lurz P, Hausleiter J, Windecker S. Edge-to-Edge Mitral Valve Repair With Extended Clip Arms: Early Experience From a Multicenter Observational Study. JACC. Cardiovascular interventions. 2019 Jul 22:12(14):1356-1365. doi: 10.1016/j.jcin.2019.03.023.
53. Praz F, Spargias K, Chrissoheris M, Büllesfeld L, Nickenig G, Deuschl F, Schueler R, Fam NP, Moss R, Makar M, Boone R, Edwards J, Moschovitis A, Kar S, Webb J, Schäfer U, Feldman T, Windecker S. Compassionate use of the PASCAL transcatheter mitral valve repair system for patients with severe mitral regurgitation: a multicentre, prospective, observational, first-in-man study. Lancet (London, England). 2017 Aug 19:390(10096):773-780. doi: 10.1016/S0140-6736(17)31600-8.
54. Hausleiter J, Stocker TJ, Adamo M, Karam N, Swaans MJ, Praz F. Mitral valve transcatheter edge-to-edge repair. EuroIntervention : journal of EuroPCR in collaboration with the Working Group on Interventional Cardiology of the European Society of Cardiology. 2023 Jan 23:18(12):957-976. doi: 10.4244/EIJ-D-22-00725.
55. Huang EY, Chen C, Abdullah F, Aspelund G, Barnhart DC, Calkins CM, Cowles RA, Downard CD, Goldin AB, Lee SL, St Peter SD, Arca MJ, 2011 American Pediatric Surgical Association Outcomes and Clinical Trials Committee. Strategies for the prevention of central venous catheter infections: an American Pediatric Surgical Association Outcomes and Clinical Trials Committee systematic review. Journal of pediatric surgery. 2011 Oct:46(10):2000-11. doi: 10.1016/j.jpedsurg.2011.06.017.
56. Kassar M, Praz F, Hunziker L, Pilgrim T, Windecker S, Seiler C, Brugger N. Anatomical and Technical Predictors of Three-Dimensional Mitral Valve Area Reduction After Transcatheter Edge-To-Edge Repair. Journal of the American Society of Echocardiography : official publication of the American Society of Echocardiography. 2022 Jan:35(1):96-104. doi: 10.1016/j.echo.2021.08.021.

57. Singh GD, Smith TW, Rogers JH. Multi-MitraClip therapy for severe degenerative mitral regurgitation: "anchor" technique for extremely flail segments. Catheterization and cardiovascular interventions : official journal of the Society for Cardiac Angiography & Interventions. 2015 Aug:86(2):339-46. doi: 10.1002/ccd.25811.
58. Oguz D, Padang R, Rashedi N, Pislaru SV, Nkomo VT, Mankad SV, Malouf JF, Guerrero M, Reeder GS, Eleid MF, Rihal CS, Thaden JJ. Risk for Increased Mean Diastolic Gradient after Transcatheter Edge-to-Edge Mitral Valve Repair: A Quantitative Three-Dimensional Transesophageal Echocardiographic Analysis. Journal of the American Society of Echocardiography : official publication of the American Society of Echocardiography. 2021 Jun:34(6):595-603.e2. doi: 10.1016/j.echo.2021.01.018.
59. Naqvi TZ. Echocardiography in percutaneous valve therapy. JACC. Cardiovascular imaging. 2009 Oct:2(10):1226-37. doi: 10.1016/j.jcmg.2009.08.004.
60. Hahn RT, Saric M, Faletra FF, Garg R, Gillam LD, Horton K, Khalique OK, Little SH, Mackensen GB, Oh J, Quader N, Safi L, Scalia GM, Lang RM. Recommended Standards for the Performance of Transesophageal Echocardiographic Screening for Structural Heart Intervention: From the American Society of Echocardiography. Journal of the American Society of Echocardiography : official publication of the American Society of Echocardiography. 2022 Jan:35(1):1-76. doi: 10.1016/j.echo.2021.07.006.
61. Lancellotti P, Tribouilloy C, Hagendorff A, Popescu BA, Edvardsen T, Pierard LA, Badano L, Zamorano JL, Scientific Document Committee of the European Association of Cardiovascular Imaging. Recommendations for the echocardiographic assessment of native valvular regurgitation: an executive summary from the European Association of Cardiovascular Imaging. European heart journal. Cardiovascular Imaging. 2013 Jul:14(7):611-44. doi: 10.1093/ehjci/jet105.
62. Alkhouli M, Rihal CS, Holmes DR Jr. Transseptal Techniques for Emerging Structural Heart Interventions. JACC. Cardiovascular interventions. 2016 Dec 26:9(24):2465-2480. doi: 10.1016/j.jcin.2016.10.035.
63. Sherif MA, Paranskaya L, Yuecel S, Kische S, Thiele O, D'Ancona G, Neuhausen-Abramkina A, Ortak J, Ince H, Öner A. MitraClip step by step; how to simplify the procedure. Netherlands heart journal : monthly journal of the Netherlands Society of Cardiology and the Netherlands Heart Foundation. 2017 Feb:25(2):125-130. doi: 10.1007/s12471-016-0930-7.
64. Swaans MJ, Van den Branden BJ, Van der Heyden JA, Post MC, Rensing BJ, Eefting FD, Plokker HW, Jaarsma W. Three-dimensional transoesophageal echocardiography in a patient undergoing percutaneous mitral valve repair using the edge-to-edge clip technique. European journal of echocardiography : the journal of the Working Group on Echocardiography of the European Society of Cardiology. 2009 Dec:10(8):982-3. doi: 10.1093/ejechocard/jep101.
65. Maslow A, Mahmood F, Poppas A, Singh A. Three-dimensional echocardiographic assessment of the repaired mitral valve. Journal of cardiothoracic and vascular anesthesia. 2014 Feb:28(1):11-17. doi: 10.1053/j.jvca.2013.05.007.
66. Saitoh T, Izumo M, Furugen A, Tanaka J, Miyata-Fukuoka Y, Gurudevan SV, Tolstrup K, Siegel RJ, Kar S, Shiota T. Echocardiographic evaluation of iatrogenic atrial septal defect after catheter-based mitral valve clip insertion. The American journal of cardiology. 2012 Jun 15:109(12):1787-91. doi: 10.1016/j.amjcard.2012.02.023.
67. Writing Committee Members, Otto CM, Nishimura RA, Bonow RO, Carabello BA, Erwin JP 3rd, Gentile F, Jneid H, Krieger EV, Mack M, McLeod C, O'Gara PT, Rigolin VH, Sundt TM 3rd, Thompson A, Toly C. 2020 ACC/AHA Guideline for the Management of Patients With Valvular Heart Disease: Executive Summary: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Journal of the American College of Cardiology. 2021 Feb 2:77(4):450-500. doi: 10.1016/j.jacc.2020.11.035.
68. Nickenig G, Estevez-Loureiro R, Franzen O, Tamburino C, Vanderheyden M, Lüscher TF,

Moat N, Price S, Dall'Ara G, Winter R, Corti R, Grasso C, Snow TM, Jeger R, Blankenberg S, Settergren M, Tiroch K, Balzer J, Petronio AS, Büttner HJ, Ettori F, Sievert H, Fiorino MG, Claeys M, Ussia GP, Baumgartner H, Scandura S, Alamgir F, Keshavarzi F, Colombo A, Maisano F, Ebelt H, Aruta P, Lubos E, Plicht B, Schueler R, Pighi M, Di Mario C, Transcatheter Valve Treatment Sentinel Registry Investigators of the EURObservational Research Programme of the European Society of Cardiology. Percutaneous mitral valve edge-to-edge repair: in-hospital results and 1-year follow-up of 628 patients of the 2011-2012 Pilot European Sentinel Registry. Journal of the American College of Cardiology. 2014 Sep 2:64(9):875-84. doi: 10.1016/j.jacc.2014.06.1166.

69. Schnitzler K, Hell M, Geyer M, Kreidel F, Münzel T, von Bardeleben RS. Complications Following MitraClip Implantation. Current cardiology reports. 2021 Aug 13:23(9):131. doi: 10.1007/s11886-021-01553-9.
70. Sticchi A, Bartkowiak J, Brugger N, Weiss S, Windecker S, Praz F. Retrograde Retrieval of a Novel Large Mitral Clip After Embolization Into the Left Ventricle. JACC. Case reports. 2021 Oct 20:3(14):1561-1568. doi: 10.1016/j.jaccas.2021.08.024.
71. Hamm K, Barth S, Diegeler A, Kerber S. Stroke and thrombus formation appending to the MitraClip: what is the appropriate anticoagulation regimen? The Journal of heart valve disease. 2013 Sep:22(5):713-5
72. Nakajima Y, Kar S. First experience of the usage of a GORE CARDIOFORM Septal Occluder device for treatment of a significant residual commissural mitral regurgitation jet following a MitraClip procedure. Catheterization and cardiovascular interventions : official journal of the Society for Cardiac Angiography & Interventions. 2018 Sep 1:92(3):607-610. doi: 10.1002/ccd.27438.
73. Mangieri A, Melillo F, Montalto C, Denti P, Praz F, Sala A, Winkel MG, Taramasso M, Tagliari AP, Fam NP, Rubbio AP, De Marco F, Bedogni F, Toggweiler S, Schofer J, Brinkmann C, Sievert H, Van Mieghem NM, Ooms JF, Paradis JM, Rodés-Cabau J, Brochet E, Himbert D, Perl L, Kornowski R, Ielasi A, Regazzoli D, Baldetti L, Masiero G, Tarantini G, Latib A, Laricchia A, Gattas A, Tchetchè D, Dumonteil N, Francesco G, Agricola E, Montorfano M, Lurz P, Crimi G, Maisano F, Colombo A. Management and Outcome of Failed Percutaneous Edge-to-Edge Mitral Valve Plasty: Insight From an International Registry. JACC. Cardiovascular interventions. 2022 Feb 28:15(4):411-422. doi: 10.1016/j.jcin.2021.11.040.
74. Alessandrini H, Dreher A, Harr C, Wohlmuth P, Meincke F, Hakmi S, Ubben T, Kuck KH, Hassan K, Willems S, Schmoeckel M, Geidel S. Clinical impact of intervention strategies after failed transcatheter mitral valve repair. EuroIntervention : journal of EuroPCR in collaboration with the Working Group on Interventional Cardiology of the European Society of Cardiology. 2021 Apr 20:16(17):1447-1454. doi: 10.4244/EIJ-D-20-01008.
75. Lavall D, Reil JC, Segura Schmitz L, Mehrer M, Schirmer SH, Böhm M, Laufs U. Early Hemodynamic Improvement after Percutaneous Mitral Valve Repair Evaluated by Noninvasive Pressure-Volume Analysis. Journal of the American Society of Echocardiography : official publication of the American Society of Echocardiography. 2016 Sep:29(9):888-98. doi: 10.1016/j.echo.2016.05.012..

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