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2014 AHA/ACC Valvular Heart Disease Guideline

2014 AHA/ACC Guideline for the Management of Patients With Valvular Heart Disease: Executive Summary

A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines

Developed in Collaboration With the American Association for Thoracic Surgery, American Society of Echocardiography, Society for Cardiovascular Angiography and Interventions, Society of Cardiovascular Anesthesiologists, and Society of Thoracic Surgeons

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1. Prophylaxis against IE is not recommended in patients with VHD who are at risk of IE for nondental procedures (e.g., TEE, esophagogastroduodenoscopy, colonoscopy, or cystoscopy) in the absence of active infection (44). (Level of Evidence: B)

2.5. Evaluation of Surgical and Interventional Risk
See Table 5 for risk assessment combining STS risk estimate, frailty, major organ system dysfunction, and procedure-specific impediments.

Table 5. Risk Assessment Combining STS Risk Estimate, Frailty, Major Organ System Dysfunction, and Procedure-Specific Impediments

<table>
<thead>
<tr>
<th>Low Risk (Must Meet ALL Criteria in This Column)</th>
<th>Intermediate Risk (Any 1 Criterion in This Column)</th>
<th>High Risk (Any 1 Criterion in This Column)</th>
<th>Prohibitive Risk (Any 1 Criterion in This Column)</th>
</tr>
</thead>
<tbody>
<tr>
<td>STS PROM* &lt;4% AND 4% to 8% OR &gt;8% OR Predicted risk with surgery of death or major morbidity (all-cause) &gt;50% at 1 y OR</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frailty† None AND 1 Index (mild) OR ≥2 Indices (moderate to severe) OR</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Major organ system compromise not to be improved postoperatively‡ None AND 1 Organ system OR No more than 2 organ systems OR ≥3 Organ systems OR</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Procedure-specific impediment§ None Possible procedure-specific impediment Possible procedure-specific impediment Severe procedure-specific impediment</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Use of the STS PROM to predict risk in a given institution with reasonable reliability is appropriate only if institutional outcomes are within 1 standard deviation of STS average observed/expected ratio for the procedure in question.
†Seven frailty indices: Katz Activities of Daily Living (independence in feeding, bathing, dressing, transferring, toileting, and urinary continence) and independence in ambulation (no walking aid or assist required or 5-meter walk in <6 s). Other scoring systems can be applied to calculate no, mild-, or moderate-to-severe frailty.
‡Examples of major organ system compromise: Cardiac—severe LV systolic or diastolic dysfunction or RV dysfunction, fixed pulmonary hypertension; CKD stage 3 or worse; pulmonary dysfunction with FEV1 <50% or DLCO2 <50% of predicted; CNS dysfunction (dementia, Alzheimer’s disease, Parkinson’s disease, CVA with persistent physical limitation); GI dysfunction—Crohn’s disease, ulcerative colitis, nutritional impairment, or serum albumin <3.0; cancer—active malignancy; and liver—any history of cirrhosis, variceal bleeding, or elevated INR in the absence of VKA therapy.
§Examples: tracheostomy present, heavily calcified ascending aorta, chest malformation, arterial coronary graft adherent to posterior chest wall, or radiation damage.

CKD indicates chronic kidney disease; CNS, central nervous system; CVA, stroke; DLCO2, diffusion capacity for carbon dioxide; FEV1, forced expiratory volume in 1 s; GI, gastrointestinal; INR, international normalized ratio; LV, left ventricular; PROM, predicted risk of mortality; RV, right ventricular; STS, Society of Thoracic Surgeons; and VKA, vitamin K antagonist.

2.6. The Heart Valve Team and Heart Valve Centers of Excellence: Recommendations

Class I

1. Patients with severe VHD should be evaluated by a multidisciplinary Heart Valve Team when intervention is considered. (Level of Evidence: C)

Class IIa
1. **Consultation with or referral to a Heart Valve Center of Excellence is reasonable when discussing treatment options for 1) asymptomatic patients with severe VHD, 2) patients who may benefit from valve repair versus valve replacement, or 3) patients with multiple comorbidities for whom valve intervention is considered. (Level of Evidence: C)**

A competent, practicing cardiologist should have the ability to diagnose and direct the treatment of most patients with VHD. For instance, otherwise healthy patients with severe VHD who become symptomatic should nearly always be considered for intervention. However, more complex decision-making processes may be required in select patient populations, such as those who have asymptomatic severe VHD, those who are at high risk for intervention, or those who could benefit from specialized therapies such as valve repair or transcatheter valve intervention.

The management of patients with complex severe VHD is best achieved by a Heart Valve Team composed primarily of a cardiologist and surgeon (including a structural valve interventionalist if a catheter-based therapy is being considered). In selected cases, there may be a multidisciplinary, collaborative group of caregivers, including cardiologists, structural valve interventionalists, cardiovascular imaging specialists, cardiovascular surgeons, anesthesiologists, and nurses, all of whom have expertise in the management and outcomes of patients with complex VHD. The Heart Valve Team should optimize patient selection for available procedures through a comprehensive understanding of the risk–benefit ratio of different treatment strategies. This is particularly beneficial in patients in whom there are several options for treatment, such as the elderly high-risk patient with severe symptomatic aortic stenosis (AS) being considered for transcatheter aortic valve replacement (TAVR) or surgical aortic valve replacement (AVR). The patient and family should be sufficiently educated by the Heart Valve Team about all alternatives for treatment so that their expectations can be met as fully as possible using a shared decision-making approach.

The optimal care of the patient with complex heart disease is best performed in centers that can provide all available options for diagnosis and management, including the expertise for complex aortic or mitral valve repair, aortic surgery, and transcatheter therapies. This has led to the development of Heart Valve Centers of Excellence. Heart Valve Centers of Excellence 1) are composed of experienced healthcare providers with expertise from multiple disciplines; 2) offer all available options for diagnosis and management, including complex valve repair, aortic surgery, and transcatheter therapies; 3) participate in regional or national outcome registries; 4) demonstrate adherence to national guidelines; 5) participate in continued evaluation and quality improvement processes to enhance patient outcomes; and 6) publicly report their available mortality and success rates. Decisions about intervention at the Heart Valve Centers of Excellence should be dependent on the centers’ publicly available mortality rates and operative outcomes. It is recognized that some Heart Valve Centers of Excellence may have expertise in select valve problems.
3. Aortic Stenosis: Recommendations

See Table 6 for the stages of valvular AS; Tables 7 and 8 for a summary of recommendations for choice and timing of intervention; and Figure 1 for indications for AVR in patients with AS.

3.1. Stages of Valvular AS

Medical and interventional approaches to the management of patients with valvular AS depend on accurate diagnosis of the cause and stage of the disease process. Table 6 shows the stages of AS ranging from patients at risk of AS (stage A) or with progressive hemodynamic obstruction (stage B) to severe asymptomatic (stage C) and symptomatic AS (stage D). Each of these stages is defined by valve anatomy, valve hemodynamics, the consequences of valve obstruction on the left ventricle and vasculature, as well as by patient symptoms. Hemodynamic severity is best characterized by the transaortic maximum velocity (or mean pressure gradient) when the transaortic volume flow rate is normal. However, some patients with AS have a low transaortic volume flow rate due to either left ventricular (LV) systolic dysfunction with a low left ventricular ejection fraction (LVEF) or due to a small hypertrophied left ventricle with a low stroke volume. These categories of severe AS pose a diagnostic and management challenge distinctly different from the majority of patients with AS who have a high gradient and velocity when AS is severe. These special subgroups with low-flow AS are designated D2 (with a low LVEF) and D3 (with a normal LVEF).

The definition of severe AS is based on natural history studies of patients with unoperated AS, which show that the prognosis is poor once there is a peak aortic valve velocity of >4.0 m per second, corresponding to a mean aortic valve gradient >40 mm Hg. In patients with low forward flow, severe AS can be present with lower aortic valve velocities and lower aortic valve gradients. Thus, an aortic valve area should be calculated in these patients. The prognosis of patients with AS is poorer when the aortic valve area is <1.0 cm². At normal flow rates, an aortic valve area of <0.8 cm² correlates with a mean aortic valve gradient >40 mm Hg. However, symptomatic patients with a calcified aortic valve with reduced opening and an aortic valve area between 0.8 cm² and 1.0 cm² should be closely evaluated to determine whether they would benefit from valve intervention. Meticulous attention to detail is required when assessing aortic valve hemodynamics, either with Doppler echocardiography or cardiac catheterization, and the inherent variability of the measurements and calculations should always be considered in clinical-decision making.
### Table 6. Stages of Valvular AS

<table>
<thead>
<tr>
<th>Stage</th>
<th>Definition</th>
<th>Valve Anatomy</th>
<th>Valve Hemodynamics</th>
<th>Hemodynamic Consequences</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>At risk of AS</td>
<td>Bicuspid aortic valve (or other congenital valve anomaly)</td>
<td>Aortic $V_{max} &lt;$2 m/s</td>
<td>None</td>
<td>None</td>
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<tr>
<td></td>
<td></td>
<td>Aortic valve sclerosis</td>
<td></td>
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<tr>
<td>B</td>
<td>Progressive AS</td>
<td>Mild-to-moderate leaflet calcification of a bicuspid or trileaflet valve with some reduction in systolic motion or Rheumatic valve changes with commissural fusion</td>
<td>Mild AS: Aortic $V_{max}$ 2.0–2.9 m/s or mean $\Delta P &lt;$20 mm Hg Moderate AS: Aortic $V_{max}$ 3.0–3.9 m/s or mean $\Delta P$ 20–39 mm Hg</td>
<td>Early LV diastolic dysfunction may be present Normal LVEF</td>
<td>None</td>
</tr>
<tr>
<td>C: Asymptomatic severe AS</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>C1</td>
<td>Asymptomatic severe AS</td>
<td>Severe leaflet calcification or congenital stenosis with severely reduced leaflet opening</td>
<td>Aortic $V_{max}$ ≥4 m/s or mean $\Delta P \geq$40 mm Hg AVA typically is ≤1.0 cm² (or AVAi ≤0.6 cm²/m²) Very severe AS is an aortic $V_{max}$ ≥5 m/s or mean $\Delta P$ ≥60 mm Hg</td>
<td>LV diastolic dysfunction Mild LV hypertrophy Normal LVEF</td>
<td>None: Exercise testing is reasonable to confirm symptom status</td>
</tr>
<tr>
<td>C2</td>
<td>Asymptomatic severe AS with LV dysfunction</td>
<td>Severe leaflet calcification or congenital stenosis with severely reduced leaflet opening</td>
<td>Aortic $V_{max}$ ≥4 m/s or mean $\Delta P \geq$40 mm Hg AVA typically ≤1.0 cm² (or AVAi ≤0.6 cm²/m²)</td>
<td>LVEF &lt;50%</td>
<td>None</td>
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<tr>
<td>D: Symptomatic severe AS</td>
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<tr>
<td>D1</td>
<td>Symptomatic severe high-gradient AS</td>
<td>Severe leaflet calcification or congenital stenosis with severely reduced leaflet opening</td>
<td>Aortic $V_{max}$ ≥4 m/s or mean $\Delta P \geq$40 mm Hg AVA typically ≤1.0 cm² (or AVAi ≤0.6 cm²/m²) but may be larger with mixed AS/AR</td>
<td>LV diastolic dysfunction LV hypertrophy Pulmonary hypertension may be present</td>
<td>Exertional dyspnea or decreased exercise tolerance Exertional angina Exertional syncope or presyncope</td>
</tr>
<tr>
<td>D2</td>
<td>Symptomatic severe low-flow/low-gradient AS with reduced LVEF</td>
<td>Severe leaflet calcification with severely reduced leaflet motion</td>
<td>AVA ≤1.0 cm² with resting aortic $V_{max}$ &lt;4 m/s or mean $\Delta P &lt;$40 mm Hg Dobutamine stress echocardiography shows AVA ≤1.0 cm² with $V_{max}$ ≥4 m/s at any flow rate</td>
<td>LV diastolic dysfunction LV hypertrophy LVEF &lt;50%</td>
<td>HF Angina Syncope or presyncope</td>
</tr>
<tr>
<td>D3</td>
<td>Symptomatic severe low-gradient</td>
<td>Severe leaflet calcification</td>
<td>AVA ≤1.0 cm² with aortic $V_{max}$ &lt;4 m/s or</td>
<td>Increased LV</td>
<td>HF</td>
</tr>
<tr>
<td>AS with normal LVEF or paradoxical low-flow severe AS</td>
<td>with severely reduced leaflet motion</td>
<td>mean $\Delta P &lt; 40$ mm Hg</td>
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<td></td>
<td></td>
<td>$\bullet$ Indexed AVA $\leq 0.6$ cm$^2$/m$^2$ and</td>
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<td></td>
<td></td>
<td>$\bullet$ Stroke volume index $&lt; 35$ mL/m$^2$</td>
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<td></td>
<td></td>
<td>$\bullet$ Measured when patient is normotensive (systolic BP $&lt; 140$ mm Hg)</td>
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<td>relative wall thickness</td>
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<tr>
<td></td>
<td></td>
<td>$\bullet$ Small LV chamber with low stroke volume</td>
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<td></td>
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<td>$\bullet$ Restrictive diastolic filling</td>
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<td>$\bullet$ LVEF $\geq 50%$</td>
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<td>$\bullet$ Angina</td>
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<td></td>
<td></td>
<td>$\bullet$ Syncope or presyncope</td>
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AR indicates aortic regurgitation; AS, aortic stenosis; AVA, aortic valve area; AVAi, aortic valve area indexed to body surface area; BP, blood pressure; HF, heart failure; LV, left ventricular; LVEF, left ventricular ejection fraction; $\Delta P$, pressure gradient; and $V_{\text{max}}$, maximum aortic velocity.
3.2. Diagnosis and Follow-Up
The overall approach to the initial diagnosis of VHD is discussed in Section 2.3, and additional considerations specific to patients with AS are addressed here.

Class I
1. TTE is indicated in patients with signs or symptoms of AS or a bicuspid aortic valve for accurate diagnosis of the cause of AS, hemodynamic severity, LV size and systolic function, and for determining prognosis and timing of valve intervention (26, 27, 45). *(Level of Evidence: B)*

Class IIa
1. Low-dose dobutamine stress testing using echocardiographic or invasive hemodynamic measurements is reasonable in patients with stage D2 AS with all of the following (46-48), *(Level of Evidence: B)*:
   a. Calcified aortic valve with reduced systolic opening;
   b. LVEF less than 50%;
   c. Calculated valve area 1.0 cm² or less; and
   d. Aortic velocity less than 4.0 m per second or mean pressure gradient less than 40 mm Hg.
2. Exercise testing is reasonable to assess physiological changes with exercise and to confirm the absence of symptoms in asymptomatic patients with a calcified aortic valve and an aortic velocity 4.0 m per second or greater or mean pressure gradient 40 mm Hg or higher (stage C) (27, 37, 38, 49). *(Level of Evidence: B)*

Class III: Harm
1. Exercise testing should not be performed in symptomatic patients with AS when the aortic velocity is 4.0 m per second or greater or mean pressure gradient is 40 mm Hg or higher (stage D) (50). *(Level of Evidence: B)*

3.3. Medical Therapy

Class I
1. Hypertension in patients at risk for developing AS (stage A) and in patients with asymptomatic AS (stages B and C) should be treated according to standard GDMT, started at a low dose, and gradually titrated upward as needed with frequent clinical monitoring (51-53). *(Level of Evidence: B)*

Class IIb
1. Vasodilator therapy may be reasonable if used with invasive hemodynamic monitoring in the acute management of patients with severe decompensated AS (stage D) with New York Heart Association (NYHA) class IV heart failure (HF) symptoms. *(Level of Evidence: C)*

Class III: No Benefit
1. Statin therapy is not indicated for prevention of hemodynamic progression of AS in patients with mild-to-moderate calcific valve disease (stages B to D) (54-56). *(Level of Evidence: A)*

3.4. Timing of Intervention
See Table 7 for a summary of recommendations from this section.

Class I
1. AVR is recommended in symptomatic patients with severe AS (stage D1) with (57-60), (Level of Evidence: B):
   a. Decreased systolic opening of a calcified or congenitally stenotic aortic valve; and
   b. An aortic velocity 4.0 m per second or greater or mean pressure gradient 40 mm Hg or higher; and
   c. Symptoms of HF, syncope, exertional dyspnea, angina, or presyncope by history or on exercise testing.

2. AVR is recommended for asymptomatic patients with severe AS (stage C2) and an LVEF less than 50% with decreased systolic opening of a calcified aortic valve with an aortic velocity 4.0 m per second or greater or mean pressure gradient 40 mm Hg or higher (61, 62). (Level of Evidence: B)

3. AVR is indicated for patients with severe AS (stage C or D) when undergoing cardiac surgery for other indications when there is decreased systolic opening of a calcified aortic valve and an aortic velocity 4.0 m per second or greater or mean pressure gradient 40 mm Hg or higher (63, 64). (Level of Evidence: B)

Class IIa

1. AVR is reasonable for asymptomatic patients with very severe AS (stage C1) with (65, 66), (Level of Evidence: B):
   a. Decreased systolic opening of a calcified valve;
   b. An aortic velocity 5.0 m per second or greater or mean pressure gradient 60 mm Hg or higher; and
   c. A low surgical risk.

2. AVR is reasonable in apparently asymptomatic patients with severe AS (stage C1) with (27, 38), (Level of Evidence: B):
   a. A calcified aortic valve;
   b. An aortic velocity of 4.0 m per second to 4.9 m per second or mean pressure gradient of 40 mm Hg to 59 mm Hg; and
   c. An exercise test demonstrating decreased exercise tolerance or a fall in systolic blood pressure (BP).

3. AVR is reasonable in symptomatic patients with low-flow/low-gradient severe AS with reduced LVEF (stage D2) with a (67-69), (Level of Evidence: B):
   a. Calcified aortic valve with reduced systolic opening;
   b. Resting valve area 1.0 cm² or less;
   c. Aortic velocity less than 4.0 m per second or mean pressure gradient less than 40 mm Hg;
   d. LVEF less than 50%; and
   e. A low-dose dobutamine stress study that shows an aortic velocity 4.0 m per second or greater or mean pressure gradient 40 mm Hg or higher with a valve area 1.0 cm² or less at any dobutamine dose.

4. AVR is reasonable in symptomatic patients with low-flow/low-gradient severe AS (stage D3) with an LVEF 50% or greater, a calcified aortic valve with significantly reduced leaflet motion, and a valve area 1.0 cm² or less only if clinical, hemodynamic, and anatomic data support valve obstruction as the most likely cause of symptoms and data recorded when the patient is normotensive (systolic BP <140 mm Hg) indicate (Level of Evidence: C):
   a. An aortic velocity less than 4.0 m per second or mean pressure gradient less than 40 mm Hg; and
   b. A stroke volume index less than 35 mL/m²; and
   c. An indexed valve area 0.6 cm²/m² or less.

5. AVR is reasonable for patients with moderate AS (stage B) with an aortic velocity between 3.0 m per second and 3.9 m per second or mean pressure gradient between 20 mm Hg and 39 mm Hg who are undergoing cardiac surgery for other indications. (Level of Evidence: C)
Class IIb

1. AVR may be considered for asymptomatic patients with severe AS (stage C1) with an aortic velocity 4.0 m per second or greater or mean pressure gradient 40 mm Hg or higher if the patient is at low surgical risk and serial testing shows an increase in aortic velocity 0.3 m/s or greater per year. *(Level of Evidence: C)*

Table 7. Summary of Recommendations for AS: Timing of Intervention

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>COR</th>
<th>LOE</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>AVR is recommended with severe high-gradient AS who have symptoms by history or on exercise testing (stage D1)</td>
<td>I</td>
<td>B</td>
<td>(10, 57-59)</td>
</tr>
<tr>
<td>AVR is recommended for asymptomatic patients with severe AS (stage C2) and LVEF &lt;50%</td>
<td>I</td>
<td>B</td>
<td>(61, 62)</td>
</tr>
<tr>
<td>AVR is indicated for patients with severe AS (stage C or D) when undergoing other cardiac surgery</td>
<td>I</td>
<td>B</td>
<td>(63, 64)</td>
</tr>
<tr>
<td>AVR is reasonable for asymptomatic patients with very severe AS (stage C1, aortic velocity ≥5.0 m/s) and low surgical risk</td>
<td>IIa</td>
<td>B</td>
<td>(65, 66)</td>
</tr>
<tr>
<td>AVR is reasonable in asymptomatic patients (stage C1) with severe AS and decreased exercise tolerance or an exercise fall in BP</td>
<td>IIa</td>
<td>B</td>
<td>(27, 38)</td>
</tr>
<tr>
<td>AVR is reasonable in asymptomatic patients with low-flow/low-gradient severe AS with reduced LVEF (stage D2) with a low-dose dobutamine stress study that shows an aortic velocity ≥4.0 m/s (or mean pressure gradient ≥40 mm Hg) with a valve area ≤1.0 cm² at any dobutamine dose</td>
<td>IIa</td>
<td>B</td>
<td>(67-69)</td>
</tr>
<tr>
<td>AVR is reasonable in symptomatic patients who have low-flow/low-gradient severe AS (stage D3) who are normotensive and have an LVEF ≥50% if clinical, hemodynamic, and anatomic data support valve obstruction as the most likely cause of symptoms</td>
<td>IIa</td>
<td>C</td>
<td>N/A</td>
</tr>
<tr>
<td>AVR is reasonable for patients with moderate AS (stage B) (aortic velocity 3.0–3.9 m/s) who are undergoing other cardiac surgery</td>
<td>IIa</td>
<td>C</td>
<td>N/A</td>
</tr>
<tr>
<td>AVR may be considered for asymptomatic patients with severe AS (stage C1) and rapid disease progression and low surgical risk</td>
<td>IIb</td>
<td>C</td>
<td>N/A</td>
</tr>
</tbody>
</table>

AS indicates aortic stenosis; AVR, aortic valve replacement by either surgical or transcatheter approach; BP, blood pressure; COR, Class of Recommendation; LOE, Level of Evidence; LVEF, left ventricular ejection fraction; and N/A, not applicable.

Figure 1. Indications for AVR in Patients With AS
Arrows show the decision pathways that result in a recommendation for AVR. Periodic monitoring is indicated for all patients in whom AVR is not yet indicated, including those with asymptomatic AS (stage D or C) and those with low-gradient AS (stage D2 or D3) who do not meet the criteria for intervention.

*AVR should be considered with stage D3 AS only if valve obstruction is the most likely cause of symptoms, stroke volume index is <35 mL/m², indexed AVA is ≤0.6 cm²/m², and data are recorded when the patient is normotensive (systolic BP <140 mm Hg).

AS indicates aortic stenosis; AVA; aortic valve area; AVR, aortic valve replacement by either surgical or transcatheter approach; BP, blood pressure; DSE, dobutamine stress echocardiography; ETT, exercise treadmill test; LVEF, left ventricular ejection fraction; ΔPmean, mean pressure gradient; and Vmax, maximum velocity.

### 3.5. Choice of Intervention
See Table 8 for a summary of recommendations from this section.

**Class I**

1. Surgical AVR is recommended in patients who meet an indication for AVR (Section 3.4) with low or intermediate surgical risk (Section 2.5 in the full-text guideline) (70, 71). *(Level of Evidence: A)*

2. For patients in whom TAVR or high-risk surgical AVR is being considered, a Heart Valve Team consisting of an integrated, multidisciplinary group of healthcare professionals with expertise in VHD, cardiac imaging, interventional cardiology, cardiac anesthesia, and cardiac surgery should collaborate to provide optimal patient care. *(Level of Evidence: C)*
3. TAVR is recommended in patients who meet an indication for AVR (Section 3.4) who have a prohibitive risk for surgical AVR (Section 2.5 in the full-text guideline) and a predicted post-TAVR survival greater than 12 months (72, 73). *(Level of Evidence: B)*

Class IIa

1. TAVR is a reasonable alternative to surgical AVR in patients who meet an indication for AVR (Section 3.4) and who have high surgical risk for surgical AVR (Section 2.5 in the full-text guideline) (74, 75). *(Level of Evidence: B)*

Class IIb

1. Percutaneous aortic balloon dilation may be considered as a bridge to surgical AVR or TAVR in patients with severe symptomatic AS. *(Level of Evidence: C)*

Class III: No Benefit

1. TAVR is not recommended in patients in whom existing comorbidities would preclude the expected benefit from correction of AS (72). *(Level of Evidence: B)*

### Table 8. Summary of Recommendations for AS: Choice of Surgical or Transcatheter Intervention

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>COR</th>
<th>LOE</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Surgical AVR is recommended in patients who meet an indication for AVR (Section 3.4) with low or intermediate surgical risk (Section 2.5 in the full-text guideline)</td>
<td>I</td>
<td>A</td>
<td>(70, 71)</td>
</tr>
<tr>
<td>For patients in whom TAVR or high-risk surgical AVR is being considered, members of a Heart Valve Team should collaborate to provide optimal patient care</td>
<td>I</td>
<td>C</td>
<td>N/A</td>
</tr>
<tr>
<td>TAVR is recommended in patients who meet an indication for AVR for AS who have a prohibitive surgical risk and a predicted post-TAVR survival &gt;12 mo</td>
<td>I</td>
<td>B</td>
<td>(72, 73)</td>
</tr>
<tr>
<td>TAVR is a reasonable alternative to surgical AVR in patients who meet an indication for AVR (Section 3.4) and who have high surgical risk (Section 2.5 in the full-text guideline)</td>
<td>IIa</td>
<td>B</td>
<td>(74, 75)</td>
</tr>
<tr>
<td>Percutaneous aortic balloon dilation may be considered as a bridge to surgical or transcatheter AVR in severely symptomatic patients with severe AS</td>
<td>IIb</td>
<td>C</td>
<td>N/A</td>
</tr>
<tr>
<td>TAVR is not recommended in patients in whom existing comorbidities would preclude the expected benefit from correction of AS</td>
<td>III: No Benefit</td>
<td>B</td>
<td>(72)</td>
</tr>
</tbody>
</table>

AS indicates aortic stenosis; AVR, aortic valve replacement; COR, Class of Recommendation; LOE, Level of Evidence; N/A, not applicable; and TAVR, transcatheter aortic valve replacement.

### 4. Aortic Regurgitation: Recommendations

#### 4.1. Stages of Chronic Aortic Regurgitation

The most common causes of chronic aortic regurgitation (AR) in the United States and other developed countries are bicuspid aortic valve and calcific valve disease. In addition, AR frequently arises from primary diseases causing dilation of the ascending aorta or the sinuses of Valsalva. Another cause of AR is rheumatic heart disease (the leading cause in many developing countries). In the majority of patients with AR, the disease course is chronic and slowly progressive with increasing LV volume overload and LV adaptation via chamber dilation and hypertrophy. Management of patients with AR depends on accurate diagnosis of the cause and stage...
considered for patients with severe MS (MVA ≤ 1.5 cm², stages C and D) who have had recurrent embolic events while receiving adequate anticoagulation.

AF indicates atrial fibrillation; COR, Class of Recommendations; LOE, Level of Evidence; MS, mitral stenosis; MVA, mitral valve area; NYHA, New York Heart Association; and PMBC, percutaneous mitral balloon commissurotomy.

**Figure 3. Indications for Intervention for Rheumatic MS**

AF indicates atrial fibrillation; LA, left atrial; MR, mitral regurgitation; MS, mitral stenosis; MVA, mitral valve area; MVR, mitral valve surgery (repair or replacement); NYHA, New York Heart Association; PCWP, pulmonary capillary wedge pressure; PMBC, percutaneous mitral balloon commissurotomy; and T ½, pressure half-time.

7. Mitral Regurgitation: Recommendations

7.1. Stages of Chronic MR

In assessing the patient with chronic MR, it is critical to distinguish between chronic primary (degenerative) MR and chronic secondary (functional) MR, as these 2 conditions have more differences than similarities.
In chronic primary MR, the pathology of ≥1 of the components of the valve (leaflet, chordae tendineae, papillary muscles, annulus) causes valve incompetence with systolic regurgitation of blood from the left ventricle to the LA (Table 13). The most common cause of chronic primary MR in developed countries is mitral valve prolapse, which has a wide spectrum of etiology and presentation. Younger populations present with severe myxomatous degeneration with gross redundancy of both anterior and posterior leaflets and the chordal apparatus (Barlow’s valve). Alternatively, older populations present with fibroelastic deficiency disease, in which lack of connective tissue leads to chordal rupture. The differentiation between these 2 etiologies has important implications for operative intervention. Other less common causes of chronic primary MR include IE, connective tissue disorders, rheumatic heart disease, cleft mitral valve, and radiation heart disease. If the subsequent volume overload of chronic primary MR is prolonged and severe, it causes myocardial damage, HF, and eventual death. Correction of the MR is curative. Thus, MR is “the disease.”

In chronic secondary MR, the mitral valve is usually normal (Table 14). Instead, severe LV dysfunction is caused either by CAD, related myocardial infarction (ischemic chronic secondary MR), or idiopathic myocardial disease (nonischemic chronic secondary MR). The abnormal and dilated left ventricle causes papillary muscle displacement, which in turn results in leaflet tethering with associated annular dilation that prevents coaptation. Because MR is only 1 component of the disease (severe LV dysfunction, coronary disease, or idiopathic myocardial disease are the others), restoration of mitral valve competence is not by itself curative; thus, the best therapy for chronic secondary MR is much less clear than it is for chronic primary MR. The data are limited, and there is greater difficulty in defining the severity of MR in patients with secondary MR than in those with primary MR. In patients with secondary MR, adverse outcomes are associated with a smaller calculated effective regurgitant orifice compared to primary MR due to multiple reasons. The MR will likely progress due to the associated progressive LV systolic dysfunction and adverse remodeling. In addition, there is an underestimation of effective regurgitant orifice area by the 2-dimensional echocardiography–derived flow convergence method due to the crescentic shape of the regurgitant orifice. There are the additional clinical effects of a smaller amount of regurgitation in the presence of compromised LV systolic function and baseline elevated filling pressures.
<table>
<thead>
<tr>
<th>Grade</th>
<th>Definition</th>
<th>Valve Anatomy</th>
<th>Valve Hemodynamics*</th>
<th>Hemodynamic Consequences</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>At risk of MR</td>
<td>• Mild mitral valve prolapse with normal coaptation</td>
<td>• No MR jet or small central jet area &lt;20% LA on Doppler</td>
<td>• None</td>
<td>• None</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Mild valve thickening and leaflet restriction</td>
<td>• Small vena contracta &lt;0.3 cm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>B</td>
<td>Progressive MR</td>
<td>• Severe mitral valve prolapse with normal coaptation</td>
<td>• Central jet MR 20%–40% LA or late systolic eccentric jet MR</td>
<td>• Mild LA enlargement</td>
<td>• None</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Rheumatic valve changes with leaflet restriction and loss of central coaptation</td>
<td>• Vena contracta &lt;0.7 cm</td>
<td>• No LV enlargement</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Prior IE</td>
<td>• Regurgitant volume &lt;60 mL</td>
<td>• Normal pulmonary pressure</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Thickening of leaflets with radiation heart disease</td>
<td>• Regurgitant fraction &lt;50%</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• ERO &lt;0.40 cm²</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Angiographic grade 1–2+</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C</td>
<td>Asymptomatic severe MR</td>
<td>• Severe mitral valve prolapse with loss of coaptation or flail leaflet</td>
<td>• Central jet MR &gt;40% LA or holosystolic eccentric jet MR</td>
<td>• Moderate or severe LA enlargement</td>
<td>• None</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Rheumatic valve changes with leaflet restriction and loss of central coaptation</td>
<td>• Vena contracta ≥0.7 cm</td>
<td>• LV enlargement</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Prior IE</td>
<td>• Regurgitant volume ≥60 mL</td>
<td>• Pulmonary hypertension may be present at rest or with exercise</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Thickening of leaflets with radiation heart disease</td>
<td>• Regurgitant fraction ≥50%</td>
<td>• C1: LVEF &gt;60% and LVESD &lt;40 mm</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• ERO ≥0.40 cm²</td>
<td>• C2: LVEF ≤60% and LVESD ≥40 mm</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Angiographic grade 3–4+</td>
<td></td>
<td></td>
</tr>
<tr>
<td>D</td>
<td>Symptomatic severe MR</td>
<td>• Severe mitral valve prolapse with loss of coaptation or flail leaflet</td>
<td>• Central jet MR &gt;40% LA or holosystolic eccentric jet MR</td>
<td>• Moderate or severe LA enlargement</td>
<td>• Decreased</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Rheumatic valve changes with leaflet restriction and loss of central coaptation</td>
<td>• Vena contracta ≥0.7 cm</td>
<td>• LV enlargement</td>
<td>exercise tolerance</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Prior IE</td>
<td>• Regurgitant volume ≥60 mL</td>
<td>• Pulmonary hypertension present</td>
<td>• Exertional dyspnea</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Thickening of leaflets with radiation heart disease</td>
<td>• Regurgitant fraction ≥50%</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• ERO ≥0.40 cm²</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Angiographic grade 3–4+</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Several valve hemodynamic criteria are provided for assessment of MR severity, but not all criteria for each category will be present in each patient. Categorization of MR severity as mild, moderate, or severe depends on data quality and integration of these parameters in conjunction with other clinical evidence.

ERO indicates effective regurgitant orifice; IE, infective endocarditis; LA, left atrium/atrial; LV, left ventricular; LVEF, left ventricular ejection fraction; LVESD; left ventricular end-systolic dimension; and MR, mitral regurgitation.
Table 14. Stages of Secondary MR

<table>
<thead>
<tr>
<th>Grade</th>
<th>Definition</th>
<th>Valve Anatomy</th>
<th>Valve Hemodynamics*</th>
<th>Associated Cardiac Findings</th>
<th>Symptoms</th>
</tr>
</thead>
</table>
| A     | At risk of MR | • Normal valve leaflets, chords, and annulus in a patient with coronary disease or cardiomyopathy | • No MR jet or small central jet area <20% LA on Doppler  
• Small vena contracta <0.30 cm | • Normal or mildly dilated LV size with fixed (infarction) or inducible (ischemia) regional wall motion abnormalities  
• Primary myocardial disease with LV dilation and systolic dysfunction | Symptoms due to coronary ischemia or HF may be present that respond to revascularization and appropriate medical therapy |
| B     | Progressive MR | • Regional wall motion abnormalities with mild tethering of mitral leaflet  
• Annular dilation with mild loss of central coaptation of the mitral leaflets | • ERO <0.20 cm²†  
• Regurgitant volume <30 mL  
• Regurgitant fraction <50% | • Regional wall motion abnormalities with reduced LV systolic function  
• LV dilation and systolic dysfunction due to primary myocardial disease | Symptoms due to coronary ischemia or HF may be present that respond to revascularization and appropriate medical therapy |
| C     | Asymptomatic severe MR | • Regional wall motion abnormalities and/or LV dilation with severe tethering of mitral leaflet  
• Annular dilation with severe loss of central coaptation of the mitral leaflets | • ERO ≥0.20 cm²†  
• Regurgitant volume ≥30 mL  
• Regurgitant fraction ≥50% | • Regional wall motion abnormalities with reduced LV systolic function  
• LV dilation and systolic dysfunction due to primary myocardial disease | Symptoms due to coronary ischemia or HF may be present that respond to revascularization and appropriate medical therapy |
| D     | Symptomatic severe MR | • Regional wall motion abnormalities and/or LV dilation with severe tethering of mitral leaflet  
• Annular dilation with severe loss of central coaptation of the mitral leaflets | • ERO ≥0.20 cm²†  
• Regurgitant volume ≥30 mL  
• Regurgitant fraction ≥50% | • Regional wall motion abnormalities with reduced LV systolic function  
• LV dilation and systolic dysfunction due to primary myocardial disease | HF symptoms due to MR persist even after revascularization and optimization of medical therapy  
• Decreased exercise tolerance  
• Exertional dyspnea |

*Several valve hemodynamic criteria are provided for assessment of MR severity, but not all criteria for each category will be present in each patient. Categorization of MR severity as mild, moderate, or severe depends on data quality and integration of these parameters in conjunction with other clinical evidence.

†The measurement of the proximal isovelocity surface area by 2D TTE in patients with secondary MR underestimates the true ERO due to the crescentic shape of the proximal convergence.

2D indicates 2-dimensional; ERO, effective regurgitant orifice; HF, heart failure; LA, left atrium; LV, left ventricular; MR, mitral regurgitation; and TTE, transthoracic echocardiogram.
7.2. Chronic Primary MR

7.2.1. Diagnosis and Follow-Up

Class I
1. TTE is indicated for baseline evaluation of LV size and function, right ventricular (RV) function and left atrial size, pulmonary artery pressure, and mechanism and severity of primary MR (stages A to D) in any patient suspected of having chronic primary MR (6, 23, 146-162). \(\text{(Level of Evidence: B)}\)
2. CMR is indicated in patients with chronic primary MR to assess LV and RV volumes, function, or MR severity and when these issues are not satisfactorily addressed by TTE (157, 163, 164). \(\text{(Level of Evidence: B)}\)
3. Intraoperative TEE is indicated to establish the anatomic basis for chronic primary MR (stages C and D) and to guide repair (165, 166). \(\text{(Level of Evidence: B)}\)
4. TEE is indicated for evaluation of patients with chronic primary MR (stages B to D) in whom noninvasive imaging provides nondiagnostic information about severity of MR, mechanism of MR, and/or status of LV function. \(\text{(Level of Evidence: C)}\)

Class IIa
1. Exercise hemodynamics with either Doppler echocardiography or cardiac catheterization is reasonable in symptomatic patients with chronic primary MR where there is a discrepancy between symptoms and the severity of MR at rest (stages B and C) (167, 168). \(\text{(Level of Evidence: B)}\)
2. Exercise treadmill testing can be useful in patients with chronic primary MR to establish symptom status and exercise tolerance (stages B and C). \(\text{(Level of Evidence: C)}\)

7.2.2. Medical Therapy

Class IIa
1. Medical therapy for systolic dysfunction is reasonable in symptomatic patients with chronic primary MR (stage D) and LVEF less than 60% in whom surgery is not contemplated (169-173). \(\text{(Level of Evidence: B)}\)

Class III: No Benefit
1. Vasodilator therapy is not indicated for normotensive asymptomatic patients with chronic primary MR (stages B and C1) and normal systolic LV function (173-178). \(\text{(Level of Evidence: B)}\)

7.2.3. Intervention

See Table 15 for a summary of recommendations from this section.

Class I
1. Mitral valve surgery is recommended for symptomatic patients with chronic severe primary MR (stage D) and LVEF greater than 30% (156, 179). \(\text{(Level of Evidence: B)}\)
2. Mitral valve surgery is recommended for asymptomatic patients with chronic severe primary MR and LV dysfunction (LVEF 30% to 60% and/or LVESD ≥40 mm, stage C2) (150-153, 180-182). \(\text{(Level of Evidence: B)}\)
3. Mitral valve repair is recommended in preference to mitral valve replacement (MVR) when surgical treatment is indicated for patients with chronic severe primary MR limited to the posterior leaflet (155, 183-198). \(\text{(Level of Evidence: B)}\)
4. Mitral valve repair is recommended in preference to MVR when surgical treatment is indicated for patients with chronic severe primary MR involving the anterior leaflet or both leaflets when a successful and durable repair can be accomplished (195-197, 199-203). \(\text{(Level of Evidence: B)}\)
5. Concomitant mitral valve repair or MVR is indicated in patients with chronic severe primary MR undergoing cardiac surgery for other indications (204). *(Level of Evidence: B)*

**Class IIa**

1. Mitral valve repair is reasonable in asymptomatic patients with chronic severe primary MR (stage C1) with preserved LV function (LVEF >60% and LVESD <40 mm) in whom the likelihood of a successful and durable repair without residual MR is greater than 95% with an expected mortality rate of less than 1% when performed at a Heart Valve Center of Excellence (149, 203, 205-209). *(Level of Evidence: B)*

2. Mitral valve repair is reasonable for asymptomatic patients with chronic severe nonrheumatic primary MR (stage C1) and preserved LV function (LVEF >60% and LVESD <40 mm) in whom there is a high likelihood of a successful and durable repair with 1) new onset of AF or 2) resting pulmonary hypertension (pulmonary artery systolic arterial pressure >50 mm Hg) (154, 205, 210-215). *(Level of Evidence: B)*

3. Concomitant mitral valve repair is reasonable in patients with chronic moderate primary MR (stage B) when undergoing cardiac surgery for other indications. *(Level of Evidence: C)*

**Class IIb**

1. Mitral valve surgery may be considered in symptomatic patients with chronic severe primary MR and LVEF less than or equal to 30% (stage D). *(Level of Evidence: C)*

2. Mitral valve repair may be considered in patients with rheumatic mitral valve disease when surgical treatment is indicated if a durable and successful repair is likely or when the reliability of long-term anticoagulation management is questionable (194, 202, 203). *(Level of Evidence: B)*

3. Transcatheter mitral valve repair may be considered for severely symptomatic patients (NYHA class III to IV) with chronic severe primary MR (stage D) who have favorable anatomy for the repair procedure and a reasonable life expectancy but who have a prohibitive surgical risk because of severe comorbidities and remain severely symptomatic despite optimal GDMT for HF (216). *(Level of Evidence: B)*

**Class III: Harm**

1. MVR should not be performed for the treatment of isolated severe primary MR limited to less than one half of the posterior leaflet unless mitral valve repair has been attempted and was unsuccessful (195-198). *(Level of Evidence: B)*

---

### Table 15. Summary of Recommendations for Chronic Primary MR

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>COR</th>
<th>LOE</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>MV surgery is recommended for symptomatic patients with chronic severe primary MR (stage D) and LVEF &gt;30%</td>
<td>I</td>
<td>B</td>
<td>(156, 179)</td>
</tr>
<tr>
<td>MV surgery is recommended for asymptomatic patients with chronic severe primary MR and LV dysfunction (LVEF 30%–60% and/or LVESD ≥40 mm, stage C2)</td>
<td>I</td>
<td>B</td>
<td>(150-153, 180-182)</td>
</tr>
<tr>
<td>MV repair is recommended in preference to MVR when surgical treatment is indicated for patients with chronic severe primary MR limited to the posterior leaflet</td>
<td>I</td>
<td>B</td>
<td>(155, 183-198)</td>
</tr>
<tr>
<td>MV repair is recommended in preference to MVR when surgical treatment is indicated for patients with chronic severe primary MR involving the anterior leaflet or both leaflets when a successful and durable repair can be accomplished</td>
<td>I</td>
<td>B</td>
<td>(195-197, 199-203)</td>
</tr>
<tr>
<td>Concomitant MV repair or replacement is indicated in patients with chronic severe primary MR undergoing cardiac surgery for other indications</td>
<td>I</td>
<td>B</td>
<td>(204)</td>
</tr>
</tbody>
</table>
MV repair is reasonable in asymptomatic patients with chronic severe primary MR (stage C1) with preserved LV function (LVEF >60% and LVESD <40 mm) in whom the likelihood of a successful and durable repair without residual MR is >95% with an expected mortality rate of <1% when performed at a Heart Valve Center of Excellence

<table>
<thead>
<tr>
<th>Recommendation</th>
<th>Class</th>
<th>Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>MV repair is reasonable in asymptomatic patients with chronic severe primary MR (stage C1) and preserved LV function in whom there is a high likelihood of a successful and durable repair with 1) new onset of AF or 2) resting pulmonary hypertension (PA systolic arterial pressure &gt;50 mm Hg)</td>
<td>IIa</td>
<td>B</td>
</tr>
</tbody>
</table>

Concomitant MV repair is reasonable in patients with chronic moderate primary MR (stage B) undergoing cardiac surgery for other indications

<table>
<thead>
<tr>
<th>Recommendation</th>
<th>Class</th>
<th>Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>MV surgery may be considered in symptomatic patients with chronic severe primary MR and LVEF ≤30% (stage D)</td>
<td>IIb</td>
<td>C</td>
</tr>
</tbody>
</table>

MV repair may be considered in patients with rheumatic mitral valve disease when surgical treatment is indicated if a durable and successful repair is likely or if the reliability of long-term anticoagulation management is questionable

<table>
<thead>
<tr>
<th>Recommendation</th>
<th>Class</th>
<th>Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transcatheter MV repair may be considered for severely symptomatic patients (NYHA class III/IV) with chronic severe primary MR (stage D) who have a reasonable life expectancy but a prohibitive surgical risk because of severe comorbidities</td>
<td>IIb</td>
<td>B</td>
</tr>
</tbody>
</table>

MVR should not be performed for treatment of isolated severe primary MR limited to less than one half of the posterior leaflet unless MV repair has been attempted and was unsuccessful

<table>
<thead>
<tr>
<th>Recommendation</th>
<th>Class</th>
<th>Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>MV repair is reasonable in asymptomatic patients with chronic severe primary MR (stage C1) with preserved LV function (LVEF &gt;60% and LVESD &lt;40 mm) in whom the likelihood of a successful and durable repair without residual MR is &gt;95% with an expected mortality rate of &lt;1% when performed at a Heart Valve Center of Excellence</td>
<td>IIa</td>
<td>B</td>
</tr>
</tbody>
</table>

AF indicates atrial fibrillation; COR, Class of Recommendation; LOE, Level of Evidence; LV, left ventricular; LVEF, left ventricular ejection fraction; LVESD, left ventricular end-systolic dimension; MR, mitral regurgitation; MV, mitral valve; MVR, mitral valve replacement; N/A, not applicable; NYHA, New York Heart Association; and PA, pulmonary artery.

7.3. Chronic Secondary MR

7.3.1. Diagnosis and Follow-Up

Class I

1. TTE is useful to establish the etiology of chronic secondary MR (stages B to D) and the extent and location of wall motion abnormalities and to assess global LV function, severity of MR, and magnitude of pulmonary hypertension. (Level of Evidence: C)

2. Noninvasive imaging (stress nuclear/positron emission tomography, CMR, or stress echocardiography), cardiac CT angiography, or cardiac catheterization, including coronary arteriography, is useful to establish etiology of chronic secondary MR (stages B to D) and/or to assess myocardial viability, which in turn may influence management of functional MR. (Level of Evidence: C)

7.3.2. Medical Therapy

Class I

1. Patients with chronic secondary MR (stages B to D) and HF with reduced LVEF should receive standard GDMT therapy for HF, including ACE inhibitors, ARBs, beta blockers, and/or aldosterone antagonists as indicated (128, 217-221). (Level of Evidence: A)

2. Cardiac resynchronization therapy with biventricular pacing is recommended for symptomatic patients with chronic severe secondary MR (stages B to D) who meet the indications for device therapy (222, 223). (Level of Evidence: A)
7.3.3. Intervention

See Table 16 for a summary of recommendations for this section and Figure 4 for indications for surgery for MR.

Class IIa

1. Mitral valve surgery is reasonable for patients with chronic severe secondary MR (stages C and D) who are undergoing CABG or AVR. (Level of Evidence: C)

Class IIb

1. Mitral valve repair or replacement may be considered for severely symptomatic patients (NYHA class III to IV) with chronic severe secondary MR (stage D) who have persistent symptoms despite optimal GDMT for HF (224-235). (Level of Evidence: B)
2. Mitral valve repair may be considered for patients with chronic moderate secondary MR (stage B) who are undergoing other cardiac surgery. (Level of Evidence: C)

Table 16. Summary of Recommendations for Chronic Severe Secondary MR

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>COR</th>
<th>LOE</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>MV surgery is reasonable for patients with chronic severe secondary MR (stages C and D) who are undergoing CABG or AVR</td>
<td>IIa</td>
<td>C</td>
<td>N/A</td>
</tr>
<tr>
<td>MV surgery may be considered for severely symptomatic patients (NYHA class III/IV) with chronic severe secondary MR (stage D)</td>
<td>IIb</td>
<td>B</td>
<td>(224-235)</td>
</tr>
<tr>
<td>MV repair may be considered for patients with chronic moderate secondary MR (stage B) who are undergoing other cardiac surgery</td>
<td>IIb</td>
<td>C</td>
<td>N/A</td>
</tr>
</tbody>
</table>

AVR indicates aortic valve replacement; CABG, coronary artery bypass graft; COR, Class of Recommendation; LOE, Level of Evidence; MR, mitral regurgitation; MV, mitral valve; N/A, not applicable; and NYHA, New York Heart Association.

Figure 4. Indications for Surgery for MR
Mitral Regurgitation

Primary MR

Severe MR

Vena contracta ≥ 0.7 cm
RVol ≥ 60 mL
RF ≥ 50%
ERO ≥ 0.4 cm²
LV dilation

Symptomatic (stage D)

LVEF > 30%

NO

MV Surgery* (IIa)

MV Surgery* (I)

Asymptomatic (stage C)

LVEF 30% to ≤ 60%
or LVESD ≥ 40 mm (stage C2)

LVEF ≤ 60% and LVESD ≥ 40 mm (stage C1)

New onset AF or PASP ≥ 50 mm Hg (stage C1)

Likelihood of successful repair > 95% and Expected mortality < 1%

NO

YES

MV Repair (IIa)

Periodic Monitoring

MV Surgery* (IIb)

Periodic Monitoring

Secondary MR

Progressive MR

Vena contracta < 0.7 cm
RVol < 60 mL
RF < 50%
ERO < 0.4 cm²

Symptomatic severe MR (stage D)

Asymptomatic severe MR (stage C)

Progressive MR (stage B)

CAD Rx

HF Rx Consider CRT

Persistent NYHA class III/IV symptoms

*Mitral valve repair is preferred over MVR when possible.

AF indicates atrial fibrillation; CAD, coronary artery disease; CRT, cardiac resynchronization therapy; ERO, effective regurgitant orifice; HF, heart failure; LV, left ventricular; LVEF, left ventricular ejection fraction; LVESD, left ventricular end-systolic dimension; MR, mitral regurgitation; MV, mitral valve; MVR, mitral valve replacement; NYHA, New York Heart Association; PASP, pulmonary artery systolic pressure; RF, regurgitant fraction; RVol, regurgitant volume; and Rx, therapy.

8. Tricuspid Valve Disease: Recommendations

8.1. Stages of TR

Trace-to-mild degrees of TR of no physiological consequence are commonly detected on TTE in subjects with anatomically normal valves. Primary disorders of the tricuspid apparatus that can lead to more significant degrees of TR include rheumatic disease, prolapse, congenital disease (Ebstein’s), IE, radiation, carcinoid, blunt chest wall trauma, RV endomyocardial biopsy–related trauma, and intra-annular RV pacemaker or implantable cardioverter-defibrillator leads. Approximately 80% of cases of significant TR are functional in nature and related to tricuspid annular dilation and leaflet tethering in the setting of RV remodeling due to pressure and/or volume overload. The tricuspid annulus is a saddle-shaped ellipsoid that becomes planar and circular as it dilates in an anterior-posterior direction and will often not return to its normal size and configuration after relief of RV