

Eligibility and Disqualification Recommendations for Competitive Athletes With Cardiovascular Abnormalities: Task Force 5: Valvular Heart Disease A Scientific Statement From the American Heart Association and American College of Cardiology

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A search of the literature identifies no prospective clinical trials examining the management of athletes or very physically active, asymptomatic people with abnormal cardiac valves. There are also few clinical trials on nonathletes with aortic or mitral valve disease. Consequently, recommendations for athletic participation in people with these conditions are based on cohort analyses of nonathletic subjects and consensus opinion.

The 2014 American Heart Association/American College of Cardiology “Guideline for the Management of Patients With Valvular Heart Disease”¹ defines stages of valve disease that are useful for subgrouping patients with aortic and mitral valve disease. In stage A are asymptomatic people at risk for developing clinically important valve stenosis or regurgitation, such as patients with bicuspid aortic valves or mitral valve prolapse without obstruction or regurgitation. Patients in stage A may have physical findings consistent with the underlying valve pathology, such as a mitral valve click or an aortic ejection sound, but do not have the pathognomonic findings of valvular malfunction. Stage B includes asymptomatic

patients with mild to moderate valvular heart disease with normal left ventricular (LV) systolic function. Stage C designates asymptomatic patients with severe valvular heart disease with evidence of preserved systolic function (stage C1) or LV dysfunction (C2), and stage D designates patients with symptomatic severe valvular heart disease with or without LV dysfunction. Eligibility for competitive sports is a pertinent issue for people with valvular heart disease in stages A, B, and C, whereas symptomatic patients in stage D are not candidates for competition and under most circumstances should be referred for valve replacement or repair. Athletic competition is also a relevant issue in asymptomatic patients who have undergone successful valve surgery.

Aortic Valve Disease

Aortic valve disease is usually caused by degenerative changes in a bicuspid or tricuspid aortic valve. Calcification of trileaflet aortic valves is an increasingly common cause of aortic stenosis (AS) in middle-aged and elderly people because of increased longevity in the United States and other developed

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nations. Bicuspid aortic valves occur in 1.5% to 2.0% of the population and thus are common findings in young athletes.² In developed countries, there is a very low incidence of rheumatic aortic valve disease, but this pathogenesis predominates in athletes from developing nations. Additional causes of outflow obstruction can be nonvalvular and include subvalvular and supra-valvular AS, both produced by cardiomyopathies and congenital abnormalities in the left ventricle and ascending aorta, as discussed in other sections of this document. Primary diseases of the aorta are common causes of aortic valve regurgitation (AR),¹ and this pathogenesis should be considered in athletes presenting with AR.

Aortic Stenosis

AS is a well-known cause of exertion-related sudden cardiac death but is responsible for <4% of sudden deaths in young athletes.³ The severity of AS is best evaluated with the combination of the history, physical examination, and Doppler echocardiography. A history of decreasing exercise tolerance, exertional dyspnea, or exercise-induced angina in an athlete with a systolic murmur should raise the possibility of severe AS. A decreased volume and delayed upstroke of the carotid pulse, as well as a greater intensity and duration of the systolic murmur, also suggest clinically important AS.

Assessment of congenital AS in children and adolescents and recommendations for participation in athletics in these age groups are discussed in the Task Force 4 report⁴ on congenital heart disease in this document. The following discussion pertains to recommendations in fully grown athletes in late adolescence and adulthood. Doppler echocardiography is the standard method to assess AS,⁴ and its severity is graded as shown in Table 1.

Clinicians should combine features of the history, physical examination, and echocardiogram in evaluating the severity of AS, as well as integrating the various echocardiographic measures of jet velocity, mean gradient, and calculated valve area, because each has limitations. In young patients with abnormal aortic valves, it is also important to assess the size and morphology of the ascending aorta to exclude concomitant aortopathy, as discussed below. Athletes with mild or moderate AS (stage B) should be evaluated yearly, because the valve can narrow progressively. Exercise testing with electrocardiographic and blood pressure monitoring is useful in evaluating ostensibly asymptomatic athletes with AS because it may reveal unexpectedly low exercise tolerance, exercise hypotension, or electrocardiographic abnormalities that may alter the exercise recommendations. Doppler echocardiography can underestimate the severity of the aortic

valve gradient, so further evaluation is warranted in athletes with Doppler evidence of mild or moderate AS who have symptoms or LV hypertrophy.

Evaluation

Athletes with bicuspid aortic valves without stenosis (stage A) should undergo yearly physical examinations for detection of new onset of heart murmurs. Athletes with mild to moderate AS (stage B) should have a yearly history, physical examination, and Doppler echocardiogram to evaluate disease severity. Exercise testing should be performed in athletes with mild and moderate AS to ensure that their effort tolerance is commensurate with the proposed athletic activity and that they do not develop exercise hypotension or electrocardiographic evidence of ischemia.

Recommendations

1. Athletes with AS should be evaluated yearly to determine whether sports participation can continue (Class I; Level of Evidence C).
2. Athletes with mild AS (stage B) and a normal maximal exercise response can participate in all sports (Class IIa; Level of Evidence C).
3. Athletes with moderate AS (stage B) can participate in low and moderate static or low and moderate dynamic competitive sports (classes IA, IB, and IIA) if exercise tolerance testing to at least the level of activity achieved in competition and the training regimen demonstrates satisfactory exercise capacity without symptoms, ST-segment depression, or ventricular tachyarrhythmias, and with a normal blood pressure response (Class IIa; Level of Evidence C).
4. Asymptomatic athletes with severe AS (stage C) should not participate in competitive sports, with the possible exception of low-intensity (class IA) sports (Class III; Level of Evidence C).
5. Symptomatic patients with AS (stage D) should not participate in competitive sports (Class III; Level of Evidence C).

Aortic Regurgitation

The common causes of chronic AR include bicuspid aortic valve disease, congenital connective tissues disorders such as Marfan syndrome, rheumatic heart disease, and idiopathic or hypertensive dilation of the ascending aorta.¹ Chronic AR is usually asymptomatic and well tolerated for years, but when severe, it produces a gradual increase in LV dimensions. The diagnosis during the asymptomatic stages of AR (stages B and C) is suggested on physical examination by a wide arterial pulse pressure, a diastolic murmur heard along the sternal border, or a systolic outflow murmur related to the increased forward stroke volume. Doppler echocardiography is useful in confirming the diagnosis and grading the severity of AR.^{1,5} AR produces both pressure and volume loading of the LV but is usually well tolerated for decades, with normal LV systolic performance despite the increased LV volume until the LV cannot tolerate further increases in the volume overload.

It is often difficult to differentiate the LV dilatation produced in athletes by exercise training from the dilatation produced by

Table 1. Severity of Aortic Stenosis by Doppler Echocardiography

Severity	Jet Velocity, m/s	Mean Gradient, mm Hg	Aortic Valve Area, cm ²
Mild	<3	<20	>1.5
Moderate	3–4	20–40	1–1.5
Severe	>4	>40	<1.0

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chronic severe AR in its early and advanced stages. Therefore, assessment of LV enlargement in highly trained athletes with known or suspected AR must take this issue into consideration. Progressively severe AR can result in LV volumes that exceed the normal physiological responses to athletic training, but there is overlap in LV volume encountered in normal athletes and patients with AR. Up to 45% of trained male athletes have LV end-diastolic dimension (LVEDD) >55 mm,^{6,7} but only 14% of even elite male athletes have LVEDD >60 mm, and LVEDD rarely exceeds 70 mm.^{6,7} LVEDD >55 mm occurs in <10% of elite women athletes and is >60 mm in only 1%.⁸ Hence, athletes with severe AR and LVEDD exceeding these values have a high likelihood that severe AR is contributing to the LV dilation and should be evaluated carefully for decreasing exercise tolerance and absence of ventricular augmentation with exercise. Similarly, LV end-systolic dimension (LVESD) may also be increased with athletic training. Among elite athletes, the upper limit of LVESD is 49 mm for men and 38 mm for women.⁷ It may be helpful to normalize LVEDD and LVESD for body size,⁹ because larger athletes have larger ventricular volumes. Data indexed for body surface area and height in athletes are available for LVEDD^{6,8} but not LVESD. The reported upper limit of LVEDD indexed for body surface area is 35.3 mm/m² for men and 40.8 mm/m² for women.⁸ Values for LVEDD and LVESD for elite athletes as reported by Pelliccia et al^{7,8} are summarized in Table 2.

The LV ejection fraction response to exercise is also maintained in patients with chronic AR until there is severe LV dilation.¹⁰ An ejection fraction <50% at rest in an athlete with severe AR indicates LV decompensation. Serial assessment of the LVESD is valuable in assessing the progressive effects of severe AR in those with normal LV ejection fractions. In patients with severe AR, the 2014 American Heart Association/American College of Cardiology “Guideline for the Management of Patients With Valvular Heart Disease”¹¹ defines preserved systolic function (stage C1) as LV ejection fraction ≥50% and LVESD ≤50 mm or indexed LVESD ≤25 mL/m².

Evaluation

Athletes with AR should undergo a yearly history and physical examination with Doppler echocardiography. Exercise testing to at least the level of activity achieved in competition and the training regimen is helpful in confirming asymptomatic status and assessing blood pressure responses. The usefulness of assessing

LV function with exercise in athletes has not been established. Patients with AR often have underlying bicuspid aortic valves. In these patients, it is important to also assess the morphology of the aortic root and ascending aorta to rule out associated aortopathy. Recommendations for sports participation for athletes with bicuspid aortic valves and dilated aortas are provided in the Task Force 7 recommendations of this report.¹¹

Recommendations

- 1. Athletes with AR should be evaluated annually to determine whether sports participation can continue (Class I; Level of Evidence C).**
- 2. Exercise testing to at least the level of activity achieved in competition and the training regimen is helpful in confirming asymptomatic status in athletes with AR and assessing blood pressure responses (Class I; Level of Evidence C).**
- 3. Athletes with mild to moderate degrees of AR (stage B) with normal LV ejection fraction and no or mild LV dilatation can participate in all competitive sports if they have normal exercise tolerance on exercise testing (Class I; Level of Evidence C).**
- 4. Athletes with mild to moderate degrees of AR with normal LV ejection fraction and moderate LV dilatation (LVESD <50 mm [men], <40 mm [women], or <25 mm/m² [either sex]) can reasonably participate in all competitive sports if they have normal exercise tolerance on exercise testing (Class IIa; Level of Evidence C).**
- 5. It may be reasonable for athletes with severe AR, LV ejection fraction ≥50% (stage C1), and LVESD <50 mm (men), <40 mm (women), or <25 mm/m² (either sex) to participate in all competitive sports if they have normal exercise tolerance, and Doppler echocardiography indicates no progression of AR severity or severity of LV dilatation (Class IIb; Level of Evidence C).**
- 6. It may be reasonable for athletes with AR and aortic dimensions of 41 to 45 mm to participate in sports with low risk of bodily contact (Class IIb; Level of Evidence C).**
- 7. Athletes with severe AR and symptoms (stage D), LV systolic dysfunction with ejection fraction <50% (stage C2), LVESD >50 mm or >25 mm/m² (stage C2), or severe increase in LVEDD (>70 mm or ≥35.3 mm/m² [men], >65 mm or ≥40.8 mm/m² [women]) should not participate in competitive sports (Class III; Level of Evidence C).**

Bicuspid Aortic Valves

Bicuspid aortic valve is present in 1% to 2% of the population^{1,2} and is the marker of connective tissue abnormalities that affect both the aortic valve and aorta. That patients with bicuspid aortic valves are at increased risk for AS and AR is well known, but these patients are also at increased risk for aortic enlargement and aortic dissection, although the absolute risk for these events is quite small,^{1,12} and it is not known whether restriction of physical activity limits the risk or the rate of aortic enlargement or dissection.

Table 2. Left Ventricular Dimensions in Elite Athletes

	Men		Women	
	Mean±SD	Upper Limit	Mean±SD	Upper Limit
LVESD, mm*	38.2±3.2	49	32.9±2.9	38
LVEDD, mm*	58.8±3.4	70	52.2±3.2	60
LVEDD, mm†	54.2±2.0	66	48.9±3.8	66
LVEDD/BSA, mm/m ² †	28.1±2.3	35.3	29.8±2.5	40.8
LVEDD/height, mm/m†	30.1±2.1	36.8	29.3±1.9	35.9

BSA indicates body surface area; LVEDD, left ventricular end-diastolic dimension; and LVESD, left ventricular end-systolic dimension.

*Data from 114 Olympic athletes (89 men, 25 women).⁷

†Data from 1338 elite athletes (738 men, 600 women).⁸

Evaluation

Patients with bicuspid aortic valves should undergo echocardiography to evaluate both aortic valve function and the size of the aortic sinuses and ascending aorta. The Task Force 7 report¹¹ in this document contains recommendations for sports participation in athletes with bicuspid aortic valves and dilated aortas.

Mitral Valve Disease

Mitral Stenosis

The pathogenesis of mitral stenosis (MS) is almost always rheumatic. Most patients with significant MS will be sufficiently symptomatic during exercise that participation in competitive sports is not an issue, but patients with mild to moderate MS may be asymptomatic even with strenuous exercise. MS rarely causes sudden death; however, exercise (with an increase in heart rate and cardiac output) can cause sudden marked increases in pulmonary capillary and pulmonary artery pressures, at times resulting in sudden acute pulmonary edema.¹³ Furthermore, the long-term effect of repeated exertion-related increases in pulmonary artery wedge and pulmonary artery pressures on the lungs or right ventricle is unknown, nor is the effect of even periodic strenuous exercise on the likelihood of developing atrial fibrillation. When atrial fibrillation occurs, even patients with mild MS must receive anticoagulation therapy. The above considerations must be understood by the patient and the family in considering participation in strenuous competitive activity. Another problem associated with MS is systemic embolization, which occurs most commonly in the presence of atrial fibrillation, but there is no evidence that this potential complication is provoked by strenuous exercise.

Clues regarding the hemodynamic severity of MS may often be obtained from the history and physical examination, but accurate noninvasive assessment of severity requires 2-dimensional and Doppler echocardiography in the majority of patients. MS is categorized as severe when the mitral valve area is $<1.5 \text{ cm}^2$, which corresponds to a mean transmitral gradient of 5 to 10 mm Hg at normal resting heart rates.¹ The mean pressure gradient is highly dependent on the transvalvular flow and diastolic filling period and will vary greatly with increases in heart rate during exercise. A mean transmitral gradient $>15 \text{ mm Hg}$ or pulmonary artery wedge pressure $>25 \text{ mm Hg}$ during exercise is indicative of significant MS.

Evaluation

In patients with MS and minimal or no symptoms who wish to engage in competitive sports, exercise stress testing should be performed to at least the level of activity that approximates the exercise demands of the sport, particularly when there is a question as to the severity of the MS. In addition, pulmonary artery systolic pressure during exercise can be estimated noninvasively by Doppler echocardiography and may be helpful in making a decision as to how much activity is safe, even if the severity of MS in an individual patient is estimated to be only mild.¹

Recommendations

1. Athletes with MS should be evaluated annually to determine whether sports participation can continue (Class I; Level of Evidence C).
2. Exercise testing to at least the level of activity achieved in competition and the training regimen is useful in confirming asymptomatic status in patients with MS (Class I; Level of Evidence C).
3. It is reasonable for athletes with mild MS (mitral valve area $>2.0 \text{ cm}^2$, mean gradient $<10 \text{ mm Hg}$ at rest) in sinus rhythm to participate in all competitive sports (Class IIa; Level of Evidence C).
4. Athletes with severe MS (mitral valve area $<1.5 \text{ cm}^2$) in either sinus rhythm or atrial fibrillation should not participate in competitive sports, with the possible exception of low-intensity (class IA) sports (Class III; Level of Evidence C).
5. Patients with MS of any severity who are in atrial fibrillation or have a history of atrial fibrillation, who must receive anticoagulation therapy, should not engage in any competitive sports involving the risk of bodily contact (Class III; Level of Evidence C).

Mitral Regurgitation

Mitral regurgitation (MR) has a variety of possible causes, the most common of which in an athletic population is mitral valve prolapse (myxomatous mitral valve disease). Other common causes are rheumatic heart disease, infective endocarditis, and connective tissue diseases (such as Marfan syndrome). Secondary forms of MR can develop in patients with coronary artery disease and dilated cardiomyopathy because of tethering of the mitral leaflets and restricted leaflet closure. The recommendations outlined in this section are for athletes with primary valvular MR rather than MR secondary to coronary artery disease or other conditions that cause LV dilation or systolic dysfunction.

MR is detected by the characteristic systolic murmur, confirmed and quantified by Doppler echocardiography.^{1,5} The severity of the MR is related to the magnitude of the regurgitant volume, which results in LV dilation and increases in left atrial pressure and volume. The majority of people with mild or moderate MR are asymptomatic (stage B). The increased LV diastolic volume enhances total LV stroke volume enough to accommodate the regurgitant volume and to maintain the forward stroke volume within normal limits. The low impedance presented by regurgitation into the left atrium unloads the left ventricle during ventricular systole, such that measures of LV pump function, such as ejection fraction, tend to overestimate true myocardial performance.¹⁴ For purposes of this discussion, LV systolic dysfunction in subjects with MR is defined as LV ejection fraction $<60\%$ or LVESD $>40 \text{ mm}$.¹ As with AR, the distinction between LV dilation caused by athletic training versus that caused by severe MR is difficult when the LVEDD is $<60 \text{ mm}$ (or $<40 \text{ mm/m}^2$). However, LVEDD measurements $>60 \text{ mm}$ strongly suggest the presence of severe MR and perhaps the need for surgical mitral valve repair and thus warrant further investigation.

In general, exercise produces no significant change or a mild decrease in the regurgitant fraction because of reduced systemic vascular resistance. However, patients with elevation of heart rate (increased systolic ejection time per minute) or blood pressure with exercise may manifest marked increases in regurgitant volume and pulmonary capillary pressures.

Evaluation

Athletes with MR should undergo yearly physical examinations, Doppler echocardiograms, and exercise stress testing to at least the level of activity that approximates the exercise demands of the sport. In addition, pulmonary artery systolic pressure during exercise can be estimated noninvasively by Doppler echocardiography and may be helpful in making a decision as to how much activity is safe, particularly in athletes with greater severity of MR.¹ In patients with MR secondary to previous infective endocarditis or ruptured chordae, the valve tissues theoretically could be further damaged or torn by marked sustained increases in LV systolic pressure, and thus, the recommendations below should be tempered in patients with these mechanisms of MR.

Recommendations

1. Athletes with MR should be evaluated annually to determine whether sports participation can continue (*Class I; Level of Evidence C*).
2. Exercise testing to at least the level of activity achieved in competition and the training regimen is useful in confirming asymptomatic status in patients with MR (*Class I; Level of Evidence C*).
3. Athletes with mild to moderate MR who are in sinus rhythm with normal LV size and function and with normal pulmonary artery pressures (stage B) can participate in all competitive sports (*Class I; Level of Evidence C*).
4. It is reasonable for athletes with moderate MR in sinus rhythm with normal LV systolic function at rest and mild LV enlargement (compatible with that which may result solely from athletic training [LVEDD <60 mm or <35 mm/m² in men or <40 mm/m² in women]) to participate in all competitive sports (stage B) (*Class IIa; Level of Evidence C*).
5. Athletes with severe MR in sinus rhythm with normal LV systolic function at rest and mild LV enlargement (compatible with that which may result solely from athletic training [LVEDD <60 mm or <35.3 mm/m² in men or <40 mm/m² in women]) can participate in low-intensity and some moderate-intensity sports (classes IA, IIA, and IB) (stage C1) (*Class IIb; Level of Evidence C*).
6. Athletes with MR and definite LV enlargement (LVEDD ≥65 mm or ≥35.3 mm/m² [men] or ≥40 mm/m² [women]), pulmonary hypertension, or any degree of LV systolic dysfunction at rest (LV ejection fraction <60% or LVESD >40 mm) should not

participate in any competitive sports, with the possible exception of low-intensity class IA sports (*Class III; Level of Evidence C*).

7. Athletes with a history of atrial fibrillation who are receiving long-term anticoagulation should not engage in sports involving any risk of bodily contact (*Class III; Level of Evidence C*).

Athletic Participation After Cardiac Valve Surgery

Despite advances in cardiac surgery, the long-term mortality after valve replacement surgery is greater than that of a normal population of similar age. A transvalvular gradient of varying severity is present in most patients after valve replacement, which may be aggravated during exercise.^{1,15} Moreover, after implantation of a mechanical prosthesis, which is common in young patients requiring valve replacement, chronic anticoagulation is required. These considerations are important in determining an athlete's suitability for competition after valve replacement. In patients who have undergone aortic valve repair or, more commonly, mitral valve repair, a different set of issues regarding the risks of physical trauma during athletic competition must be considered.

In assessing the athlete's capacity for physical activity after valve surgery, exercise stress testing to at least the level of activity performed in the competitive sport is valuable. In some cases, assessment of prosthetic valve function during exercise will also provide useful information.

Recommendations

1. It is reasonable for athletes with aortic or mitral bioprosthetic valves, not taking anticoagulant agents, who have normal valvular function and normal LV function to participate in low-intensity and some moderate-intensity competitive sports (classes IA, IB, IC, and IIA) (*Class IIa; Level of Evidence C*).
2. Athletes with aortic or mitral mechanical prosthetic valves taking anticoagulant agents with normal valvular function and normal LV function can reasonably participate in low-intensity competitive sports if there is low likelihood of bodily contact (classes IA, IB, and IIA) (*Class IIa; Level of Evidence C*).
3. It is reasonable for patients with MS who have undergone successful percutaneous mitral balloon valvotomy or surgical commissurotomy to participate in competitive sports based on the residual severity of the MS or MR and pulmonary artery pressures at rest and with exercise (*Class IIa; Level of Evidence C*).
4. Athletes who have undergone mitral valve repair for MR or surgical aortic valve repair, have no or mild residual AR or MR, and have normal LV systolic function may be considered for participation in sports at the discretion of the managing physician if there is low likelihood of bodily contact (classes IA, IB, and IIA) (*Class IIa; Level of Evidence C*).

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Reviewer Disclosures

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Eligibility and Disqualification Recommendations for Competitive Athletes With Cardiovascular Abnormalities: Task Force 5: Valvular Heart Disease: A Scientific Statement From the American Heart Association and American College of Cardiology

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of the Council on Clinical Cardiology, Council on Cardiovascular Disease in the Young,
Council on Cardiovascular and Stroke Nursing, Council on Functional Genomics and
Translational Biology, and the American College of Cardiology

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