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Task Force II: Acquired Valvular Heart Disease

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I. General Considerations

Valvular heart disease is manifest clinically as either valvular stenosis or regurgitation. The following disorders will be considered here: mitral valve stenosis, mitral valve regurgitation, aortic valve stenosis, aortic valve regurgitation, tricuspid valve stenosis, tricuspid valve regurgitation and multivalvular disease. Also, persons having had valvuloplasty and bioprosthetic and prosthetic valve replacement will be discussed.

Diagnosis of the *type* of valvular disease usually can be made on the basis of physical examination alone because the presence of valvular disease is revealed by the characteristic murmurs. Estimation of the *severity* of the valvular disease is more difficult, but in asymptomatic patients in whom the cardiac output is not decreased, the severity usually can be estimated noninvasively using history of symptoms, physical examination, electrocardiogram, chest radiograph and Doppler echocardiography techniques. In regurgitant lesions, radioisotope volume studies and ejection fraction with and without exercise are helpful. The reliability of newer techniques, such as Doppler echocardiography, in estimating severity of valvular regurgitation is still being investigated, whereas the value of the Doppler method in estimating the severity of aortic stenosis has now been demonstrated in several laboratories. At present, Doppler techniques can distinguish severe aortic valve stenosis from mild but are less reliable in quantifying aortic stenosis of borderline severity (1). Only rarely are cardiac catheterization and angiography necessary to classify valvular lesions into the general categories of hemodynamically mild, moderate or severe. When symptoms may be unreliable (for instance, where secondary gain in denying symptoms is likely), then exercise tolerance testing and cardiac catheterization can be useful.

Although there is information about sudden death and development of symptoms with exercise in patients with valvular heart disease, there is little information about the rate of progression of severity of the valvular disease or factors that influence the rate of progression. There is also little information on the influence of strenuous exertion on the progression of ventricular dysfunction, especially when that strenuous exertion is periodic in nature.

II. Types of Valve Defects

A. Mitral Valve Stenosis

Definition and evaluation. The etiology of mitral stenosis, for practical purposes, is rheumatic. Most patients with significant mitral stenosis will be sufficiently symptomatic so that they will not wish to engage in competitive sports. Patients with mild to moderate mitral stenosis may be asymptomatic even with strenuous exercise. Fortunately, mitral stenosis rarely causes sudden death. The pathophysiology of the lesion results in increases in left atrial and pulmonary capillary pressure, leading to pulmonary hypertension, which is at times severe and which may ultimately result in right ventricular failure. Exercise, with an increase in heart rate and cardiac output, can cause sudden marked increases in pulmonary capillary and pulmonary artery pressures (2). Another problem that may not be influenced by strenuous exercise associated with mitral stenosis is systemic embolization, occurring more commonly with atrial fibrillation. The long-term effect of repeated increases in pulmonary artery wedge and pulmonary artery pressures on the lung or on the right ventricle is unknown. This must be understood by the patient and the family in considering participation in strenuous competitive activity.

The hemodynamic severity of mitral stenosis may be estimated noninvasively by the history, physical examination, electrocardiogram and chest radiograph. Two-dimensional echocardiography also can be used to classify the size of the mitral valve orifice into the general categories of mild, moderate and severe obstruction. Doppler echocardiography also can be used to quantify the severity of mitral stenosis (3). Symptom limitation is not likely to be a problem because people who wish to engage in competitive sports may deny symptoms, causing the history to be unreliable. For these reasons, a stress test to the level of activity approxi-

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mating the exercise demands of the competitive sport in question should be performed and can be quite helpful in assessing the applicant's exercise capacity.

Because the patient can have a marked elevation in pulmonary artery wedge and pulmonary artery pressures without having severe symptoms, it is recommended that patients with recognized mitral stenosis who wish to engage in competitive sports have cardiac catheterization to estimate valve area and height of pulmonary artery and pulmonary artery wedge pressure with exercise. Particularly when there is a question as to the severity of the mitral stenosis, catheterization should be performed. In estimating the severity of mitral stenosis, some associated mitral regurgitation is common and should be taken into account in calculating mitral valve area.

Hemodynamic severity of the mitral stenosis can be categorized as: *mild:* mitral valve area greater than 1.5 cm^2 or on exercise with pulmonary artery wedge pressure 20 mm Hg or less or pulmonary artery systolic pressure less than 35 mm Hg; *moderate:* mitral valve area 1.1 to 1.4 cm^2 or on exercise pulmonary artery wedge pressure 25 mm Hg or less or pulmonary artery systolic pressure 50 mm Hg or less; and *severe:* mitral valve less than 1.1 cm^2 or on exercise with pulmonary artery wedge pressure of greater than 26 mm Hg or pulmonary systolic pressure greater than 50 mm Hg.

In patients with mitral stenosis a markedly elevated heart rate or cardiac output for sustained periods of time could result in marked elevation in left atrial and pulmonary capillary pressure. Also, sustained elevation in systemic arterial pressure might increase left ventricular filling pressure and increase left atrial pressure. Given these precautions, the following recommendations can be made.

Recommendations.

1. Asymptomatic patients in sinus rhythm with mild mitral stenosis may participate in all competitive sports.
2. Patients in atrial fibrillation with mild mitral stenosis, those in sinus rhythm or atrial fibrillation with moderate stenosis and those with peak pulmonary artery systolic pressure at rest or during exercise less than 50 mm Hg can participate in sports of low intensity (class I.B), and selected patients may engage in some sports with high to moderate static and low dynamic demands (class I.A.3).
3. Patients in either sinus rhythm or atrial fibrillation with severe mitral stenosis should not participate in any competitive sports.

B. Mitral Valve Regurgitation

Definition and evaluation. Mitral regurgitation, unlike mitral stenosis, has a variety of etiologies. The most common cause is mitral valve prolapse; other causes are rheumatic heart disease, infective endocarditis, coronary heart disease, other connective tissue diseases such as the Marfan syndrome and dilated cardiomyopathy. The recommendations outlined in this section are for those patients with primary valvular mitral regurgitation rather than mitral regurgitation secondary to coronary disease or diseases causing left ventricular dilation.

Mitral regurgitation can be detected by the characteristic physical findings. The severity of the mitral regurgitation is related by the magnitude of the regurgitant volume, which results in increased diastolic filling of the left ventricle and increased left atrial pressure. The increased diastolic left ventricular volume increases the left ventricular stroke volume enough to accommodate the regurgitant volume and to keep the forward stroke volume normal. The low impedance presented by regurgitation into the left atrium unloads the left ventricle during ventricular systole and allows for apparently good indexes of left ventricular global function despite true decreases in intrinsic contractility of the left ventricular myocardium (4).

The severity of chronic mitral regurgitation can be adequately judged by noninvasive techniques such as history of symptoms, physical examination, heart size on chest radiograph, electrocardiogram and echocardiogram. Generally, in chronic mitral regurgitation, the left ventricular diastolic volume reflects the severity of the mitral regurgitation. The problems faced by patients with mitral regurgitation are: 1) inability to increase effective forward cardiac output appropriately, 2) increase in left atrial and pulmonary capillary pressure, and, finally, 3) right-sided heart failure. Embolization is less common than with mitral stenosis and is probably not influenced by competitive activity. Atrial fibrillation occurs with enlargement of the left atrium.

Echocardiography and radioisotope angiography can be used to follow the left ventricular function serially in such patients. A decrease in ejection fraction or increase in end-systolic or end-diastolic volume probably indicates a decrease in left ventricular contractility. The etiology of mitral regurgitation may be important in making recommendations concerning heavy physical activity. For instance, mitral regurgitation related to coronary heart disease has a worse prognosis than primary forms of mitral regurgitation, and considerations as to competitive athletics should be based on the coronary heart disease. Patients with mitral regurgitation secondary to previous infective endocarditis might have weakened valves, which theoretically could be further damaged or torn by marked sustained increases in left ventricular systolic pressure. Similarly, mitral valve prolapse with underlying myxomatous changes in the valve may be in danger of further prolapse by strenuous activity, but no information is available on these points.

The effect of exercise on patients with mitral regurgitation has been studied in a limited number of patients (5). In general, in these studies there was no significant change or a mild decrease in the regurgitant fraction because the forward flow and the regurgitant flow increased to the same

extent. There is usually no change or a small decrease in ejection fraction with exercise, although the ejection fraction response may be completely normal in young, asymptomatic individuals. Static exercise that increases arterial pressure is likely to worsen the regurgitation and to elevate the left atrial pressure and, therefore, is probably deleterious (6).

It is known that chronic excessive prolonged volume overload leads to a progressive decrease in myocardial contractility. The presence of intermittent repetitive increases in volume load may therefore be a deleterious factor in the progression of the disease.

Severity of the mitral regurgitation can be estimated by the effect on left ventricular size and systolic function. Because the chest radiograph can be misleading as to the size of the left ventricle, the best way to follow up the patient noninvasively is with combined M-mode and two-dimensional echocardiography or radioisotope angiography, or both.

Recommendations.

1. Asymptomatic patients in sinus rhythm with normal left ventricular size and function may participate in all competitive sports.
2. Asymptomatic patients in sinus rhythm or atrial fibrillation with mild left ventricular enlargement and normal left ventricular function at rest may participate in sports of low intensity (class I.B) and selected patients may engage in some sports with high to moderate dynamic and low static demands (class I.A.2). When atrial fibrillation is present, exercise tolerance testing can ensure that the ventricular rate response to exercise will not be excessive.
3. Symptomatic patients or asymptomatic patients with definite left ventricular enlargement or any degree of left ventricular dysfunction at rest should not participate in any competitive sports.

C. Aortic Valve Stenosis

Definition and evaluation. The diagnosis of aortic stenosis is easily made by physical examination. There are three common etiologies: 1) rheumatic, 2) congenital, and 3) calcific or degenerative. It is recognized that the basis of the calcific or degenerative stenosis is frequently a congenital abnormality of the aortic valve.

The severity of aortic stenosis can usually be judged noninvasively by the history, physical examination and electrocardiographic and echocardiographic findings. Continuous Doppler techniques may allow for reliable assessment of the severity of the stenosis as long as cardiac output is normal. It is frequently necessary to perform cardiac catheterization to ascertain the severity of the stenosis because with all degrees of aortic stenosis the patient can be entirely asymptomatic. Symptoms of left ventricular failure, syncope or angina pectoris occur late in the course of aortic stenosis (7). When there is any doubt as to the severity of the aortic stenosis, or if the patient has any symptoms, cardiac catheterization is necessary. Sudden death in patients with hemodynamically or pathologically mild aortic stenosis, or both, is rare. Although sudden death is common in patients with severe symptomatic aortic stenosis, it also can occur in apparently completely asymptomatic patients (8).

Cardiac catheterization should be performed to assess hemodynamic severity of the stenosis. The severity of aortic stenosis with demonstrated normal cardiac output and aortic pressure measured in the central aorta can be categorized as follows: *mild:* a transvalvular peak systolic pressure gradient of 20 mm Hg or less; *moderate:* a transvalvular gradient of 21 to 39 mm Hg; and *severe:* a transvalvular gradient of 40 mm Hg or more.

Because the hemodynamic severity of aortic stenosis can progress, periodic reevaluation of severity is necessary and should be performed by a physician with expertise in cardiology (9). This reassessment may include Doppler echocardiography to estimate the gradient and may require periodic catheterization.

In patients with aortic valve stenosis, a markedly elevated cardiac output or peripheral vascular resistance for sustained periods of time could result in a marked increase in left ventricular systolic pressure. Given these precautions, the following recommendations can be made.

Recommendations.

1. Patients with mild aortic stenosis may participate in all competitive sports.
2. Asymptomatic patients with mild to moderate aortic stenosis can engage in all sports of low intensity (class I.B) and selected cases may participate in some sports with high to moderate static and low dynamic demands (class I.A.3). Exercise tolerance testing to evaluate exercise capacity, the development of ST segment depression and arrhythmias is recommended and can be valuable in making the decision to allow patients to engage in this level of competitive sports.
3. Patients with rest or exercise-induced ventricular arrhythmias with mild or moderate aortic stenosis should only participate in sports of low intensity (class I.B).
4. Patients with severe aortic stenosis or symptomatic patients with moderate aortic stenosis should not engage in any competitive sports.

D. Aortic Valve Regurgitation

Definition and evaluation. The presence of aortic regurgitation is usually evident by physical examination alone. Like mitral regurgitation, there are multiple etiologies of aortic regurgitation. Any disease affecting the valve or the aortic valve ring and proximal ascending aorta can result in aortic regurgitation. The common etiologies are: 1) con-

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genital primarily bicuspid aortic valve; 2) rheumatic; 3) infective endocarditis; and 4) diseases affecting the proximal ascending aorta, including the Marfan syndrome ascending aortic aneurysms, dissection of the aorta, systemic hypertension and rheumatoid spondylitis, which affect both the aortic wall and the valve.

Aortic regurgitation causes problems by increasing the diastolic volume of the left ventricle and the left ventricular stroke volume, which ultimately leads to left ventricular failure.

There is also an increase in requirement for coronary blood flow to the point of frequently exceeding the coronary supply. With very severe aortic regurgitation, angina pectoris, syncope, ventricular arrhythmias and sudden death can occur. With exercise, there is a decrease in diastolic filling period and an increase in heart rate together with a decrease in peripheral vascular resistance. Thus, forward flow is increased and regurgitant volume is decreased (10).

The hemodynamic severity of aortic regurgitation can be assessed noninvasively by history and physical examination, the severity being reflected by the degree of left ventricular dilation and the peripheral signs of aortic regurgitation. Left ventricular dilation can be judged grossly by chest X-ray film and more precisely by echocardiography. Because of the importance of assessing left ventricular function and the size of the aortic root and proximal ascending aorta in determining the etiology with resulting implications for athletic participation, evaluation by echocardiography is recommended. Exercise testing can be useful in assessing exercise capacity, especially in those patients having nonspecific mild symptoms. Left ventricular function can be assessed by two-dimensional echocardiography and radioisotope angiography. Ventricular function can be followed up serially by these methods. The significance of the decrease in ejection fraction with exercise is unclear. This probably represents an increase in left ventricular wall stress, but currently not enough is known about this finding to use it to make a recommendation as to the advisability of engaging in competitive athletics (11). There are no data to define whether severe increases in physical activity alter the function of the left ventricle. Because of the possibility of progression in the severity of aortic regurgitation, annual reevaluation is recommended.

The hemodynamic severity of aortic regurgitation can be classified by the peripheral signs of aortic regurgitation and the effect of the volume overload on left ventricular volume and systolic function. Because the chest radiography can be misleading as to left ventricular volume and aortic root size, combined M-mode and two-dimensional echocardiography and radioisotope angiography are recommended in following up left ventricular size and function. Exercise testing is recommended to the level of exertion required by the proposed competitive sport. In this way, the patient's tolerance of this degree of exercise can be evaluated.

The grades of hemodynamic severity of aortic regurgitation are: *mild:* absent to slight peripheral signs of aortic regurgitation, normal left ventricular size; *moderate:* peripheral signs of aortic regurgitation with mild to moderate increases in left ventricular size with normal systolic function; and *severe:* peripheral signs of aortic regurgitation with severe left ventricular enlargement or any degree of left ventricular enlargement if left ventricular dysfunction is present.

Recommendations.

1. Asymptomatic patients with mild or moderate aortic regurgitation can participate in all sports of low intensity (class I.B) and in selected cases may engage in some sports with high to moderate dynamic and low static components (class I.A.2)
2. Patients with rest or exercise-induced left ventricular arrhythmias and mild or moderate aortic regurgitation should only participate in low intensity sports (class I.B).
3. Asymptomatic or symptomatic patients with severe aortic regurgitation or symptomatic patients with mild or moderate regurgitation cannot participate in any competitive sports.
4. Patients with aortic regurgitation associated with severe dilation of the aortic sinuses or proximal ascending aorta, or both, should not engage in any competitive sports.

E. Tricuspid Valve Regurgitation

Definition and evaluation. This lesion is far more frequent than tricuspid stenosis and is most often related to failure and dilation of the right ventricle due to pulmonary or right ventricular hypertension. Rheumatic heart disease and infective endocarditis are less common causes.

The severity of tricuspid regurgitation can be estimated noninvasively from the physical examination, chest radiograph and echocardiogram. The pathophysiology depends on whether the etiology is pulmonary hypertension and right ventricular dilation or primary triscupid valve regurgitation. With pulmonary arterial hypertension the prognosis is dependent on the underlying cause. Recommendation here will be for those patients with primary tricuspid valvular regurgitation.

Primary tricuspid valvular regurgitation leads to volume overload of the right ventricle with increased systemic venous pressure and its consequences. There is no evidence that the patient with isolated primary tricuspid valvular regurgitation is placed in jeopardy by engaging in heavy physical exertion. The long-term effects of chronic volume overload on right ventricular function are unknown but are probably deleterious.

Recommendations. Asymptomatic patients with primary tricuspid valvular regurgitation, regardless of severity, in the absence of right atrial pressure greater than 20 mm Hg and in the absence of elevation of right ventricular sys-

tolic pressure with normal right ventricular function may engage in all competitive sports.

F. Tricuspid Valve Stenosis

Isolated tricuspid stenosis is rare. Tricuspid stenosis is for the most part due to rheumatic heart disease and is nearly always associated with mitral stenosis. In the latter instance, the patient should be judged according to the severity of the mitral stenosis.

G. Multivalvular Disease

Definition and evaluation. Multivalvular disease occurs in rheumatic heart disease, myxomatous valvular disease and infective endocarditis. With left ventricular failure and the consequent left atrial and pulmonary arterial hypertension, mitral regurgitation and tricuspid regurgitation can occur. These latter problems are not addressed in this section.

The lesions can be diagnosed by physical examination with and without the aid of echocardiography. The relative severity of each of the lesions is at times difficult to assess noninvasively even with the use of echocardiography, and frequently cardiac catheterization and angiocardiography are necessary. Often the presence of one lesion, for instance aortic regurgitation, masks the severity of another lesion, for instance mitral stenosis, and invasive studies can allow the estimation of the relative severity of each.

Recommendations. In general, patients with multiple valvular disease should not participate in any competitive sports. Recommendations should be based on the most hemodynamically severe lesion and should be compatible with those given for the isolated valvular lesions.

H. Postoperative Patients With a Prosthetic or Bioprosthetic Cardiac Valve

There are several general comments that apply to all patients with valve replacement. Although most patients improve after valve replacement and many enter New York Heart Association functional class I, the long-term mortality after recovery from the operative procedure is greater than that of control groups of similar age. A gradient of varying severity across the valve is found in most patients after valve replacement (12). Although hemodynamics at rest may be essentially normal after valve replacement, many patients have an abnormal response to exercise (13). Hemolysis in patients with abnormally functioning prosthetic valves does not appear to be produced by vigorous exercise.

There are insufficient data to determine whether vigorous repetitive exercise after valve replacement has any long lasting effect on ventricular function or prosthetic valve function (14). The patient should be made aware of these deficiencies in our knowledge before deciding whether to participate in competitive athletics.

Because prosthetic valves and most tissue valves have smaller effective valve areas than the normal valve and because the prosthetic valve poppets have a greater inertia than the normal valve, these valves perform best at normal heart rates. In patients with prosthetic valves, a sustained heart rate greater than 120 to 130 beats/min might result in increases in cardiac output that are less than would normally be expected and gradients that are higher than would be expected. Given these limitations, the following recommendations can be made.

a) Patients with mitral valve disease after mitral valve surgery. Valvuloplasty is possible in many patients with mitral stenosis and in some patients with mitral regurgitation, especially those with rupture of chordae tendineae of the posterior leaflet or prolapse of the posterior leaflet. After recovery from either closed or open mitral valvuloplasty the patient should have no disability associated with the operation if there has been no injury to the left ventricle or increase in mitral regurgitation.

Recommendations.

1. For postvalvuloplasty patients with mitral stenosis or occasionally with mitral regurgitation, the recommendations as to competitive sports should be based on the residual severity of the mitral stenosis or mitral regurgitation, in the same way as the preoperative patient. The capacity to engage in physical exercise should be evaluated with an exercise tolerance test. If left ventricular dysfunction is present, then this should be considered in the same way as in the unoperated patient.
2. Anuloplasty or valvuloplasty patients with mitral valve prolapse, because of the possibility of severe trauma disrupting the repair, should not engage in sports with a danger of body collision (class II). They may participate in sports of low intensity (class I.B) and in selected cases may engage in some sports with high to moderate dynamic and low static demands (class I.A.2).
3. Patients with a prosthetic or bioprosthetic valve not taking anticoagulant agents who have normal valvular function and normal or near normal left ventricular function may participate in low intensity sports (class I.B) and in some sports with moderate to high dynamic and low static demands (class I.A.2). Patients with prosthetic or bioprosthetic valves who are taking anticoagulant agents should not engage in sports with a danger of body collision (class II).

b) Patients with aortic valve disease after aortic valve surgery. The postoperative adult patient with aortic valve disease, with few exceptions, has had aortic valve replacement with either a prosthetic valve (requiring anticoagulant agents) or bioprosthetic valve (usually without anticoagulant agents). In assessing the patient's capacity for physical ac-

tivity, exercise stress testing to the level of activity performed in the competitive sport is valuable.

Recommendations.

1. Patients with a prosthetic or bioprosthetic aortic valve not taking anticoagulant agents with no valvular dysfunction and normal or near normal left ventricular function may engage in low intensity sports (class I.B) and selected patients may be allowed to participate in some sports with a high to moderate dynamic demand and a low static demand (class I.A.2) (14).
2. Patients with prosthetic or bioprosthetic valves taking anticoagulant agents should not engage in sports with a danger of body collision (class II).

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