

Original Scientific Paper

Recommendations for the management of individuals with acquired valvular heart diseases who are involved in leisure-time physical activities or competitive sports

Klaus Peter Mellwig^a, Frank van Buuren^a, Christa Gohlke-Baerwolf^b and Hans Halvor Bjørnstad^c

^aDepartment of Cardiology, Heart and Diabetes Center North Rhine-Westphalia, Ruhr University Bochum, Bad Oeynhausen, ^bHeart Center Bad Krozingen and ^cDepartment of Heart Disease, Haukeland University Hospital, Bergen, Norway

Received 28 February 2007 Accepted 2 July 2007

Physical check-ups among athletes with valvular heart disease are of significant relevance. In athletes with mitral valve stenosis the extent of allowed physical activity is dependant on the size of the left atrium and the severity of the valve defect. Patients with mild-to-moderate mitral valve regurgitation can participate in all types of sport associated with low and moderate isometric stress and moderate dynamic stress. Patients under anticoagulation should not participate in any type of contact sport.

Asymptomatic athletes with mild aortic valve stenosis can take part in all types of sport, as long as left ventricular function and size are normal, a normal response to exercise at the level performed during athletic activities is present and there are no arrhythmias. Asymptomatic athletes with moderate aortic valve stenosis should only take part in sports with low dynamic and static stress. Aortic valve regurgitation is often present due to connective tissue disease of a bicuspid valve. Athletes with mild aortic valve regurgitation, with normal end diastolic left ventricular size and systolic function can participate in all types of sport. A mitral valve prolapse is often associated with structural diseases of the myocardium and endocardium. In patients with mitral valve prolapse Holter-ECG monitoring should also be performed to detect significant arrhythmias. All athletes with known valvular heart disease, a previous history of infective endocarditis and valve surgery should receive endocarditis prophylaxis before dental, oral, respiratory, intestinal and genitourinary procedures associated with bacteraemia. Sport activities have to be avoided during active infection with fever. *Eur J Cardiovasc Prev Rehabil* 15:95–103 © 2008 The European Society of Cardiology

European Journal of Cardiovascular Prevention and Rehabilitation 2008, 15:95–103

Keywords: heart valve disease, recommendations, sports, sports medicine

Introduction

Several epidemiologic studies have assessed the relationship between physical exercise and the risk of sudden cardiac death (SCD) in atherosclerotic coronary artery disease, which is responsible for the vast majority of SCD in the middle-aged and older athletes. An increased risk of acute coronary events exist during exercise in individuals who exercise only occasionally, whereas

habitual sport activity may offer protection from the risk of acute myocardial infarction and sudden coronary death [1]. During the preparticipation screening, personal and family history and physical examination are obtained. In case a heart murmur or abnormalities in pulses, pathological blood pressure measurements, or stigmata of Marfan syndrome are detected, further cardiac evaluation is necessary. ECG should always be performed [1]. The specific valve lesion and its degree of severity are assessed clinically and by echocardiography. Echocardiography is the most important examination in athletes who are suspected of having valve disease. A chest radiograph might be necessary.

Correspondence to Dr Klaus-Peter Mellwig, MD, Department of Cardiology, Heart and Diabetes Center North Rhine-Westphalia, Georgstr. 11, 32545 Bad Oeynhausen, Germany
Tel: +49 5731 971258; fax: 49 5731 972194;
e-mail: kpmellwig@hdz-nrw.de

Exercise testing should be obtained after evaluation of the heart valves by echocardiography. This examination allows objective assessment of functional cardiopulmonary capacity. It might show in asymptomatic patients abnormal haemodynamics; particularly hypotension or inadequate rise in blood pressure, arrhythmias, marked ST-segment depression or inadequate exercise tolerance [2,3] or the development of symptoms not present during daily life activities. In certain cases, Holter-ECG may be useful to detect arrhythmias. Cardiac catheterization may occasionally be required in those patients in whom discrepancies between clinical and echocardiographic findings regarding the severity of valve defects occur.

The information available concerning the influence of physical activity on progression of valvular disease is scarce. Moreover, little is known about the influence of isometric effort on the progression of ventricular dysfunction, especially if such an activity is only carried out occasionally. In cases of coexistence of cardiac valve dysfunction and other cardiovascular abnormalities, such as arrhythmias or coronary heart disease, a recommendation should be expressed concerning physical activity, based on the strictest guidelines [4].

Mitral valve stenosis

Mitral valve stenosis (MVS) is usually of rheumatic origin. It accounts for 12% of valve diseases in the Euro Heart Survey [5]. MVS results in increased left atrial pressure, leading to pulmonary hypertension [6,7].

The increase in pulse rate and cardiac output associated with exercise results in a marked, acute increase in pulmonary capillary wedge pressure and can lead to acute pulmonary oedema. The long-term effects of recurrent increases in pulmonary capillary wedge pressure, pulmonary artery pressure and pulmonary arterial resistance as a result of physical activity has not been adequately studied, but these haemodynamic changes are certainly of detrimental effect on lungs and right ventricular (RV) function. In patients with severe MVS, particularly in those with atrial fibrillation and in the presence of an enlarged left atrium, cerebral and peripheral embolization may occur [8].

Evaluation and diagnostic testing

On physical examination the typical mitral face may be identified. On auscultation a loud first heart sound, a diastolic rumbling murmur and a mitral opening snap are heard and may allow the diagnosis.

The degree of severity as evidenced by mitral valve opening area can be determined by two-dimensional Echo and colour-flow Doppler. Further information

concerning calcification of the leaflets, the annulus, the subvalvular structures and left ventricular (LV) function can also be obtained by echocardiography [9]. Pulmonary arterial systolic pressure at rest and during exercise can be obtained by assessing maximum flow velocity in the presence of tricuspid regurgitation.

An exercise stress test should be carried out to determine heart rate at maximum exercise, pressure gradients and pulmonary artery pressure, and possible stress-dependent disorders of cardiac rhythm. This can also be helpful in patients with atrial fibrillation.

Invasive diagnostic procedures like Swan-Ganz catheterization under exercise is indicated only in selected cases but may be helpful in those patients in whom pulmonary artery pressure cannot be determined by echocardiography because of the absence of tricuspid insufficiency, or to detect an elevated pressure in the pulmonary artery during physical exercise. Athletes who develop a pulmonary artery systolic pressure greater than 60 mmHg during exercise are likely to develop severe adverse effects on RV function over time.

These patients should also be evaluated by assessing the influence of mitral valve regurgitation (MVR) to the calculation of mitral valve opening area of MVS. Patients who develop a pulmonary arterial systolic pressure of over 60 mmHg, under stress, are at risk of severe adverse effects on RV function, and require treatment. These patients should be evaluated for valvuloplasty before considering competitive sports [2].

Classification

The degree of haemodynamic severity of MVS is assessed on the basis of mitral valve opening area [2]. The figures of the opening area are not corrected by body surface area (BSA) but are in relation to the individual.

- Mild = mitral valve opening area greater than 1.5 cm².
- Moderate = mitral valve opening area between 1.0 and 1.5 cm².
- Severe = mitral valve opening area less than 1.0 cm².

Recommendations (Table 1)

1. Patients with sinus rhythm and mild MVS can participate in all types of sport, excluding high dynamic and/or high static in combination (Table 2).
2. Individuals with moderate or severe MVS and sinus rhythm or atrial fibrillation should only participate in low dynamic and low static types of sport. These patients should undergo further cardiac evaluation and appropriate medical treatment and/or percutaneous valvuloplasty or surgical procedures as indicated.

Table 1 Recommended level of physical activity for patients with valve disease

Lesion	Evaluation	Criteria for eligibility	Recommendations	Follow up
MVS	History. PE, ECG ET, Echo	Mild stenosis, stable sinus rhythm	All sports, with exception of high dynamic and high static (III C)	Yearly
		Mild stenosis in AF and anticoagulation	Low–moderate dynamic, low–moderate static (IA,B + IIA,B), no contact sport	Yearly
		Moderate and severe stenosis (AF or sinus rhythm)	Low dynamic and low static (IA), no contact sport	Yearly
MVR	History. PE, ECG ET, Echo	Mild-to-moderate regurgitation, stable sinus rhythm normal LV size/function, normal exercise testing. If AF on anticoagulation	All sports, with exception of contact sport	Yearly
		Mild-to-moderate regurgitation, mild LV dilatation (endsystolic volume < 26 ml/m ²) normal LV function, sinus rhythm	Low–moderate dynamic, low–moderate static (IA,B + IIA,B)	Yearly
		Mild to moderate regurgitation, LV enlargement (endsystolic volume > 26 ml/m ²) or LV dysfunction, (ejection fraction < 50%)	No competitive sports	
		Severe regurgitation	No competitive sports	
AVS	History. PE, ECG ET, Echo	Mild stenosis, normal LV size and function in rest and under stress, no symptoms, no significant arrhythmias	Low–moderate dynamic, low–moderate static (IA,B + IIA,B)	Yearly
		Moderate stenosis, normal LV function At rest and under stress!	Low dynamic and low static (IA)	Yearly
		Moderate stenosis, LV dysfunction at rest or under stress, symptoms	No competitive sports	
		Severe stenosis	No competitive sports	
AVR	History. PE, ECG ET, Echo	Mild-to-moderate regurgitation, stable sinus rhythm normal LV size/function, normal exercise testing no significant arrhythmia	All sports	Yearly
		Mild-to-moderate regurgitation, proof of progressive LV dilatation	Low dynamic and low static (IA)	Yearly
		Mild-to-moderate regurgitation. Significant ventricular arrhythmia at rest or under stress, dilatation of the ascending aorta	No competitive sports	
		Severe regurgitation	No competitive sports	
TVS	History. PE, ECG ET, Echo	No symptoms	Low–moderate dynamic, low–moderate static (IA,B + IIA,B)	Every second year
TVR	History. PE, ECG ET, Echo	Mild regurgitation	All sports	
		Moderate regurgitation	Low–moderate dynamic, low–moderate static (IA,B + IIA,B)	Yearly
		Any degree, with right atrial pressure > 20 mmHg	No competitive sports	
Poly-valvular diseases	History. PE, ECG ET, Echo	See most relevant defect		
Bioprosthetic aortic or mitral valve	History. PE, ECG ET, Echo	Normal valve function and normal LV function, in stable sinus rhythm	Low–moderate dynamic, low–moderate static (IA,B + IIA,B)	Yearly
		If anticoagulation	No contact sport	Yearly
MV reconstruction	ET, Echo	According to the remaining valve defect		Yearly
Prosthetic (artificial) aortic or mitral valve	History. PE, ECG ET, Echo	Normal valve function and normal LV function and anticoagulation	Low–moderate dynamic, low–moderate static (IA,B + IIA,B), no contact sport	Yearly
Post valvuloplasty	History. PE, ECG ET, Echo	See the residual severity of the MVS or MVR	Low–moderate dynamic, Low–moderate static (IA,B + IIA,B)	Yearly
Mitral valve prolapse	History. PE, ECG ET, Echo	If unexpected syncope, or family history of sudden cardiac death, or complex supra-ventricular or ventricular arrhythmias, or long QT interval, or severe mitral regurgitation, absence of the earlier cited cases	No competitive sports, all sports	Yearly

AF, atrial fibrillation; AVR, aortic valve regurgitation; AVS, aortic valve stenosis; ECG, electrocardiography; Echo, echocardiography; ET, exercise test; LV, left ventricular; MVR, mitral valve regurgitation; MVS, mitral valve stenosis; PE, physical examination; TVR, tricuspid valve regurgitation; TVS, tricuspid valve stenosis.

Concerning the treatment of atrial fibrillation see also Ref. [4] Patients in whom anticoagulation treatment has to be administered should not participate in any type of sport that is associated with a high probability of injury.

3. MVS associated with atrial fibrillation see also chapter arrhythmia.

Mitral valve regurgitation

MVR is the second most common valve lesion after aortic valve stenosis (AVS), and was present in 31% of all valve lesions encountered in the Euro heart survey [5].

The most frequent cause for MVR is a prolapse of the leaflets. Other aetiologies are rheumatic fever, infectious

Table 2 Classification of sports

	(A) Low dynamic	(B) Moderate dynamic	(C) High dynamic
I Low static	Bowling	Table tennis	Badminton
	Cricket	Tennis (doubles)	Race walking
	Golf	Volleyball	Cross-country skiing (classic)
	Riflery	Fencing	Running (marathon)
		Baseball ^a /softball ^a	Squash ^a
II Moderate static	Archery	Lacrosse ^a	Basketball ^a
	Auto racing ^{a,b}	Field events (jumping)	Ice hockey ^a
	Diving ^b	Figure skating ^a	Biathlon
	Equestrian ^{a,b}	Running (sprint)	Ice hockey ^a
	Motorcycling ^{a,b}		Field hockey ^a
	Sailing		Rugby ^a
	Gymnastics		Soccer ^a
	Karate/judo ^a		Cross-country skiing (skating)
			Running (mid/long)
			Swimming
III High static	Bobsledding ^{a,b}	Body building ^{a,b}	Boxing ^a
	Field events (throwing)	Downhill skiing ^{a,b}	Canoeing, kayaking
	Luge ^{a,b}	Wrestling ^a	Cycling ^{a,b}
	Rock climbing ^{a,b}	Snow boarding	Decathlon
	Waterskiing ^{a,b}		Rowing
	Weight lifting ^{a,b}		Speed skating
	Windsurfing ^{a,b}		Triathlon ^{a,b}

^aDanger of bodily collision. ^bIncreased risk if syncope occurs. Modified after Mitchell *et al.* Classification of Sports. JACC 1994;24:864–866.

endocarditis, coronary heart disease (ischaemic cardiomyopathy), dilated cardiomyopathy or connective tissue diseases, for example Marfan's syndrome. MVR leads to an increase in left atrial pressure and causes increased LV diastolic filling.

Isometric exercises are associated with raised arterial blood pressure, and therefore can worsen the regurgitation, and result in an increased left atrial pressure. It is conceivable that repeated intermittent increases in volume load during physical exercise may contribute to the progression of MVR although there are data available. The risk of atrial fibrillation increases with the extent of the left atrial dilatation.

Evaluation and diagnostic testing

A midsystolic click associated with a midsystolic to late systolic murmur is typically heard in MVR associated with mitral valve prolapse (MVP). ECG and chest radiograph provide additional information.

Echocardiography and colour flow Doppler echocardiography [10–12] are appropriate when assessing the degree of severity and valve morphology [13], the haemodynamic effects on the left ventricle (end systolic and end diastolic volume), left atrium (enlargement might lead to atrial fibrillation) and LV function.

When assessing the impact of volume load on the left ventricle by the mitral insufficiency, it should be taken into consideration that end diastolic LV size in well trained and healthy athletes is higher than in nonathletes.

The ability of the left ventricle to adapt to physical activity can also be shown by radionuclide angiography. Transesophageal echocardiography can provide important additional information on mitral valve morphology in those patients who are difficult to examine with transthoracic echocardiography. Yet it is usually not necessary for routine evaluation in mild-to-moderate MVR. A 24-h Holter ECG is recommended when arrhythmias are evident and when MVR is due to prolapse of the leaflets.

Classification

Several methods are present to define severity of MVR (mild, moderate or severe). The most commonly applied are the proximal isovelocity surface area (PISA)-method and the vena contracta method [10,12]. Furthermore, the sizes of left atrium and LV have to be considered.

Recommendations (Table 1)

1. Patients with mild-to-moderate MVR, maintained sinus rhythm, a normal exercise test, with normal LV size and function may participate in all types of sport (dynamic A–C; static I–III). Cardiac evaluation should be performed once a year.
2. Patients with mild-to-moderate MVR, sinus rhythm, mild LV dilatation (end diastolic diameter < 60 mm) and normal resting LV function may participate in all types of sports that are associated with low and moderate isometric and dynamic stress (dynamic A–B, static I–II). Cardiac evaluation should be performed once a year.
3. Individuals with mild-to-moderate MVR and marked LV enlargement (end diastolic diameter > 60 mm) or resting LV dysfunction (ejection fraction < 50%) should not participate in sports. These patients should undergo further evaluation.
4. Athletes with severe MVR should avoid any type of competitive sports
5. Patients with atrial fibrillation must receive anticoagulation treatment and they should not participate in sports associated with bodily contact (combatant sport) [5]

Aortic valve stenosis

Aortic valve stenosis (AVS) is the most common valve lesion, accounting for 42% of all native valve lesions in the Euro heart survey [5]. The cause in adults is often degenerative, followed by congenital (bicuspid valve) and rheumatic origin. Syncope, dizziness, angina pectoris or dyspnoe typically occur in severe AVS [14].

Exercise-induced syncope characterizes the high-risk patient with symptomatic AVS. This valvular defect was present in 4% of young athletes with SCD [15]. Syncope can also appear in young athletes with a mild degree of AVS. SCD is rare as long as patients remain asymptomatic [16–19].

AVS imposes a pressure load on the LV. The following compensatory mechanisms develop: (a) a rise in systolic ventricular pressure owing to an increase of LV muscle mass and contractility, and (b) a lengthening of the systolic ejection phase. Characteristic symptoms of LV dilatation and signs of myocardial insufficiency appear only when further compensation of the stenosis is not possible owing to a constant increase of pressure load or failure of the compensatory mechanisms.

Evaluation and diagnostic testing

AVS is often detected by characteristic findings at auscultation. The haemodynamic severity can be determined by two-dimensional and Doppler echocardiography. Physical stress testing (e.g. bicycle ergometry) is recommended to assess symptom-free exercise tolerance and haemodynamic response to exercise, especially with regard to development of ST-segment depressions, blood pressure response and possible arrhythmias. These tests are important to identify asymptomatic patients who may need further evaluation for surgery and who should be excluded from certain types of sport. Patients who develop symptoms, abnormal haemodynamic responses during exercise testing or arrhythmias require further evaluation for surgery. In patients with symptomatic severe AVS, physical stress testing should be avoided.

If clinical evaluation and noninvasive testing are contradictory in symptomatic patients, cardiac catheterization may be necessary to determine the severity of AVS. Coronary angiography is required in athletes older than 40 years to detect coronary artery disease — if valve surgery is considered. As AVS may progress, regular evaluation is necessary. The time interval for evaluation depends on the initial degree of severity.

Classification

Severity of AVS is assessed by two-dimensional and Doppler echocardiography determining mean valve gradient and especially valve opening area (AVA).

Mild = AVA greater than 1.5 cm^2 , mean gradient $\leq 20 \text{ mmHg}$

Moderate = AVA of $1.0\text{--}1.5 \text{ cm}^2$, mean gradient $21\text{--}49 \text{ mmHg}$

Severe = AVA less than 1.0 cm^2 , mean gradient $\geq 50 \text{ mmHg}$.

Recommendations (Table 1)

1. Asymptomatic individuals with mild AVS may take part in low–moderate dynamic and low–moderate static types of sport (dynamic A–B and static I–II), as long as LV function and size is normal, a normal response to exercise at the level performed during athletic activities is present and there are no arrhythmias.
2. Patients with a history of syncope, angina pectoris or dizziness, even if only mild aortic stenosis is present, should undergo further evaluation and should not participate in competitive sports.
3. Asymptomatic athletes with moderate AVS should only take part in sports with low dynamic and static stress (dynamic A; static I).
4. Patients with moderate aortic stenosis and LV dysfunction or marked LV hypertrophy ($> 15 \text{ mm}$), or patients with severe AVS and/or relevant dilatation of the ascending aorta should not participate in sports.
5. Symptomatic patients with significant AVS require further assessment for surgery and should not perform any athletic exercises.

Aortic valve regurgitation

Diseases affecting the aortic valve, the aortic valve ring or the ascending aorta can result in aortic regurgitation (AVR). The most frequent aetiology presently is an aneurysm of the ascending aorta. Other common causes are congenital bicuspid aortic valve, rheumatic fever, infectious endocarditis and aortic root disease including Marfan's syndrome. AVR was present in 13% of patients in the Euro heart survey on valvular heart disease [5].

AVR causes dilatation of the LV cavity with increases in LV diastolic and systolic volumes. Even severe AVR can be well tolerated without symptoms for many years. As LV dysfunction proceeds, symptoms occur, typically including dyspnoea and arrhythmias and, in advanced cases, angina.

In asymptomatic patients with normal LV function SCD is rare ($< 0.2\%/ \text{year}$), the development of LV dysfunction is uncommon ($< 1.3\%/ \text{year}$) and progression to symptoms, LV impairment or death occurs at a rate of $4.3\%/ \text{year}$ [20]. Predictors of outcome are age and LV end systolic diameter at initial evaluation and ejection fraction. It is unclear how competitive exercise training affects prognosis. Dynamic exercises acutely increases heart rate, which shortens diastole and the time available for aortic regurgitation. In contrast, regular endurance training induces bradycardia, which prolongs AVR and may induce LV dysfunction. Static exertion can worsen potential regurgitation by increasing afterload acutely.

Evaluation and diagnostic testing

On clinical examination, the aortic regurgitant murmur is of high frequency and begins immediately after the aortic component of the second heart sound. The severity correlates better with the duration than with the intensity of the murmur.

Structural abnormalities of the aortic valve, ascending aorta and LV size and function can be evaluated by echocardiography. That LV cavity dimension is increased in healthy athletes as a consequence of training, should be considered when assessing LV size in the presence of AVR. Echocardiographic evaluation of aortic regurgitation requires colour-flow imaging, continuous wave Doppler of the regurgitant jet, and pulse-wave Doppler of the mitral inflow.

Exercise testing should be performed to determine symptomatic status and physical fitness [2,21]. The level of exercise reached during testing should be equivalent to that of the desired sport.

No studies are available evaluating the long-term effect of physical activity on LV function in these patients. To identify progression of aortic regurgitation and/or occurrence of LV dysfunction, annual echocardiographic evaluation and exercise tests are recommended.

Classification

The severity of AVR is determined clinically and echocardiographically [13]. Several quantitative parameters, for example blood pressure amplitude, should be considered in the determination of the degree of severity of AVR and the effect of the volume load on LV dimension [2]. Moreover, the determination of the rate of decline in regurgitant gradient measured by the slope of diastolic flow velocity is helpful to evaluate the severity of aortic regurgitation [18]. The evaluation of AVR severity, using quantitative measurements, is less well established than in MVR, and consequently, the results of quantitative measurements should be integrated with other data to come to a final conclusion as regards severity [13].

Mild = absence of peripheral signs of AVR and normal LV and atrial size and function; small dimension of the diastolic flow signal on Doppler echocardiography.

Moderate = peripheral signs of AVR, mild-to-moderate enlargement of the LV, normal systolic function, moderate dimension of the diastolic flow signal on Doppler echocardiography.

Severe = peripheral signs of AVR, marked dilatation of the LV and/or evidence of LV dysfunction; enlarged atrial size and large dimension of the diastolic flow signal on Doppler echocardiography [10,17,18].

Recommendations (Table 1)

1. Patients with mild AVR, with normal end diastolic LV size and systolic function can participate in all types of sport (dynamic A–C; static I–III).
2. In asymptomatic moderate AVR and progressive LV dilatation, sports of dynamic group A and static group I may be carried out. Regular clinical and echocardiographic evaluations at half-yearly intervals are important.
3. Individuals with mild or moderate AVR and significant ventricular arrhythmia at rest or during exercise should not participate in competitive sports.
4. Patients with severe aortic regurgitation should not participate in competitive sports, irrespective of the LV function and should be evaluated for valve surgery.
5. Athletes with AVR and marked dilatation of the ascending aorta (> 50 mm) should not participate in competitive sports.

Tricuspid valve stenosis

Tricuspid valve stenosis (TVS) is usually caused by rheumatic fever and is almost always associated with MVS. Isolated TVS is rare [20]. Two-dimensional echocardiography is valuable in determining the cause of tricuspid stenosis and Doppler echocardiography is used to determine severity. In the presence of MVS and TVS, patients should be assessed with reference to the MVS.

Recommendations (Table 1)

If patients are asymptomatic, with normal exercise tests and normal LV and RV function and with mild-to-moderate valve stenosis these athletes can participate in low-moderate dynamic and static sport (dynamic A–B and static I–II).

Tricuspid valve regurgitation

Tricuspid valve regurgitation (TVR) is often the result of RV dilatation and pulmonary hypertension, most commonly owing to mitral valve disease. Other causes are rheumatic fever, tricuspid valve prolapse or infective endocarditis. Tricuspid regurgitation leads to volume overload of the right ventricle and increased systemic venous pressure [7]. No indication exists that athletes with isolated primary tricuspid valve insufficiency are exposing themselves to an acute risk if they perform physical activities. Yet the long-term effects of chronic volume overload of the right ventricle are unknown.

Evaluation and diagnostic testing

The diagnostic feature on clinical inspection is jugular venous distension. The systolic murmur is augmented during inspiration (Carvallo sign). The severity of tricuspid valve regurgitation can be determined noninvasively by physical examination and echocardiography.

Recommendations (Table 1)

1. Athletes with mild TVR can take part in all sports.
2. Patients with isolated moderate TVR may take part in low–moderate dynamic and low–moderate static (dynamic A–B and static I–II) type of sport.
3. Athletes with any degree of TVR associated with a right atrial pressure of more than 20 mmHg should avoid competitive sports.

Multivalvular disease

Multivalvular disease frequently occurs with chronic heart failure, rheumatic fever, myxomatous valvular diseases or infectious endocarditis. These patients are usually symptomatic and haemodynamically severely impaired. Clinical examination and echocardiography determines the extent of valve disease. Multivalvular disease of mild severity may reciprocally worsen each other for their haemodynamic effects and, therefore, great caution is needed in these patients with regard to physical activity.

Recommendations (Table 1)

Recommendations should be based on the haemodynamically most relevant defect.

Athletes after valve interventions: bioprosthetic or mechanical heart valves

Although valve replacement leads to improvement in symptoms and prognosis, life expectancy after valve surgery is less than in the general population of the same age. Despite marked improvements in valve design both bioprosthetic and mechanical heart valves have gradients at rest which represent mild valve stenosis. During exercise, gradients may increase. Therefore valve and LV function should be evaluated at rest and during exercise using stress echocardiography to the level of exercise the athlete wishes to pursue. All mechanical heart valves require lifelong oral anticoagulation which further limits their potential for sports participation. Regular cardiac evaluation including echocardiography and stress testing is advisable in all patients after valve surgery at yearly intervals [22].

Recommendations (Table 1)

1. Asymptomatic athletes with normal LV function and exercise tolerance after bioprosthetic or mechanical aortic valve replacement can participate in low–moderate intensity static and dynamic sports (dynamic A–B and static I–II).
2. Moreover, athletes with a bioprosthetic mitral valve who are not receiving anticoagulation treatment with normal valve and LV function may participate in mild-to-moderate dynamic and static types of sport.
3. Athletes with a mechanical or bioprosthetic mitral or aortic valve receiving oral anticoagulation should not participate in sports with any risk of bodily contact.

- To reduce the risk of haemorrhage, close control of anticoagulation intensity is recommended.
4. All athletes with significant valve dysfunction should not continue with competitive sports.

Percutaneous mitral valvuloplasty

Percutaneous mitral valvuloplasty is the treatment of choice in patients with pure noncalcified severe MVS. In 94% of patients, a valve area of more than 1.5 cm² can be obtained, with 77% of patients being asymptomatic after 9 years [5,23,24]. Concerning pathophysiology see MVS.

Recommendations (Table 1)

In patients after mitral valvuloplasty recommendations should be based on the residual degree of valve stenosis and/or valve regurgitation present after the procedure. An exercise test, including stress echocardiography, should be carried out to the level corresponding to the desired type of sport. Residual gradients at rest and during exercise, valve area, pulmonary artery pressure and LV function determine the eligibility for various sports activities.

Mitral valve reconstruction

In patients with MVR owing to MVP, the treatment of choice is mitral valve reconstruction. If performed before the establishment of chronic atrial fibrillation most patients are in sinus rhythm after valve surgery, thus oral anticoagulation is not necessary 3 months postoperatively. Depending upon the results of surgery, these athletes can participate in sport according to the remaining valve defect.

Homografts and Ross procedure

The Ross procedure [25] has been suggested by some groups as the procedure of choice in athletes with severe AVS without connective tissue diseases, bicuspid aortic valves or Marfan's Syndrome. Several high-level athletes have competed successfully after a Ross procedure [26], yet long term results are not available.

The level of exercise that could be performed by the athlete depends on the LV function and the associated arrhythmias, but it should not exceed moderate dynamic and static type of sport.

Moreover, homografts could be implanted in patients with severe aortic valve disease who wish to continue with competitive sports. Besides the inherent risk of reoperation, the long-term effect of competitive sports on valve function and survival is not known.

Mitral valve prolapse

A MVP is largely due to two possible aetiologies. The most common cause of MVP is the myxomatous

degeneration of the mitral valve leaflets, with accumulation of proteoglycans and collagen [27,28]. It most often occurs in patients of asthenic stature and shows a family clustering. Ischaemic cardiomyopathy and hypertrophic obstructive cardiomyopathy might also cause MVP [29].

MVP often accompanies MVR and for this reason echocardiography has to be performed. Disorders of cardiac rhythm, endocarditis, syncope or embolism, however, can also occur [30]. In patients with MVP Holter-ECG monitoring should also be performed, to detect significant arrhythmias [31].

Participation in sport is permitted provided the accompanying complications are respected (Table 1). In cases of unexplained syncope, family history of SCD, complex supraventricular or ventricular arrhythmias, long QT interval or severe mitral regurgitation, no sport should be performed. In absence of earlier cited circumstance, all sports are permitted.

Prophylaxis against endocarditis

Infective endocarditis is an endovascular, microbial infection of intracardiac structures facing the blood, including infections of the large intrathoracic vessels. The early lesion is a vegetation of variable size, although destruction, ulceration or abscess might follow. The increasing accuracy of echocardiography and therapeutic progress has contributed to prognostic improvement in the last few years.

Patients with previous history of infective endocarditis, with prosthetic heart valves or acquired valve disease are considered high-risk patients and should receive antibiotic prophylaxis when exposed to risk of bacteraemia in accordance with current recommendations [32–34].

Prophylaxis should be performed before dental, oral, respiratory, oesophageal, gastrointestinal and genitourinary procedures.

Dental hygiene is of relevance for prevention of infective endocarditis. As a general rule, all sports activity should be avoided when active infection with fever is present. Resumption of sports activity can be considered when the inflammatory process is completely extinguished according to recent guidelines [33,34].

References

- Corrado D, Pelliccia A, Bjornstad HH, Vanhees L, Biffi A, Borjesson M, *et al.* Cardiovascular preparticipation screening of young competitive athletes for prevention of sudden death: proposal for a common European protocol. Consensus Statement of the Study Group of Sport Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology. *Eur Heart J* 2005; **26**:516–524.
- lung B, Gohlke-Barwolf C, Tornos P, Tribouilly C, Hall R, Butchart E, *et al.* Recommendations on the management of the asymptomatic patient with valvular heart disease. *Eur Heart J* 2002; **23**:1252–1266.
- Balady GJ, Weiner DA. Exercise testing for sports and the exercise prescription. *Cardiol Clin* 1987; **5**:183–196.
- Pelliccia A, Fagard R, Bjornstad HH, Anastassakis A, Arbustini E, Assanelli D, *et al.* Recommendations for competitive sports participation in athletes with cardiovascular disease: a consensus document from the Study Group of Sports Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology. *Eur Heart J* 2005; **26**:1422–1445.
- lung B, Baron G, Butchart EG, Delahaye F, Gohlke-Barwolf C, Lewang OW, *et al.* A prospective survey of patients with valvular heart disease in Europe: The Euro Heart Survey on Valvular Heart Disease. *Eur Heart J* 2003; **24**:1231–1243.
- Gaasch WH, Eisenhauer AC. The management of mitral valve disease. *Curr Opin Cardiol* 1996; **11**:114–119.
- Bonow RO, Cheitlin MD, Crawford MH, Douglas PS. Task Force 3: valvular heart disease. *J Am Coll Cardiol* 2005; **45**:1334–1340.
- Sanada J, Komaki S, Sannou K, Tokiwa F, Kodera K, Terada H, *et al.* Significance of atrial fibrillation, left atrial thrombus and severity of stenosis for risk of systemic embolism in patients with mitral stenosis. *J Cardiol* 1999; **33**:1–5.
- Hatle L. Doppler echocardiographic evaluation of mitral stenosis. *Cardiol Clin* 1990; **8**:233–247.
- Mohr-Kahaly S, Erbel R, Zenker G, Drexler M, Wittlich N, Schaudig M, *et al.* Semiquantitative grading of mitral regurgitation by color-coded Doppler echocardiography. *Int J Cardiol* 1989; **23**:223–230.
- Mazur W, Nagueh SF. Echocardiographic evaluation of mitral regurgitation. *Curr Opin Cardiol* 2001; **16**:246–250.
- Zoghbi WA, Enriquez-Sarano M, Foster E, Grayburn PA, Kraft CD, Levine RA, *et al.* American Society of Echocardiography: recommendations for evaluation of the severity of native valvular regurgitation with two-dimensional and Doppler echocardiography. A report from the American Society of Echocardiography's Nomenclature and Standards Committee and the Task Force on Valvular Regurgitation, developed in conjunction with the American College of Cardiology Echocardiography Committee, The Cardiac Imaging Committee, Council on Clinical Cardiology, The American Heart Association, and the European Society of Cardiology Working Group on Echocardiography. *Eur J Echocardiogr* 2003; **4**:237–261.
- Vahanian A, Baumgartner H, Bax J, Butchart E, Dion R, Filippatos G, *et al.* Guidelines on the management of valvular heart disease: The Task Force on the Management of Valvular Heart Disease of the European Society of Cardiology. *Eur Heart J* 2007; **28**:230–268.
- Galan A, Zoghbi WA, Quinones MA. Determination of severity of valvular aortic stenosis by Doppler echocardiography and relation of findings to clinical outcome and agreement with hemodynamic measurements determined at cardiac catheterization. *Am J Cardiol* 1991; **67**:1007–1012.
- Maron BJ, Shirani J, Poliac LC, Mathenge R, Roberts WC, Mueller FO. Sudden death in young competitive athletes. Clinical, demographic, and pathological profiles. *JAMA* 1996; **276**:199–204.
- Pelikka PA, Nishimura RA, Bailey KR, Tajik AJ. The natural history of adults with asymptomatic, hemodynamically significant aortic stenosis. *J Am Coll Cardiol* 1990; **15**:1012–1017.
- Otto CM, Burwash IG, Legget ME, Munt BI, Fujioka M, Healy NL, *et al.* Prospective study of asymptomatic valvular aortic stenosis. Clinical, echocardiographic, and exercise predictors of outcome (see comments). *Circulation* 1997; **95**:2262–2270.
- Rosenhek R, Binder T, Porenta G, Lang I, Christ G, Schemper M, *et al.* Predictors of outcome in severe, asymptomatic aortic stenosis. *N Engl J Med* 2000; **343**:611–617.
- Pelikka PA, Sarano ME, Nishimura RA, Malouf JF, Bailey KR, Scott CG, *et al.* Outcome of 622 adults with asymptomatic, hemodynamically significant aortic stenosis during prolonged follow-up. *Circulation* 2005; **111**:3290–3295.
- Bonow RO, Carabello BA, Chatterjee K, de Leon AC Jr, Faxon DP, Freed MD, *et al.* ACC/AHA 2006 guidelines for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (writing Committee to Revise the 1998 guidelines for the management of patients with valvular heart disease) developed in collaboration with the Society of Cardiovascular Anesthesiologists endorsed by the Society for Cardiovascular Angiography and Interventions and the Society of Thoracic Surgeons. *J Am Coll Cardiol* 2006; **48**:e1–e148.
- Kim HJ, Park SW, Cho BR, Hong SH, Park PW, Hong KP. The role of cardiopulmonary exercise test in mitral and aortic regurgitation: it can predict post-operative results. *Korean J Intern Med* 2003; **18**:35–39.

- 22 Horstkotte D, Niehues R, Schulte HD, Strauer BE. Exercise capacity after heart valve replacement. *Z Kardiol* 1994; **83** (Suppl 3): 111–120.
- 23 Guerios EE, Bueno R, Nercolini D, Tarastchuk K, Andrade P, Pacheco A, *et al.* Mitral stenosis and percutaneous mitral valvuloplasty (part 1). *J Invasive Cardiol* 2005; **17**:382–386.
- 24 Guerios EE, Bueno R, Nercolini D, Tarastchuk K, Andrade P, Pacheco A, *et al.* Mitral stenosis and percutaneous mitral valvuloplasty (part 2). *J Invasive Cardiol* 2005; **17**:440–444.
- 25 Doty DB. Aortic valve replacement with homograft and autograft. *Semin Thorac Cardiovasc Surg* 1996; **8**:249–258.
- 26 Potera C. A return to football after heart surgery. *Phys Sportsmed* 1997; **25**:16.
- 27 Freed LA, Levy D, Levine RA, Larson MG, Evans JC, Fuller DL, *et al.* Prevalence and clinical outcome of mitral-valve prolapse. *N Engl J Med* 1999; **341**:1–7.
- 28 Nesta F, Leyne M, Yosefy C, Simpson C, Dai D, Marshall JE, *et al.* New locus for autosomal dominant mitral valve prolapse on chromosome 13: clinical insights from genetic studies. *Circulation* 2005; **112**:2022–2030.
- 29 Maron BJ, Ackerman MJ, Nishimura RA, Pyeritz RE, Towbin JA, Udelson JE. Task Force 4: HCM and other cardiomyopathies, mitral valve prolapse, myocarditis, and Marfan syndrome. *J Am Coll Cardiol* 2005; **45**: 1340–1345.
- 30 Kligfield P, Levy D, Devereux RB, Savage DD. Arrhythmias and sudden death in mitral valve prolapse. *Am Heart J* 1987; **113**:1298–1307.
- 31 Furlanello F, Durante GB, Bettini R, Vergara G, Disertori M, Cozzi F, *et al.* Risk of arrhythmia in athletes with mitral valve prolapse. *Cardiologia* 1985; **30**:987–989.
- 32 Horstkotte D, Follath F, Gutschik E, Lengyel M, Oto A, Pavie A, *et al.* Guidelines on prevention, diagnosis and treatment of infective endocarditis executive summary; the task force on infective endocarditis of the European society of cardiology. *Eur Heart J* 2004; **25**:267–276.
- 33 Naber CK, Al-Nawas B, Baumgartner H, Becker HJ, Block M, Erbel R, *et al.* Prophylaxe der infektiösen Endokarditis. *Kardiologe* 2007; **1**:243–250.
- 34 Wilson W, Taubert KA, Gewitz M, Lockhart PB, Baddour LM, Levison M, *et al.* Prevention of infective endocarditis. Guidelines from the American Heart Association: a guideline from the American Heart Association Rheumatic Fever, Endocarditis, and Kawasaki Disease Committee, Council on Cardiovascular Disease in the Young, and the Council on Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, and the Quality of Care and Outcomes Research Interdisciplinary Working Group. *Circulation* 2007; **116**:1736–1754.