AAM- Author accepted manuscript version, please cite VoR – version of record : Frank van Buuren et Al., *Athletes with valvular heart disease and competitive sports: a position statement of the Sport Cardiology Section of the European Association of Preventive Cardiology*, European Journal of Preventive Cardiology, Volume 28, Issue 14, November 2021, Pages 1569–1578, https://doi.org/10.1093/eurjpc/zwab058

Athletes with Valvular Heart Disease and Competitive Sports: A Position Statement of the Sport Cardiology Section of the European Association of Preventive Cardiology

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Word Count: 3857 (excluding references)

Abstract

This paper provides an overview of the recommendations from the Sports Cardiology section of the European Association of Preventive Cardiology (EAPC) on sports participation in individuals with valvular heart disease (VHD). The aim of these recommendations is to encourage regular physical activity including sports participation, with reasonable precaution to ensure a high level of safety for all affected individuals.

Valvular heart disease is usually an age-related degenerative process, predominantly affecting individuals in their fifth decade and onwards. However, there is an increasing group of younger individuals with valvular defects. The diagnosis of cardiac disorders during routine cardiac examination often raises questions about on-going participation in competitive sport with a high dynamic or static component and the level of permissible physical effort during recreational exercise.

Although the natural history of several valvular diseases has been reported in the general population, little is known about the potential influence of chronic intensive physical activity on valve function, left ventricular remodeling pulmonary artery pressure, and risk of arrhythmia. Due to the sparsity of data on the effects of exercise on VHD, the present document is largely based on clinical experience and expert opinion.

Key words

Valvular heart disease; Physical activity; Sport; Recommendations; Pulmonary pressure; Mitral valve prolapse; Aortic regurgitation; Aortic stenosis; Mitral regurgitation; Mitral stenosis; Exercise

Introduction

Valvular heart disease (VHD) is usually an age-related degenerative process, predominantly affecting individuals in their fifth decade and onwards. Regular exercise should be encouraged in the elderly population with VHD to avoid physical deconditioning and frailty whilst balancing the risks and consequences of the possibility of accelerated progression of VHD. However, there is a pertinent population of younger individuals with congenital VHD, many of whom are asymptomatic, and some aspire to participate in competitive and leisure exercise[1] Middle-aged and older individuals constitute up to 40% of participants in mass endurance events and a significant proportion will have asymptomatic VHD. The diagnosis of VHD in an athlete during routine or pre-participation cardiac examination often raises questions about on-going participation in competitive sport with a high dynamic or static component and the level of permissible physical effort during recreational exercise. Although the natural history of several valvular diseases has been reported in the general population, little is known about the potential influence of chronic intensive physical activity on the progression of VHD. It is theoretically possible however, that an increased cardiac preload and afterload during exercise could accelerate valvular dysfunction, resulting in adverse cardiac remodeling, pulmonary hypertension and a greater propensity to cardiac arrhythmias. Since the previous European Society of Cardiology recommendations on the management of VHD over a decade ago [2,3], there have been a plethora of novel reports on the investigation and risk stratification of athletes with VHD which necessitates an update [4–11].

These recommendations for exercise in athletes with VHD focus on competitive athletes. Practical guidance for recreational athletes and mildly symptomatic patients with mild and moderate VHD is presented elsewhere[12 13] The present document is based on available scientific knowledge in the literature, clinical experience and expert opinion.

Classification of Sport

For the purposes of this document and consistent with exercise recommendations in athletes with other cardiovascular diseases, sport disciplines have been divided into 4 groups, notably skill sports, power sports, mixed sports and endurance sports in accordance with their haemodynamic responses and their long-term impact on cardiac output and remodeling (Figure 1) [14–16]. Although purely dynamic exercise is associated with a predominant increase in volume load to the ventricle and static exercise results in a predominant pressure load, most sport and the training associated with these disciplines consist of both dynamic and static components [17]. It is also true that even within a given sporting discipline, the cardiovascular load varies based on the intensity and duration of exercise, the environmental conditions, and the different role of the athlete in an organized team. The classification of sports described in Figure 1 is used to provide exercise recommendations for the different valvular defects.

General Evaluation of Athletes with VHD

The majority of athletes with VHD are asymptomatic and detected during physical examination or after investigation for an abnormal ECG. Athletes with advanced VHD may present with chest discomfort during exertion, breathlessness that is disproportionate to the amount of exercise being performed, exertional dizziness, palpitation or syncope (Figure 2). Valve defects with a predominantly regurgitant component are often better tolerated than stenotic lesions [18]. Athletes with VHD should be evaluated 6 monthly to 2-yearly based on symptoms and/or severity of valve disease. Transthoracic echocardiography (TTE) is the first line investigation to confirm the diagnosis of VHD and provides essential prognostic information in relation to severity of valve dysfunction, ventricular function and pulmonary artery pressure [19,20].

All athletes with mild to moderate VHD should have an exercise stress test or cardiopulmonary exercise test using a protocol that closely resembles the level of exertion expected from the type of sport wished to be engaged in to assess functional capacity, blood pressure response to

exercise and the presence or absence of myocardial ischaemia and arrhythmias. Exercise echocardiography and 3-D echocardiography may provide helpful additional information to quantify the severity of valve dysfunction and may be used if echocardiography is produces equivocal results.

Cardiac magnetic resonance (CMR) can also contribute additional value as it can quantify regurgitant volumes, identify the presence of left ventricular myocardial fibrosis[4,5] and provide accurate quantification of biventricular volumes and function. Transoesophageal echocardiography (TOE) should also be considered when TTE is of suboptimal quality and in athletes with prosthetic valve dysfunction.

Mitral Valve Stenosis (MS)

Mitral valve stenosis (MS) is mostly due to rheumatic heart disease. During exercise, the increase in heart rate and left atrial filling is associated with a high-pressure gradient across the mitral valve which may result in dyspnea and may even cause acute pulmonary oedema in advanced disease. Chronic elevation of pulmonary venous pressure is associated with pulmonary hypertension and right ventricular hypertrophy[19,21]. The long-term effects of regular intensive exercise on the progression of MS, the pulmonary vasculature and the right ventricle are unknown although exercise may exacerbate left atrial enlargement predisposing to atrial fibrillation [22].

Evaluation. Presence of MS may be detected by a characteristic mid diastolic rumbling murmur during auscultation. The severity of MS is generally determined by TTE. The severity of MS is defined by the mitral valve area and pulmonary artery pressure (Table 1a, Appendix). Valve area measurement using planimetry is the reference technique for the evaluation of MS, taking into consideration the challenges for its assessment [19], whereas mean transvalvular gradient and pulmonary pressure reflect its consequences and have a greater prognostic value [19].. Pulmonary

systolic arterial pressure is assessed by Doppler-echocardiography in the presence of tricuspid regurgitation. Exercise testing (or cardiopulmonary testing) can provide additional information regarding hemodynamic response and occurrence of arrhythmias (particularly atrial fibrillation). Stress echocardiography provides additional objective information by assessing changes in mitral gradient and pulmonary artery pressure [3,19,23]. Individuals who develop a severe increase of pulmonary artery systolic pressure (sPAP) i.e >40mmHg during exercise are likely to develop adverse effects on RV function over time. Invasive pressure monitoring is indicated only in selected cases, when accurate assessment of pressure in the pulmonary circulation is needed for therapeutic purposes. Individuals with severe symptomatic MS requires percutaneous mitral commissurotomy as first line-treatment in cases of favorable anatomy or alternatively mitral valve replacement. The likeliness of restenosis after valvuloplasty is low particularly in young individuals and demonstrates favorable outcome in comparison with prosthesis related complications.[24] For classification of severity of MS see Table 1a, Appendix.

Recommendations.

Asymptomatic athletes with mild MS may compete in all sports if the resting sPAP is <40 mmHg (Figure 3). Asymptomatic athletes with moderate MS (MVA 1.0-1.5 cm^2) may participate in competitive sports involving low intensity and recreational sport involving low and moderate intensity when the resting sPAP is <40mmHg and an exercise stress test shows good functional capacity and normal blood pressure response[12,25]. Individuals with severe MS or those with a resting sPAP >40 mmHg should avoid all competitive sport. Athletes with MS who are anticoagulated for AF should not participate in collision sports. Recommendations for sports participation in individuals who have undergone mitral valve annuloplasty are based on the residual degree of severity of stenosis. Asymptomatic individuals with mitral valve replacement

may participate in skill and mixed sporting disciplines provided valve function is satisfactory and there is no evidence of haemodynamic disturbance.

Mitral Regurgitation

Primary MR is often due to mitral valve prolapse (MVP), degenerative mitral valve disease, infective endocarditis or rheumatic heart disease.

Secondary MR may also occur due to failure of coaptation of the valve leaflets in cardiomyopathy and ischaemic heart disease (secondary MR). MR is associated with an elevated left atrial pressure, and increased preload on the left ventricle resulting in progressive compensatory increase in left ventricular size as the regurgitant jet becomes more severe [22,26].

Mitral valve prolapse

MVP is the most frequent cause of primary MR and may be associated with single or bi-leaflet protrusion into the left atrium of at least 2 mm beyond the long-axis annular plane [22,27]. Rhythm disorders (i.e. brady- or tachyarrhythmias), endocarditis, syncope or systemic thromboembolism are more frequent in these individuals than the general population [19]. Arrhythmic sudden cardiac death (SCD) in MVP is rare (0.2-0.4% per annum) and more common in women, occurs regardless of the degree of regurgitation, and is often associated with bileaflet prolapse, mitral annulus disjunction (MAD) and myocardial fibrosis in the papillary muscles[4,5]. The presence of syncope, T wave inversion in the inferior leads and ventricular extrasystoles conduction with right bundle branch block morphology and a superior axis should raise suspicion for myocardial fibrosis and along with MAD, are an indication for CMR.[4]

Evaluation. The majority of individuals with mild or moderate MVR are asymptomatic [23] and identified during routine auscultation or incidentally during echocardiography. The severity of MR can be assessed by 2-dimensional echocardiography with colour flow and Doppler studies. Three-dimensional echo can provide additional information in patients with complex valve lesions. TOE or cardiac MRI are advocated when TTE cannot quantify the severity of MR accurately [23,28]. The severity of MR is classified according to the regurgitant jet (Table 2a, Appendix). Exercise recommendations in athletes should also consider left ventricular size and function and pulmonary artery pressure. It is important to consider that some athletes, particularly males participating in endurance sport may show a physiologically enlarged left ventricular cavity size that would raise suspicion of severe volume overload in the context of MR. In such cases decision making should be based upon functional capacity, myocardial reserve and pulmonary artery pressure. CMR may be more useful for quantifying the regurgitant volume in such instances and is helpful to detect associated myocardial fibrosis.

In athletes with primary MR a LV end-diastolic diameter index with a cut-off of 35 mm/m^2 in men and 40mm/m^2 in women turned out to be useful to identify individuals with LV enlargement of clinical relevance[29], however, a case by case assessment is recommended in such instances when prescribing exercise. A significant increase of pulmonary artery pressure with exercise of >50mmHg has been reported to be of prognostic value [21]. An exercise stress test is recommended in athletes with MR to check functional capacity, haemodynamic response and complex arrythmias.

24-h Holter monitoring is recommended to identify complex ventricular arrhythmias in MVP. The risk of SCD is independent of severity of MR, therefore, risk stratification in these individuals is challenging. Risk factors for arrhythmic SCD in MVP family history of premature sudden cardiac death, inverted T waves in the inferior leads, VPBs with RBBB morphology at rest or during

exercise, mitral disjunction, NSVT, and in some cases late Gadolinium enhancement in the basal inferior wall on cardiac MRI. For classification of severity of MR, see Table 2a; Appendix.

Recommendations:

Asymptomatic athletes with mild MR may participate in all competitive sports (Figure 3). Asymptomatic athletes with moderate MR may also compete in all sports if the LVEDD is ≤ 60 mm (or 35mm/m^2 in males and 40 mm/m^2 in females), the LV ejection fraction is ≥60% and resting pulmonary artery pressure is <50 mmHg and a maximal exercise stress test shows excellent functional capacity, normal haemodynamic response and absence of significant ventricular arrhythmias during an exercise test.

Athletes with MVP and mild to moderate regurgitation can engage in all competitive sport in the absence of T-wave inversion in the inferior leads on the 12-lead ECG, ventricular arrhythmias on 24h-Holter ECG, and family history of SCD Athletes anticoagulated for atrial fibrillation should avoid collision sports.

Aortic Valve Stenosis

Aortic stenosis (AS) is most commonly due to degenerative calcific stenosis and usually affects the elderly population. Progressive disease causes mechanical obstruction to left ventricular outflow resulting in compensatory left ventricular hypertrophy and impaired diastolic function. Left ventricular size is usually within normal limits although an enlarged left atrial diameter may reflect hemodynamic burden in patients with asymptomatic severe aortic stenosis [30,31]. Severity of AS is determined by the pressure gradient across the aortic valve as well as valve opening area [32]. Symptoms of syncope or presyncope usually precede angina pectoris or dyspnea which

usually appear in a late stage of the disease. Occurrence of SCD is far more probable in symptomatic athletes.

Bicuspid Aortic Valve

Congenital bicuspid aortic valve (BAV) affects 1-2% of the general population with a higher male predisposition and may be complicated by aortic stenosis or regurgitation in young individuals [13,33–35]. Approximately 50% of individuals with a BAV are also at increased risk of aortopathy [34]. Over one third of BAV individuals develop significant aortic stenosis and/or aortic regurgitation in the fifth decade. In patients with aortopathy the risk of aortic aneurysm, dissection or rupture is 0.1% per annum [34]. The long-term significance of intensive exercise on the AV and aortic root are unknown. Medium term studies have shown that competitive exercise does not appear to have a detrimental effect on the AV or left ventricular morphology and function[32,36]. Similarly, a medium term (5-year follow up) study in a small cohort of athletes showed no differences in aortic root dimensions in athletes with BAV compared with athletes with a trileaflet AV[7]. In a study with BAV elite athletes, the annual growth rates for sinuses of Valsalva were 0.11 ± 0.59 mm and 0.21 ± 0.44 mm for proximal ascending aorta over 3 year follow up [11]. Moreover, aortic regurgitation was the only functional abnormality, but no significant progression was found. A normally functioning BAV usually does not represent a limit for competitive sport [37]. However, affected athletes require regular clinical surveillance to monitor for progressive valvular dysfunction and aortic root dilatation. Athletes with a BAV and associated AS/AR should be managed similar to individuals with trileaflet AS or AR. Several factors influence aortic root dimensions, including body size, age, height, gender, and blood pressure control [38] An abnormally enlarged aortic root is defined as range as an aortic root with a Z-score ≥2 which takes these factors into consideration.[39] Raw and corrected aortic measures at all levels were significantly greater in sports with a high dynamic component in both sexes. Among athletes, an aortic root > 40 mm in males (1.8%) and >34 mm in females (1.5%) is uncommon and unlikely to

represent the physiological consequence of exercise training [10,40,41]. Another study from the British athletes suggest that the upper limit for aortic diameter (sinuses of Valsalva) in females is 38 mm [40]. A rapid increase in aortic dilatation (≥0.5 cm/year) may also be suggestive of an aortopathy [42].

Evaluation. AS is frequently detected by auscultation. Determination of the pressure gradient across the aortic valve and the aortic valve area are initially assessed by Doppler-echocardiography. Exercise testing is recommended in individuals with mild to moderate or moderate AS assess for myocardial ischaemia, haemodynamic response and arrhythmias. Stress echocardiography to evaluate the dynamic component of valvular abnormalities and to unmask subclinical myocardial dysfunction that could be missed at rest is of utmost importance in individuals with at least moderate AS [43–45]. In patients with reduced systolic function and a valve opening area < 1.5 cm^2 the use of stress echocardiography is often helpful when attempting to differentiate true severe aortic stenosis from pseudo-severe aortic stenosis and provides guidance for adequate therapy through evaluating the contractile reserve of the left ventricle [32,46]. For classification of severity of AS see Table 1b; Appendix.

Recommandations: Asymptomatic athletes with mild AS and normal LV size and LV function may participate in all competitive sports (Figure 3). Asymptomatic athletes with moderate AS and normal LV function, who show good functional capacity, normal haemodynamic response and absence of complex arrhythmias during a maximal exercise test, may also engage in all competitive sport. Athletes with moderate AS and coexisting symptoms, LV dysfunction at rest or under stress and complex ventricular arrhythmias should be advised to refrain from both competitive and recreational sport but may perform the usual physical activity recommendations to maintain cardiovascular health.

In the context of BAV, individuals with an aortic root above the normal range or ≥43mm in tall men and ≥39mm in tall women should not participate in sports associated with increased loading conditions on the aorta such as power lifting and isometric exercises[12,47] The general recommendations advise that in individuals with BAV and an aortic root dimension of 45-50 mm only skilled or mixed or low intensity endurance sports are recommended.[12]

Aortic Valve Regurgitation

Recognised causes of AR include BAV, rheumatic fever, infective endocarditis, Marfan's syndrome, aortic dissection, systemic arterial hypertension and rheumatoid spondylitis. AR causes dilatation of the LV cavity with increases in LV diastolic and systolic volumes. Bradycardia can worsen the hemodynamic pattern, due to lengthening of the diastolic duration and increase of the regurgitant volume. AR causes both pressure and volume loading of the left ventricle. Isometric exercise is associated with increased aortic wall tension and may worsen AR [48,49]. Athletes with chronic AR can remain asymptomatic for many years. Once LV function deteriorates, there may be a rapid onset of symptoms including dyspnea on exertion, arrhythmias and, in advanced cases, angina. In patients with AR, static exercise causes a reduction of preload resulting in a reduction in LV stroke volume and regurgitant volume [50]. Assessment of LV enlargement can be challenging in athletes with AR as the valve defect itself and also training can induce an enlarged cavity size. As LV dysfunction proceeds, symptoms occur, typically including dyspnea on exertion, arrhythmias and, in advanced cases, angina [18,51].

Evaluation. AR severity determined by the colour flow regurgitant jet and continuous wave signal jet (see Table 2b; Appendix for classification of severity of AR) and flow reversal with the descending aorta. CMR is recommended as a complementary tool for the accurate assessment of AR severity and aortic valve morphology in cases where image quality is suboptimal by echocardiography.[52,53] Furthermore, tissue characterization by cardiac MRI may offer incremental diagnostic information.[54] LV size is also generally assessed by TTE. In consideration

that LV cavity dimension is increased in healthy athletes as a consequence of training, this should be considered when assessing LV size in the presence of AR.

Exercise testing (or cardiopulmonary testing) can be helpful for evaluating of functional capacity, haemodynamic response and inducible cardiac arrhythmias.

Recommendations:

Athletes with mild AR, normal LV size and function and normal exercise testing with no arrhythmia can do all sports (Figure 1). Those with moderate AR with a non-dilated LV and a left ventricular ejection fraction >50%, a good functional capacity and an aortic root <43mm may engage in moderate intensity sport. Athletes with severe AR may engage in low to moderate intensity sport. However, a case by case evaluation is recommended in such instances as individuals with a mildly enlarged left ventricular cavity (LVEDD 55-65 mm) and normal functional capacity may be permitted to do moderate intensity sport. Individuals with severe AR with progressive LV dilatation or ventricular arrhythmia at rest or under stress should not engage in competitive sports.

Tricuspid Valve Stenosis

Tricuspid valve stenosis (TS) is commonly due to rheumatic fever and is frequently associated with MS [55]. Isolated TS is rare. In athletes with coexisting MS and TS, recommendations should be based on the severity of MS. Asymptomatic athletes with mild TS may participate in all sports. Grading of TS is challenging. However, a mean gradient pressure gradient across the tricuspid valve ≥5mmHg at normal heart rate is considered indicative of clinically significant TS [19].

Recommendations:

Asymptomatic athletes with mild TS and normal ventricular function may participate in all sports. Individuals with moderate or severe stenosis should avoid sports.

Tricuspid Valve Regurgitation

Tricuspid regurgitation (TR) may be due to acquired disease such as rheumatic fever, infective endocarditis and carcinoid syndrome or congenital disease such as Ebstein's anomaly. TR in most cases co-exists or is functional secondary to right ventricular dilatation from other cardiac pathologies including left sided VHD with co-existent raised sPAP >40mmHg and systolic dysfunction. The severity of TR is generally determined by TTE. Dense and triangular early peaking regurgitant signal on colour flow, vena contracta flow, proximal isovelocity surface area (PISA), early diastolic filling velocity and RV outflow velocity are established echocardiographic parameters to assess the severity of TR [3,56] (see Table 2c; Appendix for classification of its severity).

Recommendations:

Individuals with mild TR may participate in all sports. Individuals with moderate TR with normal biventricular systolic function, normal exercise testing, and a systolic pressure (PAP) < 40 mmHg at rest, all sports are allowed. Individuals with any degree of TR, sPAP > 50 mmHg at rest and right ventricular dysfunction may participate in low intensity sport only. Individuals with a right atrial pressure of more than 20mmHg and any degree of TR should avoid competitive sports [3,57].

Multi-valvular diseases

Multi-valvular diseases frequently occur in connection with rheumatic fever, myxomatous valvular diseases or infective endocarditis. These conditions can be assessed quantitatively by Doppler-echocardiography. Deterioration of valvular disease entity may impact on other abnormal valves with unfavorable hemodynamic effects [19,58]. Therefore, close follow-up is warranted in these athletes with regard to participation in competitive sports. Exercise recommendations are governed by the most severe valvular abnormality.

Prosthetic/Bioprosthetic Heart Valves and/or valve repair in Exercising Individuals

Although patients improve clinically after heart valve replacement, the long-term mortality is higher than in a healthy control population. Furthermore, patients with normal hemodynamic patterns at rest may have abnormal values under physical stress. A proportion of individuals warranting surgical correction for mitral regurgitation may undergo valve repair instead of replacement. There are no data on the natural history of a valve replacement or repair in individuals who exercise intensively, therefore the current consensus recommendations are relatively conservative. Therefore, exercise testing, should be performed up to the intensity consistent with that of the sport the athlete wishes to pursue. Given that artificial valves are associates some flow limitation, we recommend that athletes who have had valve replacements have the same exercise limitations as asymptomatic athletes with moderate native valve disease provided ventricular function is preserved and pulmonary artery pressure is within normal limits [58,59]. Often anticoagulation is mandatory for mechanical prosthesis and those with atrial fibrillation which further limits their choice of competitive sports. As with native valves, athletes with prosthetic valves or valve repair should undergo annual reevaluation (Figure 2).

Prophylaxis for Infective Endocarditis

Patients with previous history of infective endocarditis, patients with prosthetic heart valves or congenital valve disease are considered high risk patients and should receive antibiotic prophylaxis when exposed to risk of bacteremia in accordance with the ESC recommendations[60] and systematic maintenance of endocarditis prophylaxis must be strictly observed. Non-specific hygiene measures should be applied in all patients with valvular heart disease.

Declaration of conflicting interests

The authors declare no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Declaration of Authors contributions

FVB, SG, SS & MP contributed to the conception/design of the work, acquisition, analysis, interpretation of data and drafting of the manuscript.
PA, JN, AP, VR, MB, FC, ES, HH, SC, DC, LS, AB, AP, CS, NP, HR, ALG, LF, NB, FDA, KPM critically revised the manuscript.
All authors give final approval and agree to be accountable for the integrity and accuracy of the manuscript.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

References:

1. Nkomo VT, Gardin JM, Skelton TN, Gottdiener JS, Scott CG, Enriquez-Sarano M. Burden of valvular heart diseases: a population-based study. *Lancet (London, England)*. 2006;368:1005–1011.

2. Pelliccia A, Fagard R, Bjornstad HH, Anastassakis A, Arbustini E, Assanelli D, Biffi A, Borjesson M, Carre F, Corrado D, Delise P, Dorwarth U, Hirth A, Heidbuchel H, Hoffmann E, Mellwig KP, Panhuyzen-Goedkoop N, Pisani A, Solberg EE, van-Buuren F, Vanhees L, Blomstrom-Lundqvist C, Deligiannis A, Dugmore D, Glikson M, Hoff PI, Hoffmann A, Hoffmann E, Horstkotte D, Nordrehaug JE, Oudhof J, McKenna WJ, Penco M, Priori S, Reybrouck T, Senden J, Spataro A, Thiene G. 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 My. *Eur Heart J*. 2005;26:1422–1445.

3. Mellwig KP, Van Buuren F, Gohlke-Baerwolf C, Bjørnstad HH. Recommendations for the management of individuals with acquired valvular heart diseases who are involved in leisure-time physical activities or competitive sports. *Eur J Prev Cardiol*. 2008;15:95–103.

4. Basso C, Iliceto S, Thiene G, Perazzolo Marra M. Mitral Valve Prolapse, Ventricular Arrhythmias, and Sudden Death. *Circulation*. 2019;140:952–964.

5. Basso C, Perazzolo Marra M, Rizzo S, De Lazzari M, Giorgi B, Cipriani A, Frigo AC, Rigato I, Migliore F, Pilichou K, Bertaglia E, Cacciavillani L, Bauce B, Corrado D, Thiene G, Iliceto S. Arrhythmic Mitral Valve Prolapse and Sudden Cardiac Death. *Circulation*. 2015;132:556–566.

6. Tzemos N, Therrien J, Yip J, Thanassoulis G, Tremblay S, Jamorski MT, Webb GD, Siu SC. Outcomes in adults with bicuspid aortic valves. *JAMA*. 2008;300:1317–1325.

7. Galanti G, Stefani L, Toncelli L, Vono MCR, Mercuri R, Maffulli N. Effects of sports activity in athletes with bicuspid aortic valve and mild aortic regurgitation. *Br J Sports Med*. 2010;44:275–279.

8. Harris KM, Tung M, Haas TS, Maron BJ. Under-recognition of aortic and aortic valve disease and the risk for sudden death in competitive athletes. J. Am. Coll. Cardiol. 2015;65:860–862.

9. Spataro A, Pelliccia A, Rizzo M, Biffi A, Masazza G, Pigozzi F. The natural course of bicuspid aortic valve in athletes. *Int J Sports Med*. 2008;29:81–85.

10. Boraita A, Heras M-E, Morales F, Marina-Breysse M, Canda A, Rabadan M, Barriopedro M-I, Varela A, de la Rosa A, Tunon J. Reference Values of Aortic Root in Male and Female White Elite Athletes According to Sport. *Circ Cardiovasc Imaging*. 2016;9.

11. Boraita A, Morales-Acuna F, Marina-Breysse M, Heras M-E, Canda A, Fuentes M-E, Chacon A, Diaz-Gonzalez L, Rabadan M, Parra Laca B, Perez de Isla L, Tunon J. Bicuspid aortic valve behaviour in elite athletes. *Eur Heart J Cardiovasc Imaging*. 2019;20:772–780.

12. Pelliccia A, Sharma S, Gati S, Bäck M, Börjesson M, Caselli S, Collet J-P, Corrado D, Drezner JA, Halle M, Hansen D, Heidbuchel H, Myers J, Niebauer J, Papadakis M, Piepoli MF, Prescott E, Roos-Hesselink JW, Graham Stuart A, Taylor RS, Thompson PD, Tiberi M, Vanhees L, Wilhelm M. 2020 ESC Guidelines on sports cardiology and exercise in patients with cardiovascular disease: The Task Force on sports cardiology and exercise

in patients with cardiovascular disease of the European Society of Cardiology (ESC). *Eur Heart J* [Internet]. 2020;Available from: https://doi.org/10.1093/eurheartj/ehaa605

13. Gati S, Malhotra A, Sharma S. Exercise recommendations in patients with valvular heart disease. *Heart*. 2019;105:106–110.

14. Niebauer J, Borjesson M, Carre F, Caselli S, Palatini P, Quattrini F, Serratosa L, Adami PE, Biffi A, Pressler A, Schmied C, van Buuren F, Panhuyzen-Goedkoop N, Solberg E, Halle M, La Gerche A, Papadakis M, Sharma S, Pelliccia A. Recommendations for participation in competitive sports of athletes with arterial hypertension: a position statement from the sports cardiology section of the European Association of Preventive Cardiology (EAPC). *Eur Heart J*. 2018;39:3664–3671.

15. Borjesson M, Dellborg M, Niebauer J, LaGerche A, Schmied C, Solberg EE, Halle M, Adami E, Biffi A, Carre F, Caselli S, Papadakis M, Pressler A, Rasmusen H, Serratosa L, Sharma S, van Buuren F, Pelliccia A. Recommendations for participation in leisure time or competitive sports in athletes-patients with coronary artery disease: a position statement from the Sports Cardiology Section of the European Association of Preventive Cardiology (EAPC). *Eur Heart J*. 2019;40:13–18.

16. Pelliccia A, Solberg EE, Papadakis M, Adami PE, Biffi A, Caselli S, La Gerche A, Niebauer J, Pressler A, Schmied CM, Serratosa L, Halle M, Van Buuren F, Borjesson M, Carre F, Panhuyzen-Goedkoop NM, Heidbuchel H, Olivotto I, Corrado D, Sinagra G, Sharma S. Recommendations for participation in competitive and leisure time sport in athletes with cardiomyopathies, myocarditis, and pericarditis: position statement of the Sport Cardiology Section of the European Association of Preventive Cardiology (EAPC). *Eur Heart J*. 2019;40:19–33.

17. Levine BD, Baggish AL, Kovacs RJ, Link MS, Maron MS, Mitchell JH. Eligibility and Disqualification Recommendations for Competitive Athletes With Cardiovascular Abnormalities: Task Force 1: Classification of Sports: Dynamic, Static, and Impact: A Scientific Statement From the American Heart Association and American Colle. *Circulation*. 2015;132:e262-6.

18. Maganti K, Rigolin VH, Sarano ME, Bonow RO. Valvular heart disease: diagnosis and management. *Mayo Clin Proc* [Internet]. 2010;85:483–500. Available from: https://www.ncbi.nlm.nih.gov/pubmed/20435842

19. Baumgartner H, Falk V, Bax JJ, De Bonis M, Hamm C, Holm PJ, Iung B, Lancellotti P, Lansac E, Rodriguez Muñoz D, Rosenhek R, Sjögren J, Tornos Mas P, Vahanian A, Walther T, Wendler O, Windecker S, Zamorano JL. 2017 ESC/EACTS Guidelines for the management of valvular heart disease. *Eur Heart J* [Internet]. 2017;38:2739–2791. Available from: http://dx.doi.org/10.1093/eurheartj/ehx391

20. Malhotra A, Dhutia H, Finocchiaro G, Gati S, Beasley I, Clift P, Cowie C, Kenny A, Mayet J, Oxborough D, Patel K, Pieles G, Rakhit D, Ramsdale D, Shapiro L, Somauroo J, Stuart G, Varnava A, Walsh J, Yousef Z, Tome M, Papadakis M, Sharma S. Outcomes of Cardiac Screening in Adolescent Soccer Players. *N Engl J Med*. 2018;379:524–534.

21. Booher AM, Bach DS. Exercise hemodynamics in valvular heart disease. *Curr Cardiol Rep*. 2011;13:226–233.

22. Caselli S, Mango F, Clark J, Pandian NG, Corrado D, Autore C, Pelliccia A. Prevalence and Clinical Outcome of Athletes With Mitral Valve Prolapse. *Circulation* [Internet]. 2018;137:2080–2082. Available from: http://www.ncbi.nlm.nih.gov/pubmed/29735594

23. Lancellotti P, Tribouilloy C, Hagendorff A, Popescu BA, Edvardsen T, Pierard LA, Badano L, Zamorano JL. Recommendations for the echocardiographic assessment of native valvular regurgitation: an executive summary from the European Association of Cardiovascular Imaging. *Eur Heart J Cardiovasc Imaging*. 2013;14:611–644.

24. Bernard I, Victoria D, Raphael R, Susanna P, Bernard P, Olaf W, Michele DB, Christophe T, Arturo E, Alexander B-P, Astrid A, Hüseyin I, Cécile L, A. PB, Luc P, Michael H, Gerhard H, Frank R, Stefan W, J. BJ, Aldo M, Alec V, null null, Artan G, Julia M, Fuad S, Agnes P, Katerina L, Nikolaj I, Magdy A, Antti S, Christophe T, Srbinovska KE, Gani B, Erkin M, Andrejs E, Vaida M, Daniela C, Victoria D, Lidia T-P, Regina R, Branko B, Iveta S, Arturo E, Mesut DS, Shelley R-H. Contemporary Presentation and Management of Valvular Heart Disease. *Circulation* [Internet]. 2019;140:1156–1169. Available from: https://doi.org/10.1161/CIRCULATIONAHA.119.041080

25. Garbi M, Chambers J, Vannan MA, Lancellotti P. Valve Stress Echocardiography: A Practical Guide for Referral, Procedure, Reporting, and Clinical Implementation of Results From the HAVEC Group. *JACC Cardiovasc Imaging*. 2015;8:724–736.

26. H. GW, E. MT. Left Ventricular Response to Mitral Regurgitation. *Circulation* [Internet]. 2008;118:2298–2303. Available from: https://doi.org/10.1161/CIRCULATIONAHA.107.755942

27. Jeresaty RM. Mitral valve prolapse: definition and implications in athletes. *J Am Coll Cardiol*. 1986;7:231–236.

28. Chew PG, Bounford K, Plein S, Schlosshan D, Greenwood JP. Multimodality imaging for the quantitative assessment of mitral regurgitation. *Quant Imaging Med Surg* [Internet]. 2018;8:342–359. Available from: https://www.ncbi.nlm.nih.gov/pubmed/29774187

29. Bonow RO, Nishimura RA, Thompson PD, Udelson JE. 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. *Circulation*. 2015;132:e292–e297.

30. Stefani L, Galanti G, Innocenti G, Mercuri R, Maffulli N. Exercise training in athletes with bicuspid aortic valve does not result in increased dimensions and impaired performance of the left ventricle. *Cardiol Res Pract*. 2014;2014.

31. Langer C, Butz T, Mellwig K-P, Oepangat E, Fruend A, Faber L, Horstkotte D, Wiemer M, Van Buuren F. Elite athletes with mitral or aortic regurgitation and their cardiopulmonary capability. *Acta Cardiol*. 2013;68:475–480.

32. Baumgartner HC, Hung JC-C, Bermejo J, Chambers JB, Edvardsen T, Goldstein S, Lancellotti P, LeFevre M, Miller FJ, Otto CM. Recommendations on the echocardiographic assessment of aortic valve stenosis: a focused update from the European Association of Cardiovascular Imaging and the American Society of Echocardiography. *Eur Heart J Cardiovasc Imaging*. 2017;18:254–275.

33. Verma S, Siu SC. Aortic dilatation in patients with bicuspid aortic valve. *N Engl J Med*. 2014;370:1920–1929.

34. Michelena HI, Khanna AD, Mahoney D, Margaryan E, Topilsky Y, Suri RM, Eidem B, Edwards WD, Sundt TM 3rd, Enriquez-Sarano M. Incidence of aortic complications in patients with bicuspid aortic valves. *JAMA*. 2011;306:1104–1112.

35. Michelena HI, Desjardins VA, Avierinos JF, Russo A, Nkomo VT, Sundt TM, Pellikka PA,

Tajik AJ, Enriquez-Sarano M. Natural history of asymptomatic patients with normally functioning or minimally dysfunctional bicuspid aortic valve in the community. *Circulation*. 2008;117:2776–2784.

36. Erbel R, Aboyans V, Boileau C, Bossone E, Bartolomeo R Di, Eggebrecht H, Evangelista A, Falk V, Frank H, Gaemperli O, Grabenwoger M, Haverich A, Iung B, Manolis AJ, Meijboom F, Nienaber CA, Roffi M, Rousseau H, Sechtem U, Sirnes PA, Allmen RS von, Vrints CJM. 2014 ESC Guidelines on the diagnosis and treatment of aortic diseases: Document covering acute and chronic aortic diseases of the thoracic and abdominal aorta of the adult. The Task Force for the Diagnosis and Treatment of Aortic Diseases of the European . *Eur Heart J*. 2014;35:2873–2926.

37. Malhotra, A. Yeo,T.J., Dhutia, H, Prakash K, Keteepe-Arachi,T, D'Silva, A, Finnichiaro G, Steriotis A, Papatheodorou S, Millar L, Dassanayake S, Ensam B, Papadakis M, Tome M SS. The effect of exercise on the aortic root diameter in young elite athletes with bicuspid aortic valve disease. *Eur Heart J*. 2016;37:24–25.

38. Canciello G, Mancusi C, Izzo R, Morisco C, Strisciuglio T, Barbato E, Trimarco B, Luca N De, de Simone G, Losi MA. Determinants of aortic root dilatation over time in patients with essential hypertension: The Campania Salute Network. *Eur J Prev Cardiol* [Internet]. 2020;2047487320931630. Available from: https://doi.org/10.1177/2047487320931630

39. members AF, Erbel R, Aboyans V, Boileau C, Bossone E, Bartolomeo R Di, Eggebrecht H, Evangelista A, Falk V, Frank H, Gaemperli O, Grabenwöger M, Haverich A, Iung B, Manolis AJ, Meijboom F, Nienaber CA, Roffi M, Rousseau H, Sechtem U, Sirnes PA, Allmen RS von, Vrints CJM, (CPG) ESCC for PG, Zamorano JL, Achenbach S, Baumgartner H, Bax JJ, Bueno H, Dean V, Deaton C, Erol Ç, Fagard R, Ferrari R, Hasdai D, Hoes A, Kirchhof P, Knuuti J, Kolh P, Lancellotti P, Linhart A, Nihoyannopoulos P, Piepoli MF, Ponikowski P, Sirnes PA, Tamargo JL, Tendera M, Torbicki A, Wijns W, Windecker S, reviewers D, Nihoyannopoulos P, Tendera M, Czerny M, Deanfield J, Mario C Di, Pepi M, Taboada MJS, Sambeek MR van, Vlachopoulos C, Zamorano JL, Grimm M, Musayev O, Pasquet A, Kušljugić Z, Cikes M, Georghiou GP, Stasek J, Molgaard H, Kõvask; S, Kytö V, Jondeau G, Bakhutashvili Z, von Kodolitsch Y, Tsioufis C, Temesvári A, Rubinshtein R, Antonini-Canterin F, Lunegova O, Stradins P, Chammas E, Jonkaitiene R, Cassar A, Bjørnstad K, Widenka K, Sousa Uva M, Lighezan D, Perunicic J, Madaric J, Vilacosta I, Bäck M, Mahdhaoui A, Demirbag R, Kravchenko I. 2014 ESC Guidelines on the diagnosis and treatment of aortic diseases: Document covering acute and chronic aortic diseases of the thoracic and abdominal aorta of the adultThe Task Force for the Diagnosis and Treatment of Aortic Diseases of the European So. *Eur Heart J* [Internet]. 2014;35:2873–2926. Available from: https://doi.org/10.1093/eurheartj/ehu281

40. Gati S, Malhotra A, Sedgwick C, Papamichael N, Dhutia H, Sharma R, Child AH, Papadakis M, Sharma S. Prevalence and progression of aortic root dilatation in highly trained young athletes. *Heart* [Internet]. 2019;105:920 LP – 925. Available from: http://heart.bmj.com/content/105/12/920.abstract

41. Pelliccia A, Di Paolo FM, De Blasiis E, Quattrini FM, Pisicchio C, Guerra E, Culasso F, Maron BJ. Prevalence and clinical significance of aortic root dilation in highly trained competitive athletes. *Circulation*. 2010;122:698–706, 3 p following 706.

42. Della Corte A, Michelena HI, Citarella A, Votta E, Piatti F, Lo Presti F, Ashurov R, Cipollaro M, Forte A. Risk Stratification in Bicuspid Aortic Valve Aortopathy: Emerging Evidence and Future Perspectives. *Curr Probl Cardiol*. 2019;100428.

43. Morise AP. Exercise testing in nonatherosclerotic heart disease: hypertrophic

cardiomyopathy, valvular heart disease, and arrhythmias. *Circulation*. 2011;123:216–225.

44. Henri C, Pierard LA, Lancellotti P, Mongeon F-P, Pibarot P, Basmadjian AJ. Exercise testing and stress imaging in valvular heart disease. *Can J Cardiol*. 2014;30:1012–1026.

45. Stern H, Calavrezos L, Meierhofer C, Steinlechner E, Muller J, Hager A, Martinoff S, Ewert P, Fratz S. Physical exercise reduces aortic regurgitation: exercise magnetic resonance imaging. JACC. Cardiovasc. Imaging. 2014;7:314–315.

46. Clavel M-A, Burwash IG, Pibarot P. Cardiac Imaging for Assessing Low-Gradient Severe Aortic Stenosis. *JACC Cardiovasc Imaging*. 2017;10:185–202.

47. Gati S, Malhotra A, Sedgwick C, Papamichael N, Dhutia H, Sharma R, Child AH, Papadakis M, Sharma S. Prevalence and progression of aortic root dilatation in highly trained young athletes. *Heart*. 2019;105.

48. Babaee Bigi MA, Aslani A. Aortic root size and prevalence of aortic regurgitation in elite strength trained athletes. *Am J Cardiol*. 2007;100:528–530.

49. Aline I, D. TP. A Meta-Analysis of Aortic Root Size in Elite Athletes. *Circulation* [Internet]. 2013;127:791–798. Available from: https://doi.org/10.1161/CIRCULATIONAHA.112.000974

50. Alegret JM, Martinez-Micaelo N, La Gerche A, Franco-Bonafonte L, Rubio-Perez F, Calvo N, Montero M. Acute effect of static exercise in patients with aortic regurgitation assessed by cardiovascular magnetic resonance: role of left ventricular remodelling. *Eur Radiol*. 2017;27:1424–1430.

51. Sigvardsen PE, Larsen LH, Carstensen HG, Kuhl JT, Mogelvang R, Hassager C, Kober L, Kofoed KF. Six-minute walking test and long term prognosis in patients with asymptomatic aortic valve stenosis. *Int J Cardiol*. 2017;249:334–339.

52. Kammerlander AA, Wiesinger M, Duca F, Aschauer S, Binder C, Zotter Tufaro C, Nitsche C, Badre-Eslam R, Schönbauer R, Bartko P, Beitzke D, Loewe C, Hengstenberg C, Bonderman D, Mascherbauer J. Diagnostic and Prognostic Utility of Cardiac Magnetic Resonance Imaging in Aortic Regurgitation. *JACC Cardiovasc Imaging*. 2019;12:1474–1483.

53. G. MS, Joanna d'Arcy, Raad M, P. GJ, D. KT, M. FJ, P. BA, P. CJ, Stefan N. Aortic Regurgitation Quantification Using Cardiovascular Magnetic Resonance. *Circulation* [Internet]. 2012;126:1452–1460. Available from: https://doi.org/10.1161/CIRCULATIONAHA.111.083600

54. Antonopoulos AS, Lazaros G, Papanikolaou E, Oikonomou E, Vlachopoulos C, Tousoulis D. Aortic regurgitation in competitive athletes: The role of multimodality imaging for clinical decision-making. *Eur J Prev Cardiol*. 2019;2047487319892112.

55. Al-Hijji M, Yoon Park J, El Sabbagh A, Amin M, Maleszewski JJ, Borgeson DD. The Forgotten Valve: Isolated Severe Tricuspid Valve Stenosis. *Circulation*. 2015;132:e123-5.

56. Izgi IA, Acar E, Kilicgedik A, Guler A, Cakmak EO, Demirel M, Izci S, Yilmaz MF, Inanir M, Kirma C. A new and simple method for clarifying the severity of tricuspid regurgitation. *Echocardiography*. 2017;34:328–333.

57. Alame AJ, Karatasakis A, Karacsonyi J, Danek BA, Sorajja P, Gossl M, Garcia S, Jneid H, Kakouros N, Martinez-Parachini JR, Resendes E, Kalsaria P, Roesle M, Rangan B V,

Banerjee S, Brilakis ES. Comparison of the American College of Cardiology/American Heart Association and the European Society of Cardiology Guidelines for the Management of Patients With Valvular Heart Disease. *J Invasive Cardiol*. 2017;29:320–326.

58. Butchart EG, Gohlke-Barwolf C, Antunes MJ, Tornos P, De Caterina R, Cormier B, Prendergast B, Iung B, Bjornstad H, Leport C, Hall RJC, Vahanian A. Recommendations for the management of patients after heart valve surgery. *Eur Heart J*. 2005;26:2463–2471.

59. Parker MW, Thompson PD. Exercise in valvular heart disease: risks and benefits. *Prog Cardiovasc Dis*. 2011;53:437–446.

60. Habib G, Lancellotti P, Antunes MJ, Bongiorni MG, Casalta J-P, Del Zotti F, Dulgheru R, El Khoury G, Erba PA, Iung B, Miro JM, Mulder BJ, Plonska-Gosciniak E, Price S, Roos-Hesselink J, Snygg-Martin U, Thuny F, Tornos Mas P, Vilacosta I, Zamorano JL. 2015 ESC Guidelines for the management of infective endocarditisThe Task Force for the Management of Infective Endocarditis of the European Society of Cardiology (ESC)Endorsed by: European Association for Cardio-Thoracic Surgery (EACTS), the European Asso. *Eur Heart J* [Internet]. 2015;36:3075–3128. Available from: http://dx.doi.org/10.1093/eurheartj/ehv319

Appendix

Tables:

Tables 1a and 1b: Echocardiographic evaluation of valvular stenosis (19)

Table 1a

	Mitral valve stenosis		
	Mild	Moderate	severe
Valve opening area (cm^2)	>1.5	1.0-1.5	<1.0

Δ p_{mean} (mmHg) depending on SV and diastolic duration	<5	5-10	>10
Pulmonary artery pressure (mmHg)	<30	30-50	>50

Abbreviations: Δ p_{mean} = mean pressure difference, SV = stroke volume

Table 1b

	Aortic valve stenosis		
	Mild	Moderate	severe
V_{max} (m/s) depending on SV	2.6-2.9	3.0 – 4.0	≥4.0
Δ p_{mean} (mmHg) depending on SV	<30	30 – 40	≥40
Valve opening area (cm^2)	>1.5	1.0 – 1.5	<1.0
Valve opening area index(cm^2/m^2)	>1.0	1.0-0.6	≥0,6

Abbreviations: SV = stroke volume, LVOT = left ventricular outflow tract, V = velocity

Table 2a: Echocardiographic evaluation of mitral valve regurgitation (19)

Mitral Valve Regurgitation			
	Mild	Moderate	Severe
Qualitative			
Valve Morphology	• Thickening • Mild calcification • Prolapse	• Ring dilatation • Prolapse • Rupture of chordae or papillary muscle • Previous vegetation	• Flail leaflet • Ruptured papillary muscle • Large coaptation defect
Colour flow regurgitant jet	• Faint	• Dense • Symmetrical jet-form	• Strong • Symmetrical to early systolic peak
Other			• Large flow convergence zone

Semiquantitative			
Vena contracta width (mm)	• <3	• 3-7	• ≥7(>8 for biplane)
Upstream vein flow			• Systolic pulmonary vein flow reversal
Mitral Inflow			• E-wave dominant ≥1.5 m/s
Quantitative			
EROA (mm^2)	• <20	• 21-39	• ≥40 (primary) and • ≥20 (secondary)
Regurgitant volume (mL/beat)	• <30	• 30-59	• ≥60 (primary) and • ≥30 (secondary)
Enlargement of cardiac chambers / vessels			• LV enlargement and/or • LA enlarged

Abbreviations: EROA = effective regurgitation orifice area, LA=left atrium. LV = left ventricle,

Table 2b: Echocardiographic evaluation of aortic valve regurgitation (19)

Aortic Valve Regurgitation			
	Mild	Moderate	Severe
Qualitative			
Valve Morphology	• Bicuspid AV • Degenerative AV thickening • Mild calcification • Dilatation of aortic root or SoV	• Bicuspid AV • Degenerative AV thickening • Mild or moderate calcification • Dilatation of aortic root or SoV • Previous vegetation	• Abnormal bicuspid AV • Flail large coaptation-deficit • Thickening/calcification • Previous vegetation
Colour Flow Regurgitant Jet			• Large in central jets, variable in eccentric jets
Continuous Wave Signal of Jet	• Faint	• Dense	• Strong
Other			• Holodiastolic • Flow reversal in descending aorta • Peripheral signs of AR

Semiquantitative			
Vena contracta width (mm^2)	<3	3-6	>6
Other	PHT <400ms		PHT <200 ms
Quantitative			
EROA (mm^2)	<10	10-29	≥30
Regurgitant volume (mL/beat)	<30	>30-59	≥60
Enlargement of cardiac chambers / vessels			• Marked dilatation of the LV and/or • Evidence of LV dysfunction • Enlarged atrial size

Abbreviations: AV = aortic valve, EROA = effective regurgitation orifice area. LV = left ventricle, PHT = pressure half time, SoV = Sinus of Valsalva

Table 2c: Echocardiographic evaluation of tricuspid valve regurgitation (19)

Tricuspid Valve Regurgitation		
	Moderate	Severe
Qualitative		
Valve Morphology	• Ring dilatation, • Ebstein anomaly • Previous vegetation • Prolapse • Ruptured chordae, RV-PM electrode	• Abnormal flail • Large coaptation defect • Ring dilatation • Ebstein anomaly • Previous vegetation • Ruptured chordae • RV-PM electrode
Colour Flow Regurgitant Jet		• Large jet reaching IVC or eccentric wall impinging jet
Continuous Wave Signal of Jet		• Dense/triangular • Low velocity time integral (peak <2m/s in massive TR) • Early systolic peak
Semiquantitative		
Vena contracta width (mm^2)		≥7

Upstream vein flow		• Systolic hepatic vein flow reversal
Mitral Inflow		• E-wave dominant ≥1 m/s
Other		• PISA radius >9 mm^2
Quantitative		
EROA (mm^2)		• ≥40
Regurgitant volume (mL/beat)		• ≥45
Enlargement of cardiac chambers / vessels		• RV and/or RA enlargement • Dilated IVC

Abbreviations: EROA = effective regurgitation orifice area, IVC = Inferior Vena Cava, PM = pacemaker, PISA = proximal isovelocity surface area, RA = right atrium, RV = right ventricle

Figure 1

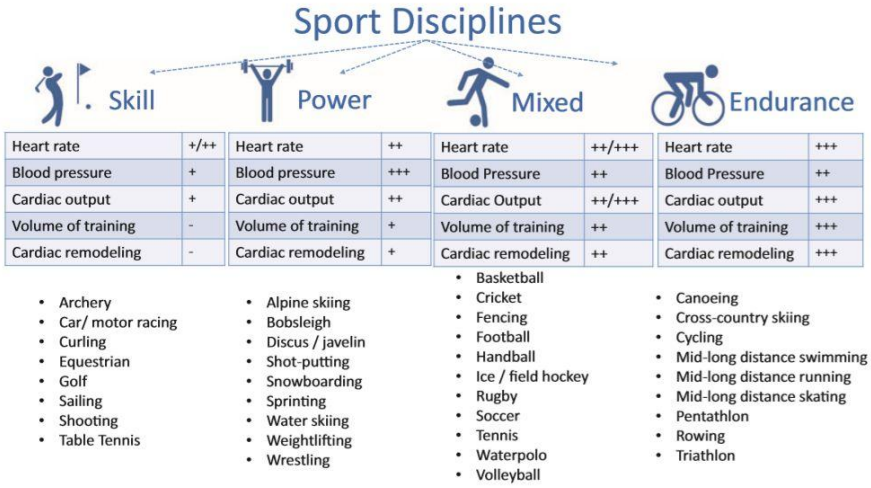


Figure 2

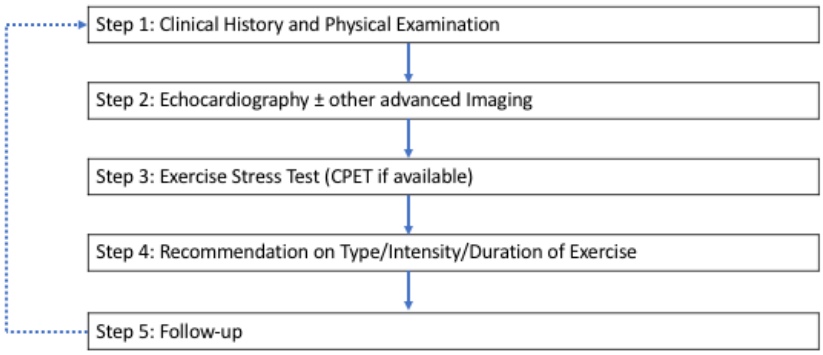
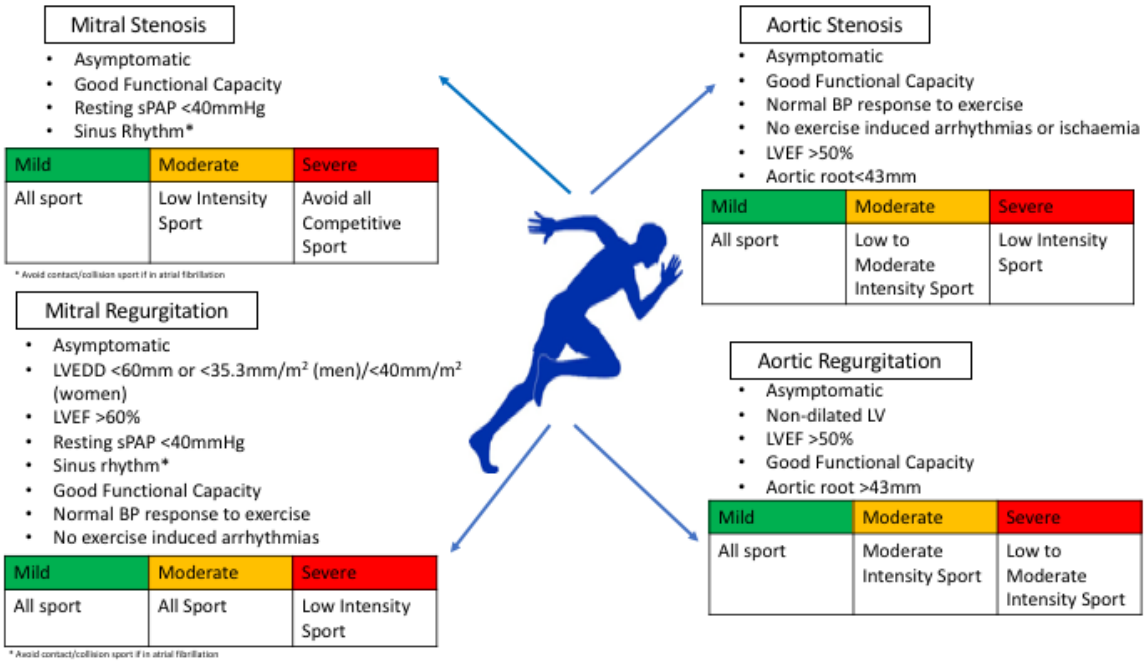

Step 1: Clinical History and Physical Examination
Step 2: Echocardiography ± other advanced Imaging
Step 3: Exercise Stress Test (CPET if available)
Step 4: Recommendation on Type/Intensity/Duration of Exercise
Step 5: Follow-up

Figure 3


Mitral Stenosis
Asymptomatic
Good Functional Capacity
Resting sPAP <40mmHg
Sinus Rhythm*
Mild | Moderate | Severe
All sport | Low Intensity Sport | Avoid all Competitive Sport
* Avoid contact/collision sport if in atrial fibrillation
Mitral Regurgitation
Asymptomatic
LVEDD <60mm or <35.3mm/m² (men)/<40mm/m² (women)
LVEF >60%
Resting sPAP <40mmHg
Sinus rhythm*
Good Functional Capacity
Normal BP response to exercise
No exercise induced arrhythmias
Mild | Moderate | Severe
All sport | All Sport | Low Intensity Sport
* Avoid contact/collision sport if in atrial fibrillation
Aortic Stenosis
Asymptomatic
Good Functional Capacity
Normal BP response to exercise
No exercise induced arrhythmias or ischaemia
LVEF >50%
Aortic root<43mm
Mild | Moderate | Severe
All sport | Low to Moderate Intensity Sport | Low Intensity Sport
Aortic Regurgitation
Asymptomatic
Non-dilated LV
LVEF >50%
Good Functional Capacity
Aortic root >43mm
Mild | Moderate | Severe
All sport | Moderate Intensity Sport | Low to Moderate Intensity Sport