Review Article

Heart and Athlete

Mohammad Alasti, MD[1*], Bita Omidvar, MD[2], Mohammad Hossein Jadbabaei, MD[2]

[1]*Imam Khomeini Hospital, Jondishapour University of Medical Sciences, Ahwaz, Iran.*
[2]*Golestan Hospital, Jondishapour University of Medical Sciences, Ahwaz, Iran.*

Abstract

Regular participation in intensive physical exercise is associated with electro-morphological changes in the heart. This benign process is called athlete's heart. Athlete's heart resembles few pathologic conditions in some aspects. So differentiation of these conditions is very important which otherwise may lead to a catastrophic event such as sudden death. The most common causes of sudden death in young athletes are cardiomyopathies, congenital coronary anomalies, and ion channelopathies. The appropriate screening strategy to prevent sudden cardiac death in athletes remains a challenging issue. The purpose of this review is to describe the characteristics of athlete's heart and demonstrate how to differentiate it from pathologic conditions that can cause sudden death.

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Introduction

The athlete's heart syndrome refers to the electro-morphological remodeling which occurs to varying extents dependent upon the sporting discipline.[1] It has been a subject of many studies over decades as the ability to differentiate a pathologic process from a physiologic process is of critical importance to the clinician and patient.

Regular participation in intensive physical exercise is associated with central and peripheral cardiovascular adaptations that facilitate the generation of a large and sustained cardiac output and enhance the extraction of oxygen from exercising muscle for aerobic glycolysis.[2]

Sports are classified according to their types, dynamic (isotonic) or static (isometric). Briefly, dynamic exercise involves changes in muscle length and joint movement with rhythmic contractions which develop a relatively small intramuscular force. Static exercise induces development of a large intramuscular force with little or no change in muscle length or joint movement. These two types of exercises should be thought of as the two opposite poles of a continuum, with most physical activities involving both static and dynamic components.[3]

During progressive dynamic exercise, cardiac output components (heart rate and stroke volume) are increased. The increase in stroke volume is achieved by both increasing of end-diastolic volume (Frank-Starling mechanism) and decreasing of end-systolic volume (increased contractile state). Total peripheral resistance deeply decreases. Thus, systolic and mean blood pressure increase moderately and diastolic blood pressure is maintained or decreases slightly. By contrast, static exercise induces a small increase in VO_2, heart rate, and cardiac output without changing of stroke volume. Total peripheral resistance does not decrease. The rising of systolic, mean, and diastolic blood pressure is linked to the muscle mass involved, the percent of maximal

[*]**Corresponding Author:** ***Mohammad Alasti,*** *Department of Cardiology, Jondishpour University of Medical Sciences, Imam Khomeini Hospital, Azadegan Avenue, Ahwaz, Iran. Tel: +98 611 4457205. Fax: +98 611 4457205. E-mail: alastip@gmail.com.*

voluntary contraction, and the contraction duration.[3]

Preserved systolic and diastolic function and regression of structural abnormalities with deconditioning are consistent with physiologic adaptation to training. Several studies evaluating transmitral flow by Doppler echocardiography have shown higher early peak diastolic filling velocity and higher ratios of early (E) to late (A) filling velocities (E/A ratio usually 1.5 to 1.9) in trained athletes when compared with control subjects, suggesting supranormal diastolic function.[2]

Increased cardiac mass is a common finding in trained athletes who have an increased left ventricle (LV) size. LV mass usually falls within the accepted normal limits for age- and sex-matched control subjects.[2]

Left atrial (LA) enlargement is another common morphologic finding. LA enlargement is usually mild to moderate. Increased diameter of the left atrium, found more commonly in athletes who have demonstrable changes in LV morphology and in those individuals active in ultra-endurance sports like cycling and marathon running, is believed to be secondary to increased volume load.[2, 4]

Mild-to-moderate structural changes of the right ventricle (RV) with preserved contractile function are the other finding in the athlete's heart syndrome. These include increased end-diastolic volume, wall thickness, and mass.[2, 5]

The differential diagnosis for individuals with clearly abnormal RV includes dilated cardiomyopathy, hypertrophic cardiomyopathy (HCM), arrhythmogenic right ventricular cardiomyopathy (ARVC), and myocarditis.[2]

The heart of a trained individual usually falls within accepted normal limits, including the following measurements: LV end diastolic dimension less than 6.0 cm, LV wall thickness less than 1.3 cm, septal-posterior wall thickness ratio less than 1.3, and LV mass less than 294 g in men and 198 g in women.[6] However, there can be significant overlap between the upper limits of normal in the athlete's heart with other forms of structural cardiac disease.[2, 7- 9]

Athletes with concomitant adaptive cardiac hypertrophy have less left-sided and more right-sided regurgitation than physically active healthy subjects with smaller hearts. The increases in the frequency and severity of tricuspid regurgitation (TR) and pulmonary regurgitation (PR) should be kept in mind when examining athletes with physiologic hypertrophy.[10]

Some factors can affect the magnitude of morphologic changes following exercise. Endurance disciplines, such as cycling, cross-country skiing, and rowing/canoeing have the greatest effect on LV cavity dimensions. Other disciplines such as soccer, basketball, handball, and other team ball sports (which include aerobic and anaerobic exercise training) show a moderate impact on LV cavity dimension. Finally, technical disciplines such as equestrian sports or yachting have only a minimal effect on cardiac dimensions.[11, 12]

For years, it has been believed that predominantly isotonic (aerobic) training leads to more significant changes in LV cavity dilatation, wall thickness, and mass. This is in contrast to athletic activities in which the training is predominantly isometric (strength) in nature, like weight lifting and wrestling, in which there may be only increased wall thickness.[2] Interestingly, a recent study showed that the most extreme increases in LV wall thickness had been observed in those athletes training in rowing and cycling. Of note, strength training was associated with only a mild increase in wall thicknesses (although often disproportionate to cavity size), whereas absolute values uncorrected for body surface area usually remained well within the accepted normal range (≤12 mm).[13]

Body size, sex, and race are the other factors that can affect the response of the heart to exercise. In general, larger male athletes have greater absolute increases in LV wall thickness, cavity dimension, mass, and left atrial dimension.[2, 14] Although some of the differences between males and females seem to be related to different body size, other mechanisms are the lower increasing of absolute blood pressure during peak exercise in women and their lower level of natural androgenic hormones.[11]

It seems that a larger proportion of African-American athletes compared with Caucasian athletes have an LV wall thickness exceeding upper normal limits.[11] The exact stimulus to the disproportionate LV wall thickening in African-American athletes remains unknown.[15]

However, there is a significant degree of variability among athletes that cannot be explained by these factors alone. Other genetic and environmental factors are believed to have a significant role in the cardiac structural changes in athletes.[16, 17]

Differentiation from cardiovascular diseases

The differential diagnosis of physiologic LV enlargement in trained athletes is based on the presence of normal LV systolic/diastolic function and absence of segmental wall motion abnormalities. Indeed, LV cavity dilation in normal athletes is associated with superior physical performance as assessed by cardiopulmonary testing.[18] An athlete with left ventricular hypertrophy (LVH) between 12 and 16 mm represents a grey zone between the extremes of physiological adaptation and mild expression of HCM. The identification of LVH in a female athlete, any adolescent athlete aged <16 years old, and any athlete participating in low intensity endurance sports is highly indicative of HCM.[13, 19] Physiological LVH is homogeneous and symmetrical. Athletes rarely exhibit differences of >2 mm between adjacent LV myocardial segments, and the ratio of the interventricular wall thickness to the LV posterior wall thickness in end-diastole is <1.5:1. In contrast, almost any pattern of hypertrophy is possible

in HCM. Most individuals (60%) with HCM demonstrate asymmetrical septal hypertrophy and 10% reveal hypertrophy confined to the LV apex. The LV cavity size is the most important discriminator between physiological LVH and HCM. Almost all athletes with physiological LVH have concomitant enlargement of the LV cavity. Typical values of LV cavity size in athletes with LVH range between 55 and 65 mm. Most individuals with HCM have a small LV cavity (<45 mm). Approximately, 25% of individuals with HCM exhibit basal, dynamic LV outflow tract obstruction and up to 70% develop obstruction with exercise. Early rapid LV filling is impaired as evidenced by the demonstration of a reversed E/A ratio, prolonged E-deceleration time (>240 ms), isovolumic relaxation times (>90 ms), and reversed systolic to diastolic wave (S/D) ratio during pulmonary vein Doppler. Measurement of myocardial velocity gradients from digitized M-mode colour Doppler reveals that individuals with HCM exhibit impaired myocardial filling during the rapid filling phase of diastole and display reduced LV posterior wall myocardial velocity gradients compared with athletes.[19, 20]

Assessment of longitudinal cardiac function with pulsed tissue Doppler at the level of the mitral valve annulus has demonstrated that individuals with morphologically mild HCM exhibit lower early diastolic velocities (Ea). An Ea of <9 cm/s in lateral mitral annulus favours pathological LVH with a sensitivity approaching 90%. The E/Ea ratio may also be useful in differentiating physiological LVH from HCM. An E/Ea >12 is indicative of high left atrial filling pressures, a hallmark of HCM; however, most trained athletes exhibit a E/Ea <8.[19, 21]

In patients with HCM, strain and strain rate are abnormal even in the absence of myocardial fibrosis on cardiac MRI.[22, 23] Also, athletes have a synchronous activation pattern in the left and right ventricle. In contrast, the HCM patients have significant inter and intraventricular activation delays.[24]

Pulsed tissue Doppler studies have shown that many HCM patients exhibit impaired longitudinal systolic function. So, finding of a mitral valve annular peak systolic velocity of <9 cm/s in an athlete with LVH should raise the suspicion of underlying pathology.[19] Sherrid et al. measured the diastolic flow in septal perforator coronary arteries of patients with HCM by transthoracic echocardiography. The peak diastolic flow in these individuals was significantly increased compared to both healthy controls and individuals with hypertrophy caused by arterial hypertension.[25]

MRI has a potential value for diagnosing HCM by virtue of its superiority over echocardiography in identifying segmental hypertrophy in the anterolateral LV free wall or at the apex.[19, 26]

In active athletes presenting with LV hypertrophy, abnormal amino terminal pro-brain natriuretic peptide (NT-pro BNP) levels indicate HCM, whereas normal values are inconclusive.[2, 27]

Commercial laboratory genetic testing is now available in HCM but it is costly and the diagnostic yield is only 60-70%. Therefore, a negative gene test does not exclude HCM.[19, 21]

The measurement of peak oxygen consumption during an exercise test is another method of differentiating physiological LVH from HCM. A peak oxygen consumption of >50 mL/kg/min in an athlete with mild LVH favours physiological adaptation.[19] Stopping the sport discipline has no effect on LVH in patients with HCM; but even in highly trained Olympic athletes, a substantial reduction (by 2-5 mm) will occur after a 3-month deconditioning period.[27]

Both athletes and ARVC patients demonstrate RV enlargement compared to controls; nonetheless, RV cavity size is not significantly larger in ARVC patients than in athletes, whereas right ventricle outflow tract (RVOT) diameter is significantly larger in ARVC subjects compared to athletes. Furthermore, all ARVC patients show localized RV wall motion abnormality, an abnormality not detected in athletes and controls.[28]

The differentiation from dilated cardiomyopathy (DCM) is generally easily made because of the absence of LV systolic dysfunction in athlete's heart, absence of familial history of DCM, and absence of arrhythmias.[29]

In trained athletes, LV cavity enlargement is associated with consistent enlargement of the RV; indeed, the physiologically dilated LV cavity maintains the ellipsoid shape, with the mitral valve normally positioned and without mitral regurgitation.[30, 31]

Electrocardiogram in athletes

Abnormal Electrocardiograms (ECGs) are common in athletes. Athletes of African descent and athletes participating in endurance sports (cycling, rowing, and triathlons) or sports with high peak levels of activity (football, basketball, track, and soccer) tend to have a higher incidence of abnormal ECG. Conversely, female athletes and athletes participating in more technically oriented sports (judo and equestrian sports) have a relatively lower incidence of abnormal ECG findings.[32, 33]

The spectrum of abnormalities includes sinus bradycardia, first and second-degree heart block, early repolarization, and LV hypertrophy.

Certain ECG abnormalities are associated with heart disease and an increased risk of sudden cardiac death (SCD). These abnormalities include the pseudo-infarct pattern seen in HCM (septal Q-waves) and Wolff-Parkinson-White (WPW) syndrome (inferior Q-waves).[34]

The ECGs of trained athletes often exhibit pure voltage criteria (based only on QRS amplitude measurements) for LVH that reflect physiological LV remodelling with increased LV wall thickness and chamber size. Isolated QRS voltage criterion for LVH is an unusual pattern in patients with HCM, in whom pathologic hypertrophy is characteristically

associated with additional ECG abnormalities such as left atrial enlargement, left axis deviation, delayed intrinsicoid deflection, T-wave inversion, and pathologic Q waves.[35]

Thus, systematic echocardiographic evaluation of athletes fulfilling isolated QRS voltage criteria at preparticipation screening is not justified, unless such subjects have other ECG changes, relevant symptoms, abnormal physical examination, or positive family history for cardiovascular diseases or premature SCD.[30, 35, 36]

The presence of T-wave inversion of 2 mm or more in at least 2 adjacent leads in an athlete is a non-specific but warning ECG sign of a potential cardiovascular disease. The significance of flat or minimally inverted (<2 mm) T waves (mostly inferior and/or lateral leads) is unclear. These changes usually revert to normal with exercise and are considered a benign ECG phenomenon resulting from increased vagal tone. However, such minor T-wave abnormalities are uncommonly encountered in the athlete heart, but are common in cardiomyopathy. This indicates that they may have a pathologic basis and should be cautiously investigated and followed up over time.[37, 38]

In asymptomatic white adolescent athletes aged less than 14 years, the presence of T-wave inversions in leads V_1-V_4 should not justify further investigations in the absence of symptoms or a family history of premature heart disease or SCD. In contrast, T-wave inversions beyond V_2 (even beyond V_1) are uncommon in post-pubertal athletes and their rarity probably warrants further investigation.[28, 35, 39]

The identification of deep T-wave inversions in the anterior and/or lateral leads is a recognized feature of apical HCM. Detailed assessment of the left ventricular apex at echocardiography and the use of a contrast agent to define the endocardial borders should, therefore, be considered.[37]

The prevalence of incomplete right bundle branch block (RBBB with QRS duration <120 ms) has been estimated to range from 35 percent to 50 percent in athletes compared with 10 percent in young healthy controls. The ECG pattern is more often noted in athletes engaged in endurance sports, with a striking male preponderance. It has been suggested that it is caused by the enlarged RV cavity size or increased cardiac muscle mass and the resultant conduction delay. Incomplete RBBB does not require further tests in the presence of a negative family/personal history and physical examination. The RBBB morphology has been shown to be reversible with deconditioning. An underlying ARVD should be suspected when the pattern of incomplete RBBB is associated with disproportionate extent of T-wave inversion (beyond V_2) or in the presence of premature ventricular beats with a LBBB morphology.[32, 37]

The early repolarization ECG pattern is another common finding among highly trained athletes, in whom it is observed in 50-80 percent of resting ECGs. The most notable ECG feature is the elevation of the QRS-ST junction (J point) of at least 0.1 mV from baseline, often associated with notching or slurring of the terminal QRS complex. Early repolarisation may vary on location, morphology, and degree. It is most often localised in precordial leads, with the greatest ST-segment elevation in mid-to-lateral leads (V_3-V_4). Maximal ST-segment displacement may also occur more laterally (leads V_5, V_6, L_1 and aVL), inferiorly (L_2, L_3 and aVF), or anteriorly (leads V_2-V_3). Slowing of the heart rate exaggerates ST-segment elevation, whereas sinus tachycardia occurring during exercise or after isoproterenol administration reduces and often eliminates early repolarisation changes. In athletes presenting with syncope or cardiac arrest which remains unexplained after a detailed clinical work-up, an ECG pattern of early repolarisation in the inferior and/or lateral leads, with a prominent terminal QRS slurring, should raise the suspicion of an underlying idiopathic ventricular fibrillation. The characteristics of benign early repolarization that differentiate it from potentially pathological ST-segment elevation include diffuse ST-segment elevation, upward concavity of the initial portion of the ST segment, notching or slurring of the terminal QRS complex, and concordant large amplitude T waves. Also, athletes exhibit an up-sloping ST segment with a mean ST_J/ST_{80} ratio ≤ 1, whereas patients with the Brugada syndrome show a down-sloping ST segment with a ST_J/ST_{80} ratio >1.[32, 37]

As opposed to the common occurrence of ST-segment elevation in athletes, the presence of ST-segment depression is rare and should warn the clinician to pursue pathologic causes.[32]

Arrhythmia in athletes

Sinus bradycardia is a common finding on the ECG of a highly trained athlete. Variable degrees of atrioventricular (AV) block are also not uncommon. The most common are first-degree (10%) and second degree Mobitz type I (8%). Although second-degree Mobitz type II and even third-degree heart block have been reported, they are exceedingly rare. Resolution of (asymptomatic) first-degree or second-degree atrioventricular (AV) block with hyperventilation or exercise confirms its functional origin and excludes any pathologic significance.[37]

Escape junctional beats or rhythm may be recorded in athletes with more severe bradycardia and result in functional AV dissociation. Sinus arrhythmia that disappears during exercise has also been reported.[37]

Most ventricular tachyarrhythmias (including non-sustained ventricular tachycardia) occurring in highly trained athletes are not associated with adverse clinical consequences and are usually abolished or substantially reduced after relatively brief periods of deconditioning.[13, 26, 40]

But complex and frequent ventricular tachyarrhythmias should raise the possibility of disease states such as myo-carditis. Periods of forced deconditioning may not be useful

in resolving the differential diagnosis of myocarditis versus athlete's heart since detraining is associated with reduction (and even abolition) of ventricular tachyarrhythmias in athletes with or without underlying pathologic substrates.[13, 31]

The risk of lone atrial fibrillation (AF) is higher in athletes compared with controls.[41, 42] According to a recent study, frequency of vigorous exercise was associated with an increased risk of developing AF in young men and joggers. This risk decreased as the population aged and was offset by known beneficial effects of vigorous exercise on other AF risk factors.[43]

Another study showed the elderly athlete may not be as healthy as believed: among former athletes, sinus node dysfunction occurred significantly more often compared with age-matched controls.[24]

Syncope in athletes

Syncope in young individuals is usually benign; however, syncope can be a warning of impending SCD. In the athlete with syncope, an echocardiogram is necessary in all but the most classic neurocardiogenic syndromes.[34, 44]

Syncope that occur during exertion are more likely to be life-threatening than those that occur at rest. What is called exercise-induced neurocardiogenic syncope is in fact syncope that occurs after exercise or during pauses in exertion. Post-exertional syncope is not likely to be life-threatening, but is likely to be caused by vasodilatation and corresponding hypotension. Syncope without prodromal symptoms is more troubling than a gradual onset of syncope. Syncope that occurs only with upright posture is less likely to be arrhythmic than that occurring during sitting or lying down. Syncope with clear and reproducible triggers of stress, excitement, or fear is more likely to be neurocardiogenic than arrhythmic. Injury secondary to syncope is more often seen in arrhythmic disorders and rarely seen in neurocardiogenic syncope. Frequent episodes of presyncope and light-headedness are less likely to be arrhythmic in origin than occasional episodes of syncope. In individuals with this family history of inherited cardiac condition, syncope is more concerning.[34]

Cardiovascular causes of sudden cardiac death

The exact incidence of SCD in young athletes is unknown. In Italian competitive athletes (age 14-35 years), the incidence was found to be 3.6/100000 and in US athletes, the incidence was lower (0.5/100000).[45]

The combined prevalence of cardiovascular diseases that predispose to SCD in the general athletic population is estimated at 0.3%.[30]

SCD in athletes is more common in men (men/women ratio ranging from 5/1 to 9/1).[21] The risk of SCD in athletes significantly increases with age.[30] Sudden collapse usually occurs with physical exertion, predominantly in the late afternoon and early evening hours, corresponding to the peak periods of competition and training, and particularly in organized team sports.[21] The most common cardiovascular cause of SCD in young athletes in US is hypertrophic cardiomyopathy, accounting for about 35% of such events.[21]

Second in frequency to HCM is a variety of congenital malformations of the coronary arteries, the most common of which is anomalous origin of the left main coronary artery from the right sinus of Valsalva. Young individuals with anomalous left main coronary artery may die suddenly as the first manifestation of their disease, although a minority experience angina, syncope, or even acute myocardial infarction. The vast majority of these events are related to exertion. Indeed, occurrence of one or more episodes of exertional syncope in a young athlete necessitates exclusion of this coronary anomaly. In athletes it may be possible to identify anomalous left main (or right) coronary artery using two-dimensional or transesophageal echocardiography, which can then lead to definitive confirmation with coronary arteriography or CT angiogram. Other unusual variants of coronary arterial anatomy, including hypoplasia of some portion of the coronary circulation, left anterior descending, or right coronary artery emanating from the pulmonary trunk, have been reported.[21] Arrhythmogenic right ventricular dysplasia (ARVD) is the most common underlying organic heart disease in Italy but its frequency is in the range of less than 5% in US.[21]

Understanding of commotio cordis has increased in recent years, and it is presently an important cause of SCD in athletes. Commotio cordis is frequently caused by projectiles which are implements of the game, and strike the chest at a broad range of velocities. Chest barriers with proven efficacy in preventing commotio cordis are not yet available and commercially available chest protectors have proven ineffective in preventing ventricular fibrillation.[21]

Sudden unexpected death in a young athlete can be attributed to illicit substance abuse (cocaine, anabolic steroids, or dietary and nutritional supplements).[21]

Less common causes of sudden death in young athletes (accounting for 3%-8%) include myocarditis, dilated cardiomyopathy, aortic dissection and rupture (usually caused by Marfan syndrome), sarcoidosis, valvular heart disease (mitral valve prolapse or aortic valve stenosis), and atherosclerotic coronary artery disease. Also, a small number of athletes die suddenly without evidence of structural cardiovascular disease, even after careful gross and histologic examination of the heart. It is likely that some are caused by previously unidentified WPW syndrome, ion channelopathies, and catecholaminergic polymorphic ventricular tachycardia (CPVT), or possibly, undetected

segmental ARVC or subtle morphologic forms of HCM.[21] In athletes over 30 years old, coronary artery disease is the underlying disease in up to 80% of patients.[34]

The true clinical significance of myocardial bridging in the athletes is unclear. It is probable that myocardial bridging is most often benign, given the disparity between its prevalence and the incidence of related cardiac events.[46, 47]

Preparticipation screening

Preparticipation cardiovascular screening is the systematic practice of evaluating athletes before participation in sports for the purpose of identifying abnormalities that may predispose to SCD.

History taking and physical examination are the cornerstone of screening protocols. Personal history of exertional chest discomfort, unexplained syncope or near-syncope, unexplained dyspnea or fatigue associated with exercise, prior recognition of a heart murmur, elevated systemic blood pressure, family history of cardiac death before age 50 years, disability from heart disease in a close relative < 50 years of age, specific knowledge of certain cardiac conditions in family members (hypertrophic or dilated cardiomyopathy, ion channelopathies and Marfan syndrome) should be inquired.[48]

Physical examination should include dynamic auscultation for heart murmur, palpation of femoral pulses to exclude aortic coarctation, evaluation for the physical stigmata of the Marfan syndrome, and measurement brachial artery blood pressure in sitting position.[49, 50] In athletes, the LV may be prominent to feel and displaced laterally. Third and fourth sounds are permissible, as is a soft mid-systolic flow murmur.[48]

In Italy, screening usually starts at the beginning of competitive athletic activity (age 12-14 years) and is repeated on a regular basis. First-line examination includes history taking, physical examination, and 12-lead ECG. The addition of 12-lead ECG has the potential to enhance the sensitivity of the screening process for the detection of cardiovascular diseases. In fact, ECG is abnormal in up to 95% of patients with HCM, which is the leading cause of sudden death in athletes. Likewise, ECG abnormalities have also been documented in the majority of athletes who died from ARVC, proven at autopsy.[35]

The screening method recommended by the American Heart Association states that 12-lead ECG is not cost-effective for screening large populations of young athletes because of its low specificity.[30, 35]

Additional tests are requested only for subjects who have positive findings at the initial evaluation. Despite different preparticipation screening strategies, athlete sudden death rates in demographically similar regions of the United States and Italy have not differed significantly in recent years. These data do not support a lower mortality rate associated with preparticipation screening programs involving routine electrocardiography and examinations by specially trained personnel.[51]

Cardiovascular disease and sport

When a cardiovascular abnormality is identified in a competitive athlete several considerations arise: (1) level of risk for SCD if participation in organized sports continues; (2) likelihood that risk would be reduced if systematic training and competition were terminated; and (3) criteria to formulate appropriate eligibility or disqualification decisions.[21]

Athletes with radiofrequency cure of supraventricular tachycardias can resume competitive athletics after 4 weeks. Athletes with first-degree or Mobitz I heart block, which does not worsen with exercise, do not need restriction of competitive athletics. However, athletes with Mobitz II or complete heart block generally require pacing. Those athletes with permanent pacemakers should not participate in competitive athletics with a danger of bodily collision.[21]

Only low intensity competitive athletics is permitted in athletes with structural heart disease and sustained ventricular arrhythmias, without regard to the method of treatment.[21]

In patients with ARVD, moderate and high level competitive athletics are contraindicated. This recommendation is independent of age, gender, and phenotypic appearance and does not differ for those athletes without symptoms or for those treated with drugs, surgery, catheter ablation, or an implantable defibrillator.[6, 21, 28]

In individuals with HCM, competitive athletics should be prohibited. Young athletes with the unequivocal diagnosis of HCM are discouraged from competitive athletic participation, with the exception of low-intensity sports like golf, billiards, and bowling.[6, 21] Annual serial echocardiography is recommended throughout adolescence in athletes with a family history of HCM.[36]

Because of the potential for SCD, competitive athletics are not recommended in the Brugada syndrome.[34] Athletes with a clinical diagnosis of myocarditis should be temporarily excluded from competitive and amateur-leisure time sport activity. This recommendation does not differ for athletes with only mild symptoms or under treatment with drugs. After resolution of the clinical presentation (at least 6 months after the onset of the disease), clinical reassessment is indicated before the athlete re-enters a competitive sport lifestyle.[52]

Athletes with a clinical diagnosis of DCM should be excluded from most competitive sports, with the possible exception of those of low intensity. This recommendation does not differ for those athletes without symptoms, those with prior treatment with drugs or major interventions with surgery, or those with an implantable cardioverter defibrillator (ICD).[52] All patients with CPVT are restricted

from participation in competitive athletics, with the possible exception of low intensity sports.[6]

Restriction from competitive sports in patients with long QT syndrome is recommended for those with syncope or resuscitated SCD, and for those with QTc above 470 in males and 480 in females.[21] Asymptomatic patients with prolonged QTc should be restricted to low-intensity competitive sports.[6]

Return to sport after a commotio cordis event should be on a case-by-case basis. There is concern that patients undergoing a commotio cordis event are more vulnerable to recurrent events.[6]

Athletes with an anomalous coronary anomaly associated with exercise-related sudden cardiac death should be excluded from athletic activities until the anomaly is surgically corrected.[46]

In athletes with coronary artery disease and ventricular arrhythmias, only low intensity competitive athletics are permitted. Athletes with documented vasospasm should also undergo atherosclerosis risk factor management because underlying atherosclerosis contributes to abnormal coronary vasomotion. Recommendations for athletic activity should be based on evidence that exercise produces vasospasm in the athlete, the ability to control symptoms with medications, and the presence of underlying atherosclerosis. Consensus guidelines favour marked restriction of physical activity in them.[46]

In patients who have hypertension and are about to engage in intense (although amateur) exercise training, a medically supervised peak or symptom limited exercise test with electrocardiography and blood pressure monitoring is warranted. Athletes with hypertension should be treated according to the general guidelines for the management of hypertension. Diuretics and beta-blockers are not recommended for first-line treatment in patients engaged in competitive or high-intensity endurance exercise.[53]

An ICD disqualifies an athlete for competitive sports, except those with a low cardiovascular demand. In addition to underlying disease, there are other reasons to stop participation in intensive sports: (1) physical activity is a likely trigger for ventricular arrhythmias; (2) transient impaired consciousness can be dangerous during certain sports; and (3) the efficacy of the ICD to interrupt malignant ventricular arrhythmias during intense exercise is probably suboptimal.[54]

Extreme ipsilateral arm movements should be avoided to prevent lead fracture, such as during volleyball, basketball, racket sports, swimming, or gymnastics. The risk of subclavian crush should be the reason for implanting single-lead devices and with a lead preferably with only one shocking coil. Sudden onset discriminator in these patients should be used carefully because ventricular arrhythmias may develop during sinus tachycardia, reducing the specificity of this parameter.[54]

Leisure-time sports resumption is allowed from 6 weeks after implant, preferably after a control stress test. When appropriate or inappropriate ICD interventions occur, a 6-week period refraining from sports should be reconsidered to evaluate the effect of changes in medical therapy or ICD programming.[54]

Conclusion

Differentiating a pathologic condition from the physiologic process in athletes is very important because the inability to diagnose the underlying heart disease can lead to sudden death during exercise, which is a catastrophic event. For all the advances, finding the appropriate screening strategy to prevent SCD in athletes still remains a challenging issue.

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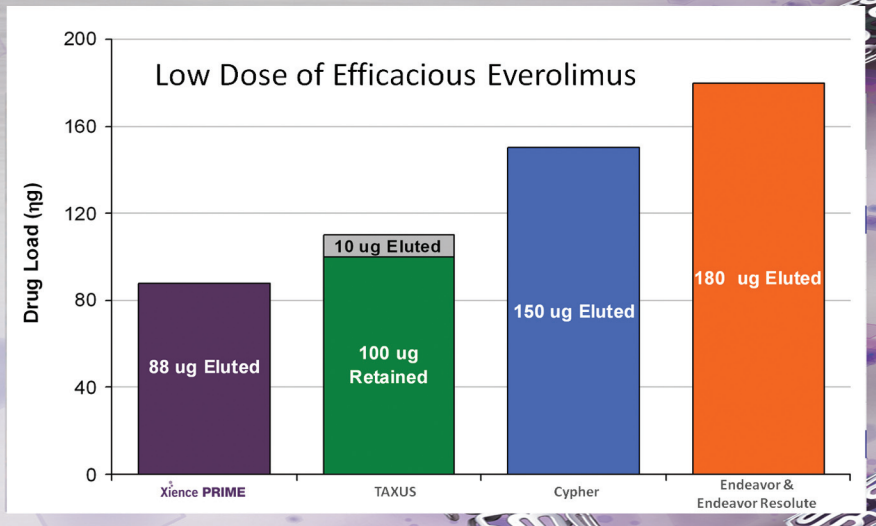

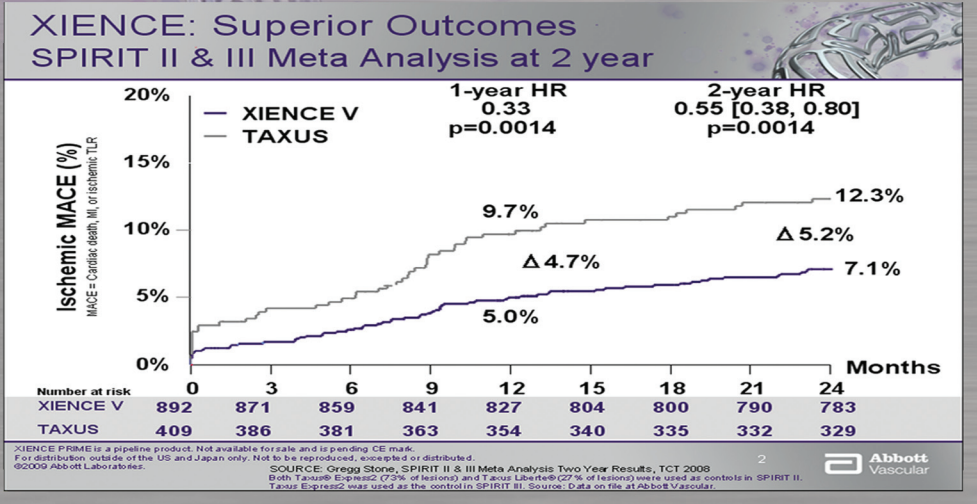

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SOURCE: Gregg Stone, SPIRIT II & III Meta Analysis Two Year Results, TCT 2008
Both Taxus® Express2 (73% of lesions) and Taxus Liberte® (27% of lesions) were used as controls in SPIRIT II.
Taxus Express2 was used as the control in SPIRIT III. Source: Data on file at Abbott Vascular.

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