



Arrhythmias in Athletes: What Is New?

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Introduction

Sports seem to be as old as mankind. Athletic performances, though, are of proven long-term benefits, test the capacity of human mind and body. Arrhythmias that occur during or shortly after exercise are labeled as exercise-induced arrhythmias. As early as 1927, Bourne commented on the development of frequent ventricular premature beats (VPBs) during exercise in patients with suspected coronary artery disease (CAD)¹. Later, in 1932, Wilson et al reported the first case of ventricular tachycardia (VT) initiated during exercise². The American College of Cardiology (ACC) has sponsored 3 prior documents which addressed eligibility and disqualification criteria for competitive athletes with cardiovascular diseases: Bethesda Conferences 16 (1985), 26 (1994), and 36 (2005), used over a 30-year period. The arrhythmias in sportsmen can vary from benign and mild ones related to physiological adaptation at one end and unexpected, arrhythmia leading to fatal events during adolescence and young adulthood at the other extreme end. Specially, the right ventricle, exposed to high strain during intense endurance activities, over a period, may lead to a pro-arrhythmic state, similar to left ventricular

cardiomyopathy. Athletes with documented rhythm disturbances should have individualized risk stratification and, usually, consultation with a cardiologist or electro-physiologist. Ridiculously, patients with heart rhythm abnormalities many a time are told about contraindications to participation in sports, which may not be required. In this review, we shall discuss the relevance of various electrophysiological abnormalities to athletic participation.

Normal Physiological Adaptations with Physical Activity

There are cardiovascular adaptations to provide a large and sustained cardiac output for enhancing the extraction of oxygen from exercising muscle for aerobic glycolysis, with regular intensive physical exercise. These physiological adaptations are described as the athlete's heart³. Due to high vagal tone, bradycardia and repolarization abnormalities can be seen in athletes on the resting ECG too. There is enhanced diastolic filling in bradycardia, resulting in an increase of stroke volume during exercise. Prolonged intensive exercise results in structural changes, such as modest left ventricular (LV) wall

hypertrophy, over a period of time. Though these changes are generally reversible and benign, there may be a diagnostic overlap with pathological conditions, like the cardiomyopathies, in which strenuous exercise can lead to increased risk of adverse events including sudden death. Clinical algorithms are needed for differentiation between physiological and pathological changes. Well-defined structural limits for physiological adaptation to exercise are needed. The most commonly available tools being the ECG and echocardiogram. Dynamic exercise, such as distance running, leads to changes in muscle length and joint movement with relatively small intramuscular force while static exercise, as weight lifting, involves large intramuscular forces with relatively minor change in muscle length and joint movement⁴. Both dynamic and static exercise result in an increase in myocardial oxygen demand: heart rate, wall tension (before and after the contraction, which determines preload and afterload), and contractile state of the LV⁵. During high-intensity dynamic exercise, there is a large increase in heart rate and an increase in stroke volume while in high-intensity static exercise, a smaller increase occurs in heart rate, and little change occurs in end-diastolic and end-systolic volumes of the LV⁶. Therefore, dynamic exercise primarily causes a volume load on the LV, whereas static exercise causes a pressure load. Mostly all sports require a combination of both types of effort.

Pathological Changes with Endurance Physical Exercises

Intense physical exercise leads to increase in pulmonary arterial pressures, the pulmonary vascular bed can only reduce its resistance by 30–50% during exercise as compared with greater reductions in systemic vascular resistance (usually in excess of 75%)^{7,8}. Following a marathon, approximately 30% of runners develop acute dilation of cardiac chambers, especially the RV and RA, and dysfunction of the RV and ventricular septum⁹. There is a link between

lifelong endurance exercise and LGE (typically with a junctional spotty pattern), in highly trained athletes⁵. Myocardial areas located at the attachment of RV wall to the septum, where spotty LGE characteristically localizes, showed a loss of compact myocardium and markedly expanded extracellular space with interstitial fibrosis¹⁰.

Arrhythmias Caused or Precipitated By Exercise

Sinus bradycardia, defined as a sinus rate <60 beats per minute (bpm), is common in the athlete (1). Generally, it is attributed to enhanced vagal tone caused by conditioning and is thus physiological. Occasionally, heart rates can be as slow as 30 to 40 bpm at rest in the highly conditioned athlete and decrease to <30 bpm during sleep¹¹. Very Common ECG findings associated with low heart rate in athletes are Sinus bradycardia, Sinus arrhythmia, Sinus pauses. The Common findings are Wandering atrial pacemaker, Junctional escape beats, First-degree AV block, Type I second-degree AV block (Wenckebach phenomenon). Uncommon ones are Type II second-degree AV block, Third-degree AV block, Sino atrial type I block, Sino atrial exit block and Ventricular escape beats¹². The three main findings in systematic review of literature in which cardiac pauses were reported in competitive athletic individuals, are: first, cardiac pauses (.3 s) are observed in apparently healthy actively competitive athletes. Second, 3 s pause threshold does not adequately discriminate between asymptomatic and potentially symptomatic competitive athletes. Finally, despite pauses well in excess of 3 s being reported, few subjects were deemed to need intervention, the vast majority had no subsequent squealae that could be attributed to athletic participation¹³. There are increased risk of AF in athletes^{14,15,16}. Patients with underlying cardiac disease such as dilated cardiomyopathies, hypertrophic cardiomyopathy, Brugada syndrome, and catecholaminergic ventricular tachycardia (VT) have an increased risk of AF. AF in a child or

adolescent athlete is uncommon and should suggest a familial inheritance or the presence of an accessory pathway. There is an association between atrial fibrillation and/or flutter and endurance exercise training^{17,18}.

Evaluation before beginning of Intense Exercise Program

There are various levels, where competitive athletes with cardiovascular disease can be diagnosed: 1) detailed evaluation by a primary care physician; 2) systematic screening of families after diagnosis of genetic disease in a relative; 3) incidental findings on clinical examination or imaging, detected during interaction for another medical problem; 4) systematic screening of large populations, as school and college athletes, for determining eligibility for competitive sports, and 5) symptoms associated or unassociated with sports¹⁹.

Guidelines for allowing sports and exercise in different conditions²⁰

I. Sinus Bradycardia

1. Athletes with sinus bradycardia, sinus exit block, sinus pauses, and sinus arrhythmia without symptoms can participate in all competitive athletic activities if no structural heart disease or other arrhythmias (Class I; Level of Evidence C).
2. Athletes with symptomatic bradycardia should be evaluated for structural heart disease and be treated by an implanted pacemaker. They should be restricted from training and athletic competition while being evaluated (Class I; Level of Evidence C).

II. AV Block

1. Asymptomatic athletes with no structural heart disease and first-degree AV block (PR interval <0.3 ms) can participate in all competitive sports unless the person is at risk for progression to higher-degree block with symptoms (Class I; Level of Evidence C).
2. Asymptomatic athletes with first-degree AV block, in whom type I second-degree AV block

appears with exercise, should be evaluated further for possible intra-His or infra-His block with EPS (Class I; Level of Evidence C).

III. Type I Second-Degree (Wenckebach) AV Block

1. Asymptomatic athletes with structurally normal hearts and Wenckebach AV block (type I second degree AV block) with improvement in conduction with exercise or recovery can participate in all competitive sports (Class I; Level of Evidence C).
2. In athletes with Wenckebach AV block and coexisting bundle-branch block or with any indication that they are at risk for progression to higher-degree AV block, EPS should be performed to identify the presence of intra-His-Purkinje or infra-His-Purkinje block that may require pacemaker therapy (Class I; Level of Evidence C).

Type II Second-Degree (Mobitz) AV Block

Athletes with Mobitz type II second-degree AV block with a wide QRS, including isolated right bundle branch block (RBBB) should receive a permanent pacemaker (Class I; Level of Evidence C).

Complete RBBB

Athletes with RBBB, who do not develop periods of type II second-degree AV block or complete heart block spontaneously or during exercise and who have no symptoms or heart disease can participate in all competitive athletics (Class I; Level of Evidence C).

Complete Left Bundle-Branch Block

1. Athletes with permanent or rate-dependent LBBB who do not develop spontaneous type II second-degree AV block (Mobitz) or complete heart block and who have no symptoms or heart disease can participate in all competitive athletics (Class I; Level of Evidence C).
2. Athletes with abnormal AV conduction characterized by an HV interval >90 ms or a His-

Purkinje block should have pacemaker implantation (Class I; Level of Evidence C).

Congenital High-Grade or Complete Heart Block

Asymptomatic athletes without heart disease who have a junctional escape rhythm that has a QRS duration <120 ms, resting ventricular rates >40 bpm that increase appropriately with exertion, and exercise capacity that approximates that of the relevant sport can participate in athletic activity without restriction (Class I; Level of Evidence C).

Acquired Complete Heart Block

Athletes with acquired complete heart block should have a permanent pacemaker placed regardless of symptoms, type of structural heart disease, and exercise capacity unless the heart block is attributable to completely reversible causes and resolves completely (Class I; Level of Evidence C).

Athletes with Permanent Pacemakers

1. Generally, athletes with permanent pacemakers should be cleared for athletic participation if there are no limiting structural heart conditions or symptoms (Class I; Level of Evidence C).
2. Athletes who are completely pacemaker dependent should not engage in sports in which there is a risk of collision that could result in damage to the pacemaker system (Class I; Level of Evidence C).
3. For athletes with permanent pacemakers, protective equipment should be considered for participation in contact sports that have the potential to damage the implanted device (Class I; Level of Evidence C).

Atrial Fibrillation

1. Athletes with low-risk AF that is well tolerated and self-terminating may participate in all competitive sports without therapy (Class I; Level of Evidence C).

2. Catheter ablation for AF could obviate the need for rate control or antiarrhythmic drugs and should be considered (Class IIa; Level of Evidence B).

Atrial Flutter

Catheter ablation for typical AFL has a high likelihood of success and should be considered (Class I; Level of Evidence B).

AV Nodal Reentry Tachycardia, AV Reciprocating Tachycardia, Atrial Tachycardia

1. The treatment of choice for athletes with regular, acute-onset, symptomatic SVTs should be catheter ablation (Class I; Level of Evidence C).
2. Athletes with short refractory period bypass tracts capable of anterograde conduction and a history of paroxysmal AF should have an ablation of the accessory pathway before clearance for competitive sports because of risk for life-threatening arrhythmias (Class I; Level of Evidence B).

Ventricular Arrhythmias

1. Athletes with single PVCs and complex forms no greater than couplets at rest and during exercise testing without structural heart disease can participate in all competitive sports. The exercise testing protocol should be based on maximal performance rather than achieving 80% to 100% of the target heart rate to come as close as possible to the level of exertion achieved during their competitive sport (Class I; Level of Evidence C).
2. Ablation of PVCs may be considered in symptomatic patients with frequent PVCs resistant to medical therapy (Class IIb; Level of Evidence C).

Sustained Monomorphic VT

Athletes with structurally normal hearts and monomorphic sustained VT amenable to catheter ablation who undergo ablation and remain free of spontaneous or induced VT at least 3 months after the procedure can resume full competitive activities (Class I; Level of Evidence C).

Sustained Polymorphic VT, Ventricular Flutter, and Ventricular Fibrillation

Athletes who have survived a cardiac arrest caused by ventricular fibrillation or VT or who have had documented symptomatic rapid VT associated with a defined nonreversible cardiac abnormality should have an ICD placed. (Class I; Level of Evidence A).

Syncope

Athletes with exercise-induced syncope should be restricted from all competitive athletics until evaluated by a qualified medical professional (Class I; Level of Evidence B).

Conclusions

This review of literature does not deny the fact that physical activity can reduce cardiovascular morbidity and mortality. But we have to know that adaptation of the athlete's heart is not always able to accommodate the sustained haemodynamic loads. Like other sports injuries, recognizing the problem in an early phase can prevent disaster and may lead to measures that allow safe and enjoyable continuation of sports participation.

Athletes are generally regarded as the healthiest individuals in society. Finally as Hippocrates noted centuries ago, "If we could give every individual the right amount of nourishment and exercise, not too little and not too much, we would have found the safest way to health"²¹. These words from 2,500 years ago still seem prudent and wise today.

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