Review Article

A Look Into The Athlete's Heart

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ABSTRACT

The concept of "athlete's heart" has been traced through early clinical and radiographic studies to modern imaging techniques. Athletes heart has excited clinicians and scientists for more than a century. . Earlier investigations in the last century have shown cardiac enlargement and bradyarrhythmias in individuals with above-normal exercise. Belief that hypertrophic cardiomyopathy is the commonest cause of sports related death in young adults is traced to weak diagnostic criteria and frequent republication of a very small group of cases.

Assessment of young adults in sports participation by echocardiographic criteria has been gaining wide acceptance.. This review provides an insight of the science of cardiac remodeling in athletes and an overview of common clinical issues that are encountered in the cardiovascular care of the athlete.

Key Words : Athlet's Heart, Echocardiography criteria, Hypertrophic cardiomyopathy

Introduction -

"No man becomes a great runner or oarsman who has not naturally a capable if not a large heart" - Osler.

The concept of "athlete's heart" has been traced through early clinical and radiographic studies to modern imaging techniques. Athletes heart has excited clinicians and scientists for more than a century. Earlier investigations in the last century have shown cardiac enlargement and bradyarrhythmias in individuals with above-normal exercise. Lower limits of criteria for the diagnosis of a "pathological" enlargement of the heart have frequently been revised as the prevalence of bigger hearts has been recognised in both endurance and above normal exercise competitors. Belief that hypertrophic cardiomyopathy is the commonest cause of sports related death in young adults is traced to weak diagnostic criteria and frequent republication of a very small group of cases. Genetically based screening tests may become available in the future, but the assessment of young adults in sports participation by echocardiographic criteria has been gaining wide acceptance. It is now well established that repetitive participation in vigorous physical exercise results in significant changes in myocardial structure and function. For most people, the development of a large heart is not a pathological sign - rather, it is a desirable outcome that will enhance performance on the sports field, and will allow longer independence in old age. This review provides an insight of the science of cardiac remodeling in athletes and an overview of common clinical issues that are encountered in the cardiovascular care of the athlete.

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Historical Aspect -

In the early days of the Olympian movement, Henschen' demonstrated enlargement of the heart in cross country skiers by careful percussion of the chest. He wrote "an enlarged heart is a good thing if it can perform more work over an extended period of time". Other features that can be detected on simple clinical examination include a prominent, displaced apical impulse and a right ventricular lift.[1] However, recognition of the large heart of endurance players became more widespread with the advent of radiography, as examination of the cardiac shadow began to supplement the very crude percussion techniques of clinicians such as Henschen. In the early 1900s, Paul Dudley White[2] studied radial pulse rate and pattern among Boston Marathons, and was the first to publish resting sinus bradycardia in long-

distance runners.[3] Early chest radiography work confirmed the physical examination findings of Darling and Henschen by showing global cardiac enlargement in trained athletes.[4] Then development of electrocardiogram enabled widespread study of electric activity in the heart of the trained athlete.[5] The recent development of 2-dimensional echocardiography led to important further advances in our understanding of the athlete's heart. Identification of ventricular chamber enlargement, myocardial hypertrophy, and atrial enlargement have led to a more comprehensive understanding of the athlete's heart. Most recently, advanced echocardiographic techniques and magnetic resonance imaging have helped to understand the functional adaptations that happen in previously reported structural characteristics of the athlete's heart.

Concept of Cardiac Remodeling -

Exercise induced cardiac remodeling has been reported in the literature.[6,7] There is a direct relationship between exercise intensity (external work) and the body's demand for oxygen. This oxygen demand is met by increasing pulmonary oxygen uptake (VO2). In the healthy human, there is a direct and inviolate relationship between VO2 and cardiac output.

Cardiac output, the product of stroke volume and heart rate, may increase 5- to 6-fold during maximal exercise effort. Coordinated autonomic nervous system function, characterized by rapid and sustained parasympathetic withdrawal along with sympathetic activation, is required for this to occur. Heart is so conditioned that heart rate in the athlete may range from <40 bpm at rest to >200 bpm in a young maximally exercising athlete. Heart rate increase is responsible for the majority of cardiac output augmentation during exercise.

In contrast, stroke volume both at rest and during exercise may increase significantly with prolonged exercise training. Cardiac chamber enlargement and the accompanying ability to generate a large stroke volume are direct results of exercise training and cardiovascular hallmarks of the endurance-trained athlete. Stroke volume rises during exercise as a result of increases in ventricular end-diastolic volume and, to a lesser degree, sympathetically mediated reduction in end-systolic volume.

Hemodynamic adaptations, specifically changes in cardiac output and peripheral vascular resistance, vary widely across sporting disciplines. Exercise activity can be segregated into two forms with defining hemodynamic differences. Isotonic exercise, as also called as endurance exercise, involves sustained elevations in cardiac output, with normal or reduced peripheral vascular resistance. In this there is primarily a volume challenge for the heart that affects all 4 chambers. This form of exercise underlies activities such as long-distance running, cycling, rowing, and swimming. In contrast, isometric exercise, further referred to as strength training, involves activity characterized by increased peripheral vascular resistance and normal or only slightly elevated cardiac output. This increase in peripheral vascular resistance causes transient but potentially marked systolic hypertension and LV afterload. Isometric exercise is seen in activities such as weightlifting, track and field throwing events. Many sports, including popular team-based activities such as soccer, lacrosse, basketball, hockey, and field hockey, involve significant elements of both endurance and strength exercise. As discussed later, sport-specific hemodynamic conditions may play an important role in cardiac remodeling.

Left Ventricle -

The impact of exercise training on LV structure has been evaluated extensive off late. Early studies with ECG demonstrated a high prevalence of increased cardiac voltage suggestive of LV enlargement in trained athletes.[8] Subsequent work with 2-dimensional echocardiography confirmed underlying LV hypertrophy and dilation.[9] Pelliccia et al[10] reported echocardiographic LV end-diastolic cavity dimensions in a large group (n=1309) of Italian elite athletes. Markedly dilated LV chambers (>60 mm) were most common in athletes with higher body mass and those participating in endurance sports (cycling, cross-country skiing, and canoeing).

Pellicciaet al[11] have also reported echocardiographic measurements of LV wall thicknesses among 947 elite Italian athletes. Within this cohort, a significant percentage of athletes (1.7%) had LV wall thicknesses =13 mm, and all of these individuals had concomitant LV cavity dilation. It must be emphasized that LV wall thickness above 13 mm is a rare finding in healthy athletes. This finding should prompt consideration of pathological hypertrophy requiring further diagnostic assessment. Furthermore, it is has been consistently shown that the most marked LV hypertrophy occurs in athletes with relatively large body size, and those of Afro-Caribbean descent. Hence a careful interpretation of LV hypertrophy in athletes, particularly with respect to differentiating adaptive from pathological hypertrophy, requires consideration of body size and ethnicity.

Morganroth et al reported for the first time that endurance-based exercise and strength-based exercise lead to distinctly different changes in LV structure.[11] This study used M-mode echocardiographic for LV measurements in wrestlers (strength training), swimmers (endurance training), and sedentary control subjects and found significant differences across these 3 groups. Specifically, athletes exposed to strength training demonstrated concentric LV hypertrophy, whereas individuals exposed to endurance training demonstrated eccentric LV enlargement. This study led to the concept of sport-specific cardiac remodeling, often referred to as the Morganroth hypothesis. The interested reader is referred to a recent comprehensive review of this topic by Naylor and colleagues.[12]

Exercise-induced remodelling in LV function have also been under investigation. Numerous investigators have examined resting LV systolic function in athletes using cross-sectional, sedentary control study designs.[13,14] These studies and a large meta-analysis show that LV ejection fraction is generally normal among athletes. Recent advances in functional myocardial imaging, including tissue Doppler echocardiography and strain echocardiography, have also suggested that exercise training may lead to changes in LV systolic function that are not detected by assessment of a global index like LV ejection fraction. The importance of these findings with respect to our understanding of exercise physiology and for differentiating athletic from pathological remodeling is an area of active investigation.

Left ventricular diastolic function has also been extensively evaluated in trained athletes with the use of conventional 2-dimensional (transmitral) and tissue Doppler echocardiography. It is now well recognized that endurance exercise training leads to enhanced early diastolic LV filling as assessed by E-wave velocity and mitral annular/LV tissue velocities.[15,16] It is likely that improved LV diastolic function is an essential mechanism of stroke volume preservation during exercise. There are sparse data examining diastolic function in strength-trained athletes, but a longitudinal study suggested that the concentric LV hypertrophy associated with strength training is accompanied by either unchanged or relative impairment of LV relaxation.[17]

Right Ventricle -

Exercise-induced cardiac remodeling affects the right ventricle (RV) along with LV. Endurance exercise requires both the LV and RV to accept and eject relatively large quantities of blood. In the absence of significant shunting, both chambers must augment function to accomplish this task. Advances in noninvasive imaging has helped in understanding how the RV responds to the repeated challenges of exercise. An initial study using M-mode echocardiographic demonstrated symmetrical RV and LV enlargement in a small (n=12) cohort of highly trained endurance athletes.[18] Henriksen et al[19] assessed RV and LV cavity and wall measurements using M-mode and 2-dimensional echocardiography in 127 male elite endurance athletes. He reported that endurance athletes demonstrated significantly larger RV cavities and a trend toward thicker RV free walls. Recently studies confirmed by magnetic resonance imaging that RV enlargement is common among endurance athletes.[20,21]

The impact of strength training on the RV remains unclear because the limited available data are inconsistent. Perseghin et al[22] compared RV and LV structure in endurance athletes (marathon runners), strength athletes (sprinters), and sedentary control subjects and found the largest RV volumes among the strength athletes. But there were no significant difference between the RV dimensions in strength and endurance athletes after adjustment for body surface area. Right ventricular structure in collegiate endurance-trained (rowers) and strength-trained (American-style football players) athletes was recently assessed before and after 90 days of team-based exercise training. There was statistically significant RV dilation in the endurance athletes but no changes in RV architecture in the strength athletes. Further elucidation of how the RV responds to different forms of exercise and its contribution to exercise capacity is an important area for future work.

The Left Atrium -

Assessment of left atrium in atheletes has been of interest. Hauser et al[23] demonstrated by an echocardiographic study that a small group of endurance athletes (n=12) had larger left atria than sedentary control subjects. Hoogsteen et al[24] compared atrial dimensions in young competitive cyclists with those in older more experienced cyclists and found larger dimensions in the older athletes. Pelliccia et al [25, 26] presented the largest data set of atrial measurements in athletes (n=1777) and demonstrated that left atrial enlargement (>40 mm in an anterior / posterior transthoracic echocardiographic view) was present in 20% of the athletes.

Cardiac Hypertrophy : Physiological vs Pathology Adaptation

There is a lot of overlap between exercise-induced cardiac remodeling and pathological structural heart disease and is the reason of wide research. Severe form of exercise-induced ventricular remodeling may be difficult to differentiate from mild forms of hypertrophic cardiomyopathy, familial or acquired dilated cardiomyopathy, and arrhythmogenic RV cardiomyopathy. The clinical task of differentiating marked exercise-induced remodeling from these important forms of disease remains important, with implications including sport restriction, pharmacological therapy, and placement of an implantable cardiac defibrillator.

Maron gray zone defines the overlap between features of the athlete's heart and characteristics of cardiomyopathy, specifically hypertrophic cardiomyopathy, that may affect young athletes.[27] A valuable schema for approaching the athletic patient with LV hypertrophy of unclear origin has been presented, and remains useful in clinical practice.[28] This diagnostic approach was developed when noninvasive cardiovascular imaging was in its infancy and, for the most part, restricted to basic 2-dimensional echocardiography. Recent advances in cardiovascular diagnostics have proven to be useful additions to these original criteria.

Functional myocardial echocardiography, including tissue Doppler and speckle-tracking imaging, permits detailed and accurate assessment of myocardial function. Tissue Doppler imaging permits assessment of myocardial relaxation and contraction velocity. In athletes, early diastolic relaxation velocity are normal or increased in athletes with LV hypertrophy resulting from exercise-induced remodeling.[29] In contrast, pathological forms of LV hypertrophy are typically associated with reduced early diastolic relaxation velocity and peak systolic tissue velocity.[30] Tissue strain and strain rate also provide useful insight into the origin of LV hypertrophy in athletes. Most of the studies have reported higher epicardial longitudinal and circumferential strains in endurance athletes. They have also reported that a progressive increase in LV apical twist may represent an important component of myocardial remodeling. Deformation parameters are more marked in RV rather than LV.

Cardiac magnetic resonance imaging (MRI) is also an important tool for the evaluation of the athlete with cardiac enlargement. Cardiac MRI allows highly accurate assessment of myocardial thickness, chamber volumes, tissue composition, extra cardiac

anatomy, and cardiac magnetic resonancederived reference values in athletes have been published.[31] The use of late enhanced gadolinium helps in assessment of myocardial fibrosis which is rarely seen in athletes. At the present time, we use cardiac magnetic resonance in athletes with either indeterminate echocardiographic imaging or clinical features that suggest a diagnosis that may not be definitively assessed by echocardiography (ie, myocarditis).

Sudden Cardiac Death -

The reason for interest in athletes' cardiac evaluation is because of sudden death occurring in these athletic individual community. Studies examining sudden death in athletes report a wide range of prevalence. The variability in sudden death prevalence statistics data may be attributed to multiple factors, such as geographic variability in the prevalence of causal diseases, characteristics of the populations studied, and case ascertainment techniques. Although there appears to be some regional difference in the relative contribution causal diseases, hypertrophic cardiomyopathy is the most common cause of sudden cardiac death in the young in the United States. Sudden death has been documented in most types of competitive sports, but may be more common during participation in physically intense sports, such as basketball, soccer, and American-style football. In addition to sport type, sex and ethnicity appear to contribute to sudden death risk, with male participants and individuals of Afro-Caribbean descent more likely to succumb to sport-related sudden death.

Most cases of sudden, sport-related death in young athletes are attributable to underlying cardiovascular pathology. Both the American Heart Association/American College of Cardiology[32] and the European Society of Cardiology[33] have established sport eligibility criteria for individuals diagnosed with these conditions. Guidelines endorsed by these 2 groups are largely similar.

The difficult situation of sudden death in young, previously asymptomatic athletes has led to considerable efforts aimed at prevention. The logic that concept of detection and management of cardiovascular disease before sport participation may reduce the incidence of sudden cardiac death has led to recommendations for preparticipation screening. The American Heart Association / American College of Cardiology[34] and the European Society of Cardiology[35] have published consensus committee-based recommendations for preparticipation athlete screening. Both governing bodies recommend a focused medical history and physical examination. The European Society of Cardiology recommends the addition of a 12-lead ECG. This addition of a 12-lead ECG to medical history and physical remains an area of intense debate. However, a number of issues, ie, the financial and manpower costs of the mandated ECG, the high rate of false-positive ECG findings, the cost of follow-up testing for those with abnormal results, the logistics of ECG acquisition and interpretation, and considerations about future insurability for athletes with detected disease, represent considerable obstacles to implementing a mandatory 12-lead ECG as part of preparticipation screening in the United States. Although additional observational data from organizations or nations using ECG are welcomed, this issue will almost certainly remain controversial until a prospective, randomized, multinational trial is conducted to provide a definitive answer. In the absence of such data, priority should be placed on widespread dissemination and implication of current history and physical examination recommendations with consideration of ECG only in localities with sufficient resources and expertise for this technique.

Conclusion and future Perspective -

There has been a lot of research involving athletes heart that has helped in understanding myocardial remodeling seen in athletes. The relationship between exercise dose (intensity, frequency, duration) and both hypertrophy and subsequent regression during detraining remains inadequately characterized. Also variability in the magnitude of remodeling within seemingly homogenous groups, likely a function of both genetic and environmental factors, remains poorly understood. The exact functional aspects of exercise-induced remodeling that facilitate preserved or enhanced stroke volume

during high-level exercise remain to be elucidated. A properly conducted multicentric longitudinal study is needed to address these areas of scientific uncertainty.

Also it is necessary to differentiate adaptive from pathological cardiomyopathy that integrates basic clinical factors with modern diagnostic tests (imaging, cardiopulmonary exercise testing, and genetic assessment). Also there should be a timely update to the consensus committee guidelines for the management of cardiovascular disease in athletes. Also a prospective, longitudinal study examining the impact of preparticipation ECG screening should be conducted to end the ongoing debate about how best to reduce the incidence of sport-related sudden cardiac death. As the general population's interest in exercise and competitive sport continues to rise, the time has come to address these important scientific and clinical issue.

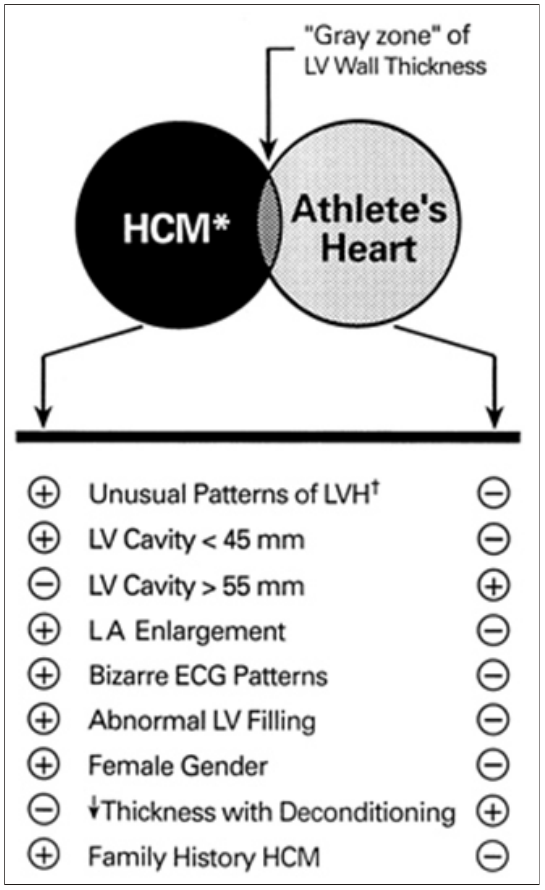

Figure 2 : **Differentiation between Atheletes heart and HCM**

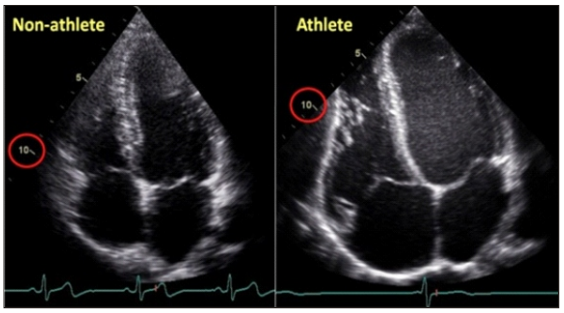

Figure 1 : **Showing difference between a normal heart and a atheletes heart. Number 10 indicates the thickness of LV wall**

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