Junior athletic heart: insights from echocardiography

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Abstract:

Athlete's heart is a constellation of structural and functional changes that occur in the heart of people who participate in sports. The echocardiographic assessment of athlete's heart can be a challenging. Although it would seem that the huge cardiac capacity of the trained athlete should be easily distinguishable from heart disease, there are several instances in which exercise-induced "physiological" myocardial remodeling, including changes in ventricular chamber size and wall thickness, can mimic pathological structural changes associated with inherited and acquired cardiac disorders. The relationship between different sports types and cardiac structures especially in adults has been investigated in many previous studies. However, there are few studies in children and adolescents about the effects of sports training on cardiac functions and structures.

Keywords: Athlete's heart, children, echocardiography, sports activity

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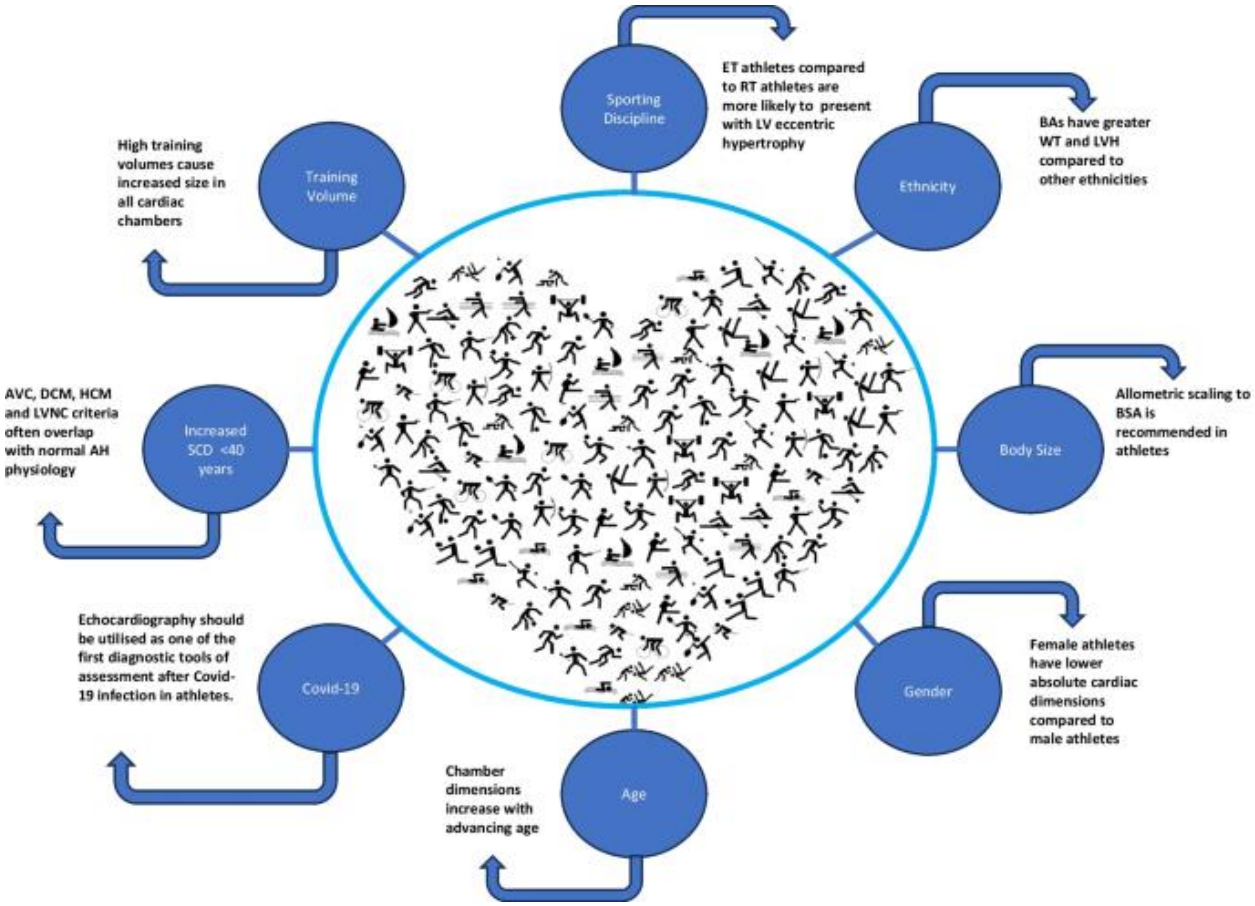
Introduction:

Exercise training is associated with an array of morphological and functional cardiac adaptations and termed the 'athlete's heart' (AH). These manifestations can include an increase in the left and right cardiac cavity sizes, increased left ventricular (LV) wall thickness and an augmentation in indices of resting and exercise systolic and diastolic function at rest and during exercise, when compared to that of non-athletic individuals [1-3].

The multifactorial nature of AH morphology (Fig. 1) and functional changes linked with prolonged training exposure (> 4 h per week) [4] can create a diagnostic challenge during echocardiographic assessment. The extent of athletic adaptation often extends beyond 'normal' limits of cardiac dimensions and function, making it challenging to differentiate between AH physiology and inherited conditions such as hypertrophic cardiomyopathy (HCM), arrhythmogenic (right ventricular) cardiomyopathy (AVC), dilated cardiomyopathy (DCM) and isolated left ventricular non-compaction (LVNC) [5].



Multifactorial nature of the athlete's heart

Exercise-Induced Cardiac Remodeling

Exercise-induced cardiac remodeling, often termed the "athlete's heart," is an adaptive increase in cardiac chamber size and wall thickness that is promoted by the volumes and pressure loads of exercise. These changes are accompanied by enhancements in lusitropic and contractile function

that enable the heart to fill and eject larger volumes than nonathletes during exercise. Maximal heart rates in athletes are similar to nonathletes, and thus, the greater exercise cardiac outputs observed in athletes are entirely caused by this ability to generate larger stroke volumes.

Exercise can have a profound influence on cardiac size and mass, and this is dependent on the type and duration of exercise. In 1975, Morganroth et al [6] provided a description of the electrical, structural, and functional changes in the athlete's myocardium that has largely remained the orthodox model for appreciating the physiology and consequence of athletic training. They compared athletes of differing endurance and strength-based training regimes and observed that cardiac mass was approximately 35% greater in athletes than nonathletes, and that cardiac volumes were up to 60% to 80% greater. They noted that endurance athletic training resulted in marked increases in volume with preservation of wall thickness, and thus, an overall increase in mass, whereas strength training resulted in an increase in wall thickness and cardiac mass but minimal change in volumes. [7]

Cardiac remodeling and genes

A key element of athletic performance, especially for endurance athletes, is cardiac function. It's interesting to consider the possibility that certain people are predestined to be elite athletes due to their larger, thicker, or more compliant hearts prior to exercise training. Longitudinal training studies in previously nonathletic persons have shown improvements in fitness and changes in heart sizes that are significantly less than those seen in elite athletes, and .In support of this theory, elite athletic hearts remain enlarged after long-term detraining [8]

The degree to which genetic factors may contribute to individual variability in physiological cardiac remodeling is unknown, while there are several ways in which an athlete's genetic composition may affect heart structure and function .genes can affect energy metabolism, autonomic nervous system basal torn responses, salt and water homeostasis

Genetic risk could be conferred by single rare variants or by combinations of low-frequency and/or common variants.[9]

Physiology of exercise:

During exercise, heart rate rises up to threefold from the resting values at peak exercise. This rise is mediated primarily by the autonomic nervous system via an increase in sympathetic activity and a reduction in parasympathetic activity which is originally one of the parameters utilized to control exercise intensity.[10]

An important parameter to be considered is Cardiac Output, a constant product of Stroke Volume and Heart Rate, thus characterizing the Volume Ejected by the Left Ventricle per minute. It increases up to 5 folds baseline and its increase is mainly due to increase in heart rate, it presents lower values comparing to values found in adults, reflecting the smaller cardiac volumes before the end of the maturation process.[11]

Stroke Volume, is defined as the amount of blood ejected from the left ventricle during systole, usually behaves as a plateau during increasing exercise intensity, it increases one and half to two folds maximum, this increase is due to:

1. Increased cardiac contractility secondary to increased adrenergic stimulation

2. Decreased afterload secondary to a dramatic decline in systemic and pulmonary vascular resistance during exercise

3. Enhanced ventricular filling secondary to the pumping action of the skeletal muscles (11)

preparticipation evaluation for junior athletes:

The intent of the PPE is to promote the health and safety of young athletes participating in training and competition and to identify those who may need additional evaluation before participation or, rarely, exclusion from sports.

Sudden cardiac death (SCD) in a young athlete is tragic and shocking, particularly because of the unexpected nature of such an event in a generally healthy population. Accordingly, strategies for the prevention of SCD in athletes have been the subject of study and debate among communities and sporting organizations.

Although preparticipation screening is widely supported, the debate regarding the best screening method for young athletes continues. The American Heart Association (AHA) published recommended screening elements for clinicians to include in the annual preparticipation assessment of athletes. In table 1 the last update of PPE.

TABLE 1

The 14-Element AHA Recommendations for Cardiac Screening of Adolescent Athletes:

Medical history	Physical examination
Personal history	11. Heart murmur
1. Exertional chest pain/discomfort	12. Femoral pulses to exclude aortic coarctation
2. Unexplained syncope/near-syncope	13. Physical stigmata of Marfan syndrome
3. Excessive exertional and unexplained dyspnea/fatigue, associated with exercise	14. Brachial artery blood pressure (sitting position)
4. Previous recognition of a heart murmur	
5. Elevated systemic blood pressure	
6. Previous restriction from participation in sports	
7. Previous testing for the heart, ordered by a physician	
Family history	
8. Premature death (sudden and unexpected or otherwise) before age 50 y due to heart disease, in ≥1 relative	
9. Disability from heart disease in a close relative <50 y of age	
10. Specific knowledge of certain cardiac conditions in family members: hypertrophic cardiomyopathy or dilated cardiomyopathy, long QT syndrome or other ion channelopathies, Marfan syndrome, or clinically important arrhythmias	

Adapted with permission from Maron BJ, Friedman RA, Kligfield P, et al. Assessment of the 12-lead ECG as a screening test for detection of cardiovascular disease in healthy general populations of young people (12–25 years of age): a scientific statement from the American Heart Association and the American College of Cardiology. Circulation. 2014;130(15):305.[12]

ECG changes:

The 12-lead ECG is the primary cardiac screening tool in athletes. Moreover, even in the USA where the ECG is less commonly used as an athletic screening tool, many pediatricians will use the ECG selectively for assessment rather than relying on clinical examination and pre participation questionnaire alone, therefore detailed knowledge of the specifics of the pediatric ECG is required when assessing pediatric athletes.13

Standardized ECG interpretation criteria, first introduced by the ESC (European society of cardiology) in 2010, distinguished physiological adaptations from pathological abnormalities, which led to improvements in interpretation accuracy. Refinement of these criteria over the last decade has been facilitated by a greater understanding of the athlete's heart [14].

." The physiological changes associated with the athlete's heart manifest as electrical changes on the athlete's ECG and, in some cases, mimic those observed in patients with cardiomyopathy and give false positive results

The ECG may be normal in up to 10% of athletes with HCM, 70% who are genotype positive for long QT syndrome, and 90% with premature coronary artery disease. Furthermore, the resting ECG is normal in almost all individuals with anomalous coronary arteries, catecholaminergic polymorphic ventricular tachycardia, and aortopathies.[15]

Normal ECG finding in junior athletes:

Sinus bradycardia:

HR in athletes is lower than non-athletes it was reported that HR may be as low as 30 beat per minute in pediatric age group.[16]

T-wave inversion

T-wave inversion V1–V3 in athletes <16 years is normal finding, although TWI affecting the lateral leads (V5-V6, I and aVL) is considered abnormal and should prompt comprehensive investigation irrespective of ethnicity, including cardiac MRI, when echocardiography is non-diagnostic.[17]

Incomplete right bundle branch block

IRBBB is defined by a QRS duration <100 ms with an RBBB pattern: terminal R wave in lead V1 (rsR′) and wide terminal S wave in leads I and V6. IRBBB is seen in less than 10% of the general population but is observed in up to 40% of highly trained athletes. (18)

Abnormal ECG findings that require further work up:

T-wave inversion (inferior or anterolateral)

ST-segment depression

pathologic Q-waves

complete left bundle branch block (LBBB)

epsilon wave

ventricular pre-excitation

and prolonged QTc [19].

Several common heritable cardiomyopathies including HCM (hypertrophic cardiomyopathy), ARVC (arrhythmogenic right ventricular cardiomyopathy) and familial DCM (dialated cardiomyopathy) may present with ECG abnormalities prior to the onset of overt heart muscle pathology.

athletes with abnormal ECGs suggestive of cardiomyopathy and initially normal clinical evaluations should be followed with serial evaluation during and after their competitive athletic careers.[20]

Cardiovascular Imaging in Athletes' Heart

4.1. Echocardiography

Transthoracic echocardiography (TTE) is the primary imaging tool for evaluating cardiac structure and function in athletes due to its accessibility, affordability, and noninvasive nature. It offers insights into exercise-induced cardiac remodeling and comprehensively evaluates cardiac structures and function. Furthermore, some authors suggested using a focused TTE in the pre-participation cardiovascular screening [21]. In recent years, progress in ultrasound technology has transformed the field of echocardiography. This test now provides detailed analyses of chamber measurements and systolic and diastolic function, using techniques such as 2D and 3D imaging and tissue Doppler imaging (TDI). It is invaluable in identifying "grey zone" athletes, where additional imaging modalities or longitudinal follow-up may be necessary.

Doppler echocardiography has refined the understanding of cardiac remodeling, identifying normal adaptive changes from disease states [22].

Left Ventricle (LV) Dimensions, Mass, and Wall Thickness

Long-term athletic activities have been shown to impact the size, wall thickness, and function of the LV (left ventricle).

There is a widespread agreement that ET (endurance training) elicits LV dilation with increased LV mass, LVEDV (left ventricular end diastolic volume) and LV end-diastolic diameter (LVEDD), whereas neither concentric hypertrophy nor remodeling are now. thought to be expressed in RT athletes [23].

Utomi et al. conducted a large meta-analysis, including 185 echocardiographic and 41 MRI datasets, comparing cardiac structures between athletes and sedentary controls. Findings showed that endurance and strength-trained athletes have larger LV dimensions than sedentary controls, with more pronounced differences in endurance athletes, especially cyclists and rowers [24].

Left Ventricular Systolic Function

Lower resting LV ejection fraction (EF) in endurance athletes has been demonstrated in previous research (12% found to have an LVEF of < 52%. This can be accounted for by the increased LVEDV in that a larger LV does not require the same contraction force as a smaller chamber to achieve the same stroke volume.

LV strain and strain rate imaging, via TDI or speckle-tracking echocardiography, may help differentiate maladaptive from physiological remodeling. [25]. TDI measures the velocity of myocardial movements using Doppler principles. It evaluates the speed at which the myocardium contracts and relaxes [26]. Strain imaging, via TDI or speckle-tracking echocardiography, measures the percentage change in myocardial fiber length during the cardiac cycle. Left ventricular global longitudinal strain (LV GLS) is considered more sensitive than LVEF for identifying subclinical LV dysfunction. (27)

Left Ventricular Diastolic Function

Diastolic function assessment is multiparametric as Doppler parameters are reliant on both atrial and LV pressures Unsurprisingly, the findings are equivocal, with many studies observing no differences in peak E velocity between sedentary controls and athletes. At odds to this, the ratios of early to late diastolic filling (E/A—blood flow velocity, e′/a′—myocardial velocity) have been increased in ET athletes [26] potentially due to enhanced early diastolic filling [28]

Right Ventricle

Exercise training can result in changes to the right ventricle (RV) function and structural remodeling, but research on the RV adaptations to high-intensity training has traditionally been less advanced than for the LV . The complex anatomy of RV makes it challenging for conventional echocardiography to assess its size and function accurately .The RV shares the hemodynamic load with the LV and responds to intensive exercise similarly, increasing in size while maintaining systolic and diastolic function

Studies by Baggish et al. typically display increased RV end-diastolic volume (RVEDV), increased wall thickness, RV mass, and RV stroke volume (RVSV) compared to controls . Long-term endurance training promotes an increase in all RV-indexed dimensions and induces a more spherical RV shape [29].

Atria

The left and right Atria undergo changes in AH, observed in echocardiograms. Studies have shown that LA remodeling in athletes is characterized by increased LA volume index (LAVI) and improved myocardial diastolic properties, which can be identified using DTI and speckle-tracking echocardiography.

A meta-analysis of nine studies involving 403 elite athletes and 297 controls revealed higher LA volumes in athletes but reduced global LA longitudinal strain (PALS), indicating decreased reservoir function. Late diastolic LA strain rate (LASR late) is also lower in athletes, while contraction strain and systolic strain (LASR syst) are similar between athletes and controls. The meta-analysis suggests that reduced LA function in athletes may contribute to a higher risk of atrial fibrillation. [30]

Aorta:

Larger aortic root dimensions tend to be present in some athletes compared with sedentary controls It is therefore recommended for clinicians who are evaluating athletes that if there is a marked aortic root dilatation present then this is likely representative of a pathological process and not a physiological adaptation to exercise. Additionally, significant ascending aorta dilatation and aortic regurgitation have been found to be uncommon in RT athletes [31],

Exercise Stress Echocardiography (ESE)

ESE is a reliable, safe, noninvasive imaging test that evaluates dynamic cardiac function. ESE's primary importance lies in detecting exercise-induced abnormalities, such as ischemia and arrhythmias, which might not be visible at rest in athletes with suspected coronary artery disease or congenital coronary artery anomalies [32]. Furthermore, ESE can assess contractile reserve during exercise in endurance athletes with LV and/or RV dilatation and mildly reduced EF at rest, and an increase of LV EF of at least 15% during exercise may support the diagnosis of AH [33].

Speckle tracking Echocardiography (STE)

More recent studies focused on the speckle tracking imaging for the assessment of LV global and regional function. The STE or strain analysis is a relatively new and non-invasive imaging method based on tracking of characteristic speckle patterns created by interference of ultrasound beams in the myocardium, and has the advantage of measuring tissue velocities and deformation in an angle-independent fashion. The technique allows for simultaneous qualification in the long and short axes and quantification of regional wall motion abnormalities in all the myocardial segments (global longitudinal, circumferential and axial strain). Before the advent of STE, three-dimensional deformation involving different regions in LV could be analyzed only by magnetic resonance imaging (MRI), which is a costly and not always available in our daily practice. Recently, STE–derived measurements have been showing high feasibility and reproducibility.34,35

. Indeed, LV hypertrophy in hypertension and HCM is related to sudden cardiac death and mortality, however, hypertrophy in athlete's heart is accepted as a physiologic response. Thus, the STE can be considered as a helpful tool to differentiate diagnosis of physiological versus pathological LV hypertrophy. Many studies have been designed to assess differences in LV deformation in athletes and in patients with essential hypertension and HCM. All these studies conclude that the reduction in longitudinal strain is an early sign of LV dysfunction. Thereby, a significant reduction in LV GLS is an uncommon feature in athletes' heart and shouldn't be considered a physiological adaptation to training and an athlete presenting significant reduced LV strain should be carefully evaluated, particularly in the presence of an important hypertrophy . Finally, data about myocardial deformation in young athletes remain limited up to now. Further larger studies using speckle tracking imaging are needed to highlight cardiac adaptation in young athletes.36,37

Power athletes had the lowest resting LV GLS values compared to mixed and endurance athletes due to static exercise regimes imposing a significant pressure load on the LV. Resting LV mechanics in athletes are characterized by a balanced decrease in GLS and global circumferential strain (GCS).

A balanced decrease in LV GLS and GCS suggests a physiological rather than pathological process [38].

Cardiac Magnetic Resonance (CMR) Imaging

Accurately describing cardiac volumes, mass, contractility, and wall motion makes CMR a useful diagnostic tool for differentiating between cardiomyopathy and normal adaptation. CMR offers sophisticated myocardial tissue characterisation with exceptional accuracy and precision, making it the gold standard for determining biventricular masses and volumes as well as measuring volumes and flow.[39]

CMR is particularly useful in cases where echocardiography results are uncertain. However, its use as a primary screening tool is limited due to availability and cost,

CMR enables accurate morphological and functional assessment, including tissue characterization by late gadolinium enhancement (LGE), which can reveal distinctive patterns in HCM and other myocardial conditions [40].

Computed Tomography (CT) Imaging

An accurate, noninvasive technique for evaluating the morphology of the coronary arteries and the size of the major vessels is cardiac computed tomography angiography (CCTA). Despite its accuracy, CCTA is not frequently used as the main imaging modality for AH since there are few research devoted to its use in athletes.
However, both CCTA and CMR can offer comprehensive anatomical assessments when there are concerns of aortopathy or abnormal coronary arteries,
furthermore at least one thorough aortic tomographic evaluation by CCTA should be carried out whenever dilatation of the ascending aorta or aortic root is suspected.[41]

Differentiation between Athlete's Heart from pathological conditions:

The integration of clinical imaging data with other clinical factors, including the presence or absence of symptoms, family history of hereditary heart disease or sudden cardiac death, and 12-lead ECG readings, is essential. Therefore, the choice of diagnostic approach should always be guided by clinical suspicions and consider the entire clinical scenario, encompassing the full spectrum of cardiovascular diseases that can affect athletes. These steps help us to differentiate athletic heart from pathological conditions such as HCM, DCM, AVC, LVNC.[42]

Dilated Cardiomyopathy (DCM)

Dilated cardiomyopathy (DCM) is a mixed bag of heart diseases with the unique features of cardiac dilatation and subnormal to poor myocardial contractility. Dilated cardiomyopathy in the pediatric age group is generally characterized by unobstructed, dilated, and contracting left ventricular chamber defects and is associated with heart failure.

In echo, morphologically, we can identify biventricular dilatation as an increase in left ventricular end-diastolic dimension (LVDD) and left ventricular end-systolic size (LSVD) with decreased LV contractility. It also shows a reduced ejection fraction along with dilatation of the left ventricle with regular or thin walls. [43]

In athletes, despite often having severely enlarged LV dimensions, they typically maintain a preserved or low-normal EF, normal wall motion, and superior diastolic performance, differentiating their condition from DCM.

ESE (Exercise stress Echocardiography), an excellent, noninvasive test for athletes, can detect exercise-induced ischemia and assess contractile reserve in athletes with ventricular dilatation and mildly reduced LVEF to help differentiate them from DCM [44].

Hypertrophic Cardiomyopathy (HCM)

Hypertrophic cardiomyopathy (HCM) is the most common form of cardiomyopathy, affecting at least 1 in 500 individuals, and is a leading cause of sudden cardiac death (SCD) in adolescents and young adults.[45]

Echocardiography is also a useful tool to differentiate HCM from the so-called 'athletic heart'. Patients with HCM, unlike athletic heart, have small LV cavity dimensions, as well as evidence of diastolic (reduced tissue Doppler velocity) and myocardial deformation (strain) abnormalities. These clinical features, along with the presence of a family history of HCM and/or reduced maximum oxygen consumption capacity help differentiate hypertrophy related to HCM from that associated with athletic training. In some instances, a period of detraining may be needed with repeat echocardiography that typically shows regression of hypertrophy in the athlete's heart, which does not occur in patients with HCM.

A helpful technique for distinguishing HCM from the so-called "athletic heart" is echocardiography. In contrast to athletes, patients with HCM exhibit small LV cavity dimensions, diastolic (lower tissue Doppler velocity), and myocardial deformation (strain) abnormalities. These clinical characteristics aid in distinguishing between hypertrophy linked to HCM and that resulting from athletic training, as does the existence of a family history of HCM. Repeat echocardiogram, which usually demonstrates regression of hypertrophy in the athlete's heart after a period of detraining and this does not occur in patients with HCM.[46]

Speckle-tracking echocardiography and CMR are also useful for differentiation. Speckle tracking analyzes LV twisting and untwisting to distinguish AH from HCM. In HCM, CMR shows myocardial fibrosis, often mid-wall and in areas of maximum hypertrophy, serving as a key pathology indicator, whereas such fibrosis is generally absent in AH [47].

Arrhythmogenic Cardiomyopathy (ACM):

arrhythmogenic cardiomyopathy, is an inherited disease characterized by the progressive replacement of the normal myocardium by fibro-adipocytic tissue, mainly in the right ventricle, but in advanced phases also in the left ventricle and the atria . Although rare, affecting only 0.05% of the general population, ARVD/C is one of the biggest challenges for cardiologists due to the severe complications which can occur—ventricular arrhythmia, heart failure, and even sudden death [48].

RV size is a key diagnostic parameter for ACM but not AH, often leading to false suspicions. Echocardiography of elite athletes revealed that RV and right atrial dimensions are greater in endurance-trained athletes compared to age- and sex-matched strength-trained athletes and

sedentary controls. However, other typical findings of ACM, such as RV bulging, thinning, and aneurysms, are usually absent in healthy athletes [48].

CMR is the preferred method for evaluating the RV. It can detect RV enlargement and identify other pathological features of ACM, such as wall motion abnormalities, RV systolic dysfunction, RV fibrofatty replacement, and sub-epicardial and mid-wall LGE, indicating fibrosis [49].

Left Ventricular Non-Compaction (LVNC)

Left ventricular non-compaction (LVNC) is a cardiomyopathy characterized by increased trabeculation and deep intertrabecular recesses within the LV. It is associated with progressive LV dilatation, impaired systolic and diastolic function, life-threatening arrhythmias, and thromboembolic events [50].

marked hyper trabeculation was observed after a period of intensive training.

LV systolic and diastolic functions remained normal despite the increased trabeculation, suggesting that the hyper trabeculation was a benign adaptation rather than a pathological condition. Considering family history, symptoms is crucial when evaluating athletes for LVNC. [51].

Cardiopulmonary Exercise Testing (CPET)

Cardiopulmonary exercise testing (CPET) is an important secondary assessment tool in paediatrics not only to objectively assess overall functional exercise capacity but also to investigate for specific metabolic, respiratory or cardiac pathology such as pulmonary vascular disease, dysfunction of the autonomic nervous system and peripheral defects in oxygen transport and oxygen utilization at the working muscle. This makes it an important tool for risk stratification in children with congenital or acquired heart disease but also to exclude exercise pathology in paediatric athletes. CPET should always be combined with exercise 12-lead ECG testing to allow assessment of underlying exercise-related arrhythmias. It is paramount that supervising personnel should be trained and familiar with the paediatric population to encourage maximal effort and deliver reliable test results. CPET can also indirectly pick up reduced stroke volume caused by ventricular dysfunction in cardiomyopathies or congenital heart disease. [52,53]

Mobile Monitoring

Mobile monitoring is becoming a diagnostic tool of choice accounting for the athlete specific and chosen training environment, portability and prolonged monitoring during exercise with potentially higher pick up rates of pathology (e.g. arrhythmias). Cableless mobile and app-based devices are available for clinical use in children and early data on feasibility and diagnostic value are encouraging . Mobile monitoring in the paediatric athlete should be used to investigate cardiac symptoms during exercise such as paroxysmal symptoms of palpitations, dizziness and syncope, postintervention (e.g. arrhythmia ablation) monitoring and risk stratification in pathologies (e.g. cardiomyopathies, congenital heart disease).[54]

Complications: The extremes of exercise-induced cardiac remodeling are potentially associated with uncommon side effects. Atrial fibrillation is more common among endurance athletes and there is speculation that other arrhythmias may also be more prevalent. It is yet to be determined

whether this arrhythmic susceptibility is a result of extreme exercise remodeling, genetic predisposition, or other factors.[55]

What happen after detraining

Although there are a lot of observational and prospective intervention studies documenting increases in cardiac volumes and mass associated with habitual endurance exercise, there are relatively few addressing the extent of reverse remodeling associated with detraining. A recent narrative review by Petek et al[16] noted significant heterogeneity in the findings to date, but some important themes included the observation that LV remodeling appears to occur within weeks of forced training, although return to normal cardiac dimensions may not occur even after decades of detraining, and that right ventricular (RV) reverse remodeling may occur at a slower rate than for the LV. [56]

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