International Journal of Chemical and Biochemical Sciences
(ISSN 2226-9614)

Journal Home page: www.iscientific.org/Journal.html

Common Pitfalls in ECG Interpretation in Pediatric Athletes

Hemat Mostafa Elsayed Ibrahim, Wafaa Fathy ELsaeed, Saed Mohamed morsy, Alshaymaa Ahmed Ali

Pediatrics Department, Faculty of medicine, Zagazig University, Egypt

Abstract

A well-known phenomenon in adults who play competitive sports is the athlete's heart. Exercise training is associated with an array of morphologic and functional cardiac adaptations and termed the 'athlete's heart unfortunately, the majority of research on training-induced cardiac remodeling has been done on adults, and the present guidelines are primarily applied to adults. However, an appropriate interpretation of resting ECG and imaging in children practicing sports is crucial, it helps us in early detection of life-threatening conditions and managing therapy and eligibility to sports competitions in the rapidly growing pediatric athlete population. As training-induced remodeling can mimic potential cardiovascular problems, leading to possible misdiagnosis. This challenge is compounded by the physiological changes in young athletes' hearts, which can resemble pathological conditions. Therefore, a careful and systematic approach is necessary to differentiate between benign adaptations and serious conditions.

Keywords: Sports • Children • Cardiovascular screening • Electrocardiography • Athlete's heart

Review article **Corresponding Author*, e-mail: hematmostafa2010@gmail.com

1. Introduction

1.1. Athlete's Heart

Exercise training is associated with an array of morphologic and functional cardiac adaptations and termed the 'athlete's heart' (AH) [1]. This physiological process is characterized by a group of clinical, electrocardiographic, and echocardiographic changes resulting from prolonged, intense physical activity. The most relevant adaptations are myocardial hypertrophy, enlargement of cardiac cavities, and the increase in cardiac mass, which may have different intensities between various athletes. The degree of these changes varies according to the athlete's body surface area, gender, age, and the kind of sport they play. These changes increase the efficiency of cardiovascular function and can be reversed by stopping sports participation. The majority of the cardiovascular system's adaptations can be attributed to the Fick principle and the requirement for better delivery of oxygenated blood to the muscles, which results in a larger left ventricle, a thicker and more effective cardiac wall, and a lower heart rate [2]. In most cases, the values found remain within normal limits, however in extreme ways of adaptation the physiological process reaches borderline values with some similarities to certain pathological processes [3]. In adults, all of these changes have already been fully explained; however, in the case of the pediatric athlete, several details remain unexplained. Current research appears to link androgenic hormones to the thickening of the cardiac wall. The resting ECG of pediatric athletes seems not to predominantly reflect exercise-induced morphological remodeling only but also maturation, Incomplete Right Bundle Branch Block (RBBB), Repolarization Changes, and Bradycardia can be observed more commonly in pediatric athletes rather than in sedentary children. The hormonal theory also explains why there are differences in heart adaptation between male and female athletes. In short, the appearance of differences between-sex in LV parameters in adolescent athletes may not only reflect, as generally thought sex-related different responses of LV to training, but also the physiological age-dependent divergence in LV parameters in early adolescence. Furthermore, in the 12–14 years age interval, sex-related differences in body size are much less pronounced than in 16-year-old people, thus limiting specific influence of anthropometric characteristics on LV [4].

2. Pre-participation evaluation

Pre-participation evaluation (PPE) is conducted in sports to identify underlying cardiovascular diseases and assess the risk of sudden cardiac death (SCD) in athletes, enabling individualized risk management strategies and enhancing athlete safety during participation in physical activities. The leading cause of SCD is highly dependent on age. Structural cardiac abnormalities are the most common cause of (SCD) in young adults, with hypertrophic cardiomyopathy (HCM) accounting for 36% of the relative incidence in the US. And in Europe, arrhythmogenic cardiomyopathy (ACM). On the other hand, congenital preexcitation, channelopathies, especially long QT syndrome (LQTS), and the abnormal origin of the coronary artery are the most frequent causes in children and adolescents. Myocarditis, Marfan syndrome, and, less frequently and mainly in athletes, commotio cordis—a condition in which a sudden hard hit to the chest results in sudden death without

cardiac damage—are additional causes of SCD [5-6]. SCDs more frequently occur during exercise. The increased adrenergic stimulation during intensive training and competition may potentially trigger ventricular arrhythmias. Furthermore, exercise can accelerate the progression of certain cardiac diseases, such as ACM. However, SCD may also occur at rest, particularly in channelopathies, such as Brugada syndrome and LQTS [7].

3. Role of electrocardiography

Many medical societies and sports governing bodies recommend PPCS consisting of a focused history and physical examination (H&P) and 12-lead electrocardiogram (ECG). The resting ECG has demonstrated its usefulness in diagnosing HCM than history and physical examination alone with a 90% specificity and a 77% greater power. Additionally, the sensitivity of the pre-participation evaluation (PPE) was enhanced by resting ECG, not only for the detection of HCM but also for other structural heart diseases such ACM that are accompanied by abnormal ECG readings. Additionally, ECG help the detection of LQTS, Brugada syndrome, Wolf-Parkinson-White (WPW), and other cardiomyopathies such as dilated cardiomyopathy and left ventricular non-compaction, so accurate ECG interpretation can assist in prevention of SCD [8]. Although ECG is crucial in pre-participation evaluation, it was criticized for high false-positive rates that led to substantial costs associated with secondary testing and unnecessary (temporary) restriction of athletes from participation. This led to substantial efforts by the scientific community to better understand difference b/w physiologic and pathologic ECG findings in athletes. The 2010 ESC (European society of cardiology) criteria were the first to divide ECG findings into two groups—common and training-related (group 1) versus uncommon and training-unrelated (group 2)—based on the prevalence of ECG findings, relation to exercise training, and association with pathological conditions associated with SCD requiring further clinical investigation to confirm (or exclude) underlying cardiovascular disease. Since then, other investigators and international panels have proposed updated guidelines for ECG interpretation in young athletes. Each revision of the ECG standards has improved specificity while maintaining the sensitivity for ECG-detectable pathological conditions associated with SCD [9].

4. Differences between pediatric and adult cut off values in ECG

Resting heart rate (HR) decreases with aging and is normally higher in children than adults; accordingly, profound sinus bradycardia, arbitrarily defined as a resting HR below 30 b.p.m. for adult athletes, has been set at 40 b.p.m. Under the age of 14 years, although the presence of I degree AV block in athletes has been deemed normal, an excessive prolongation should prompt further investigation. The cut-off for defining an excessive prolongation has been arbitrarily set at 400 msec in adult athletes. The PR interval in the pediatric population is shorter than in adults accordingly, the cut-off has been moved from 400 to 300 msec. Similarly, the QRS duration requiring further investigation has been moved from 140 to 130 msec, given that the QRS widens with the growth and, in the population of children between 4 and 16 years of age, a normal QRS should be around 100 msec. The definition of normal and abnormal ECG findings adapted to the pediatric population are reported in (Table 1). The QTc interval definition is reported. Current recommendations state that the Bazett formula should be used to adjust the QT interval. Although the Bazett and Fridericia correction formulae are not interchangeable, we recommend correcting the QT interval in borderline instances using both of them. Additionally, the Fridericia correction should be chosen in preadolescents with a resting heart rate of ≥82 b.p.m. In young athletes, low QRS voltages tend to be infrequent and usually reflect underlying cardiomyopathy, they were included for the first time among the abnormal findings [10]. ECG criteria for RVH was in high-dynamic sport athletes (Figs 1-4). Endurance sports are associated with an enlarged right ventricle. This is most likely secondary to a volume overload. Nevertheless, there is growing concern about the new concept of right ventricular fatigue in some athletes who develop a phenotype corresponding to the arrhythmogenic right ventricular the cardiomyopathy in the absence of genetic susceptibility. Importantly, this idea of a slowly progressing structural illness need years of training to appear and shouldn't manifest in young patients. It is important to remember that the heart develops until adolescence and that the right ventricle serves as the major chamber during fetal development. As a result, children typically exhibit taller R waves in V1 and relative right ventricular enlargement more than adults [11].

5. Normal ECG findings in pediatric athletes

5.1. T-wave inversion

The initial and may be the only sign of early cardiomyopathy is TWI. Because of this, it is critical to differentiate between a benign juvenile pattern and a potential cardiomyopathy that needs more investigation. TWI is prevalent among children, as opposed to adults, and is particularly prevalent in young Black and Arabic athletes. Between the ages of 5 and 11, up to 35% of children have anterior TWI (from V1 to V3); as children become older, the frequency declines. And is correlated with pubertal development and age. Consequently, anterior TWI in children under 16 who are asymptomatic and do not have a family history of SCD or cardiomyopathies does not typically indicate underlying structural heart disease and should not lead to additional testing [12-13].

5.2. Sinus arrhythmia

The heart rate usually increases slightly during inspiration and decreases a little bit during expiration. In youth and well-trained athletes, this reaction, known as sinus arrhythmia, can be greatly exaggerated, leading to an irregular heartbeat that starts in the sinus node. Up to 55% of skilled sportsmen are thought to suffer from sinus arrhythmia. Sick sinus syndrome, or sinus node dysfunction is a more serious condition where the heart rate is abnormally slow, fast, or irregular. Lack of rhythmic heart rate fluctuations, sudden, prolonged rate increases and decreases, an inappropriate rate response to exercise (both a slowed acceleration and an inappropriately rapid deceleration), and any association with clinical symptoms like exercise intolerance, presyncope, and syncope are distinguishing characteristics that point to sinus node dysfunction. In sinus arrhythmia, the P wave axis stays normal even though the heart beat is highly erratic [14].

5.3. Incomplete right bundle branch block

IRBBB is defined by a QRS duration <100 ms with 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, especially those participating in mixed athletic disciplines that incorporate both anaerobic and aerobic elements, as well as endurance training. It has been proposed that rather than an intrinsic delay in the His-Purkinje system itself, the slightly delayed RV conduction is the result of RV remodeling, which increases cavity size and hence conduction time. Further assessment is not necessary when IRBBB occurs in an asymptomatic athlete with a negative physical examination and family history. Because IRBBB can be an associated ECG finding in patients with an atrial septal defect, special attention should be paid to the auscultation of a fixed splitting of the second heart sound during physical examination. Patients with arrhythmogenic RV cardiomyopathy (ARVC) may exhibit IRBBB. Nonetheless, in ARVC, the IRBBB pattern is typically linked to additional ECG abnormalities, including low limb-lead voltages, prolonged S wave upstroke, premature ventricular beats with a left bundle branch block (LBBB) morphology, and T wave inversion involving the mid-precordial leads beyond V2 [15].

5.4. Early repolarization

Early repolarization is an ECG pattern consisting of ST elevation and/or a J wave (distinct notch) or slur on the downslope of the R wave. Traditional examples of early repolarization referred to ST elevation, but the newer definitions also include the J waves or terminal the QRS slurring [16].

5.5. QRS voltage criteria for LVH

LVH may not be accurately predicted by the ECG QRS voltage. Because the ECG relies on electrodes on the body's surface to measure the electrical activity of the heart, it is limited in its ability to detect ventricular hypertrophy. The voltage will therefore be impacted by anything that is in between the surface electrodes and the left ventricular myocardium. Therefore, LV size or mass is not the only factor that might affect ECG QRS voltage. Obesity, advanced age, and pulmonary conditions may result in lower QRS voltage, whereas males, athletes, and Black or African people have higher voltage. Intense training in athletes is also linked to morphological cardiac alterations, such as thicker walls and larger cavities, which are seen on the ECG. In trained athletes, these alterations make up physiological LVH, which typically manifest as a discrete rise in QRS amplitude. Up to 45% of athletes and 25% of young adults who are sedentary have ECGs with elevated QRS amplitudes that fulfil the ECG voltage criterion for LVH. Because of this, elevated QRS voltage is not a reliable diagnostic indicator of pathological LVH [17]. Pathological LVH is commonly associated with additional ECG features such as T-wave inversion (TWI) in the inferior and lateral leads, ST-segment depression, and pathological Q waves. Therefore, the isolated presence of high QRS voltages fulfilling voltage criterion for LVH in the absence of other ECG or clinical markers suggestive of pathology are considered part of normal and training-related ECG changes in athletes and does not require further evaluation [18].

6. Abnormal Electrocardiographic Findings in Athletes

6.1. Abnormal T wave inversion

TWI ≥1 mm in depth in two or more contiguous leads (excluding leads aVR, III and V1) in an anterior, lateral, inferolateral or inferior territory is abnormal (with exception of TWI confined to leads V1-V4 in black athletes and leads V1-V3 in all athletes aged less than 16 years. TWI affecting lateral leads (V5-V6, I & aVL) should prompt comprehensive investigation irrespective of ethnicity, including cardiac MRI, echocardiography is non-diagnostic. Echocardiographic quality is variable and may have limited ability to detect hypertrophy of anterolateral LV wall and apex. Quality of echocardiogramvaries and may not be able to identify antero lateral LV wall and apex hypertrophy.Better assessment o f myocardial hypertrophy is possible using contrast-enhanced cardiac MRI, which can also show late gadolinium enhancement, a non-specific indicator of myocardial fibrosis. In most non-black athletes' age ≥16 years, anterior TWI beyond lead V2 should prompt further evaluation given the potential overlap with ARVC. In athletes age ≥16 years with TWI beyond V2, concurrent findings of J-point elevation, ST segment elevation more likely represent athlete's heart, while the absence of J-point elevation or a coexistent depressed ST segment is more concerning for ARVC. Other ECG findings suggestive of ARVC in the presence of anterior TWI include low limb lead voltages, prolonged S wave [19-20].

6.2. ST segment depression

While ST segment depression is common among patients with cardiomyopathy, it is not a feature of athletic heart. ST segment depression (relative to the isoelectric PR segment) in excess of 0.05 mV (0.5 mm) in two or more leads should be considered an abnormal finding requiring definitive evaluation for underlying structural heart disease. Echocardiography is the minimum evaluation for athletes with ST segment depression to investigate for underlying cardiomyopathy. Cardiac MRI should be considered based on the echocardiographic findings or clinical suspicion [21].

6.3. Pathological Q waves

Q waves are defined as any initial negative deflection of the QRS complex and can be found with both physiological electrical activation of the ventricle and with certain pathological conditions, including cardiomyopathy, myocardial infarction and conduction abnormalities. Pathological Q waves were previously defined as >3 mm in depth or >40 ms in duration in two or more leads (except III and aVR). Several pathological disorders can lead to the development of exaggerated (deep or wide) Q waves or unexpected Q waves in atypical leads. HCM commonly results in asymmetric septal hypertrophy which can produce abnormal Q waves due to increased septal forces, septal fibrosis and asymmetric electrical activation. If there is clinical abnormality along with pathological Q wave present with other findings such as ST segment depression or TWI, then reevaluation by cardiac MRI is considered [22].

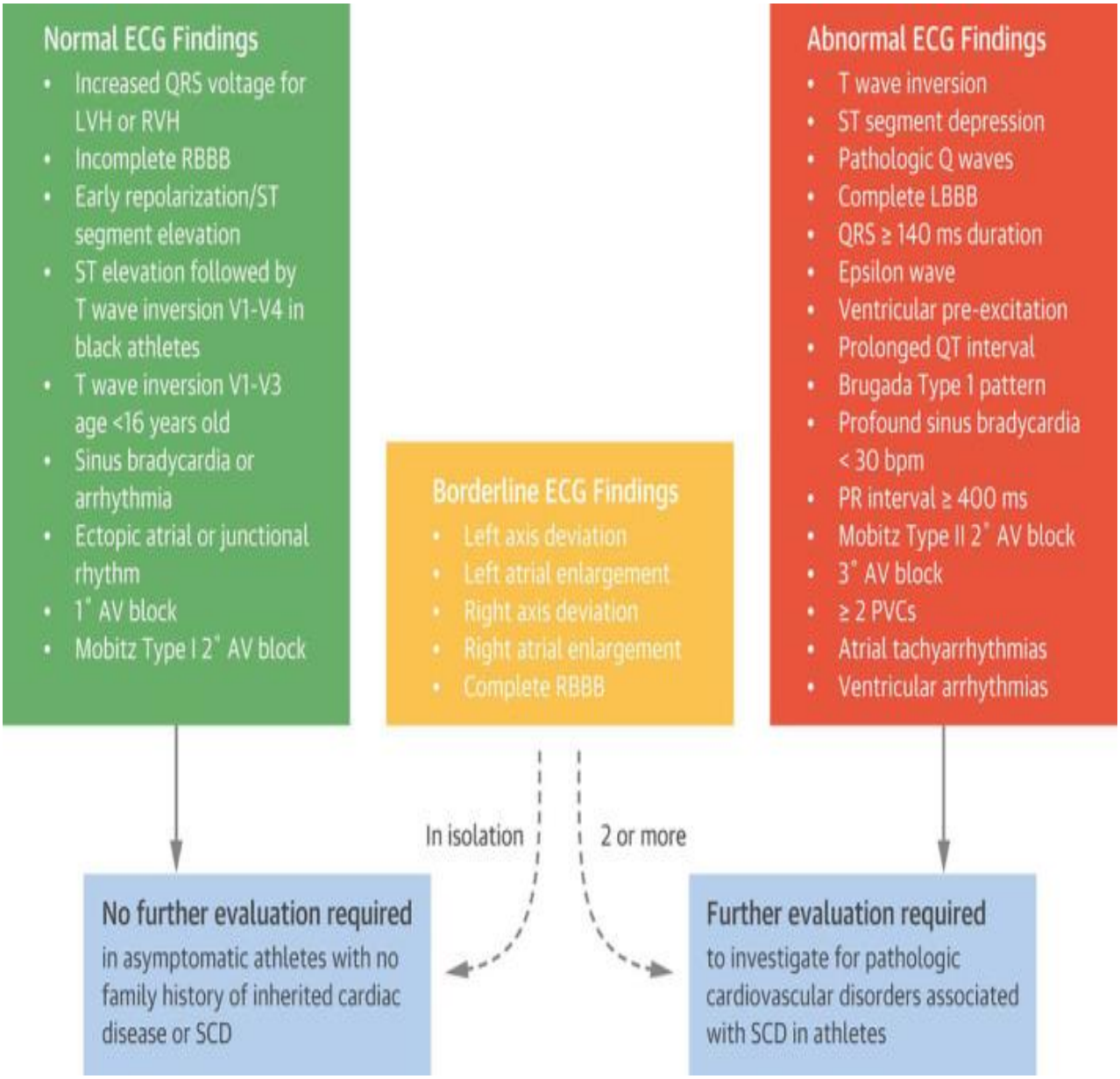


Figure 1. Proposal of revision of the international criteria for their application to pediatric athletes [9]. AV, atrioventricular; LBBB, left bundle branch block; LVH, left ventricular hypertrophy; PVC, premature ventricular contraction; RBBB, right bundle branch block; RVH, right ventricular hypertrophy; SCD, sudden cardiac death.

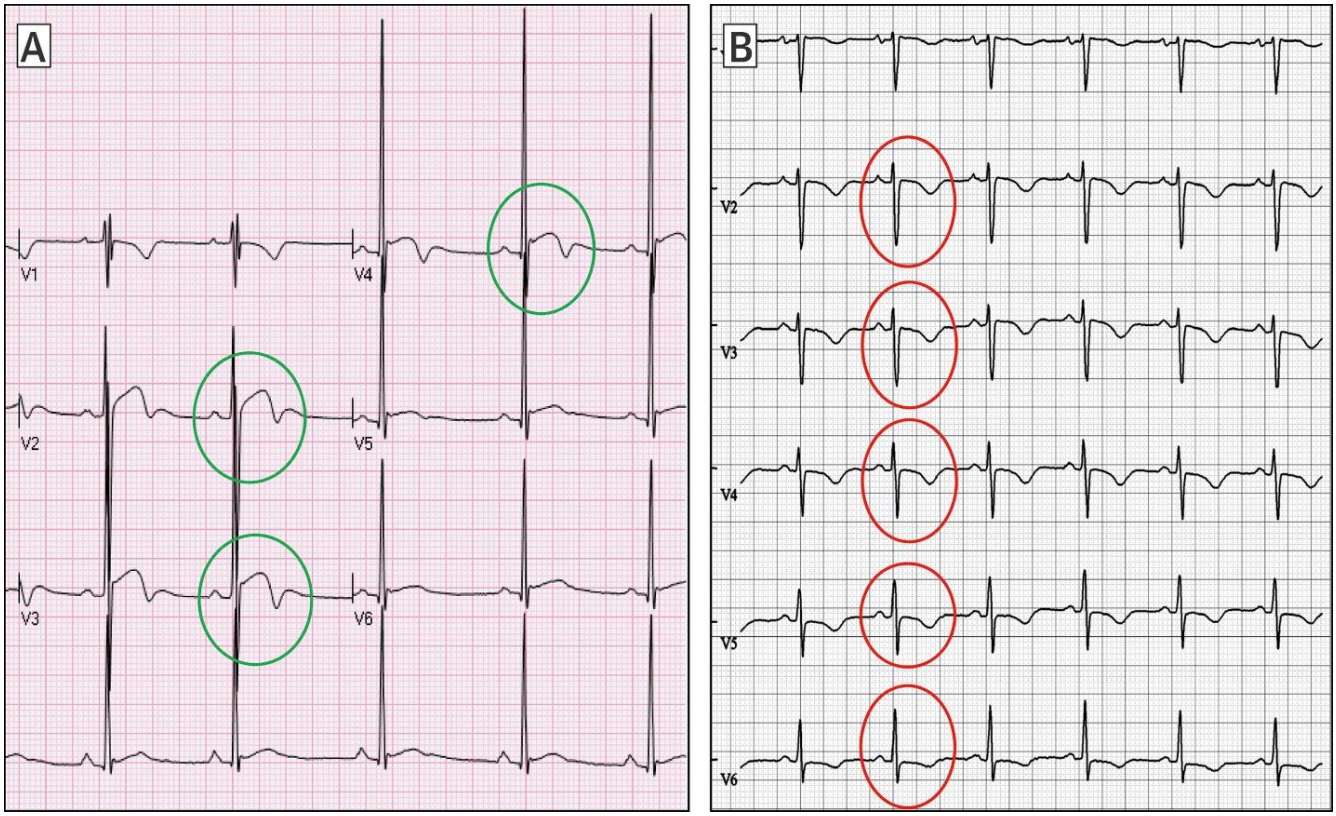
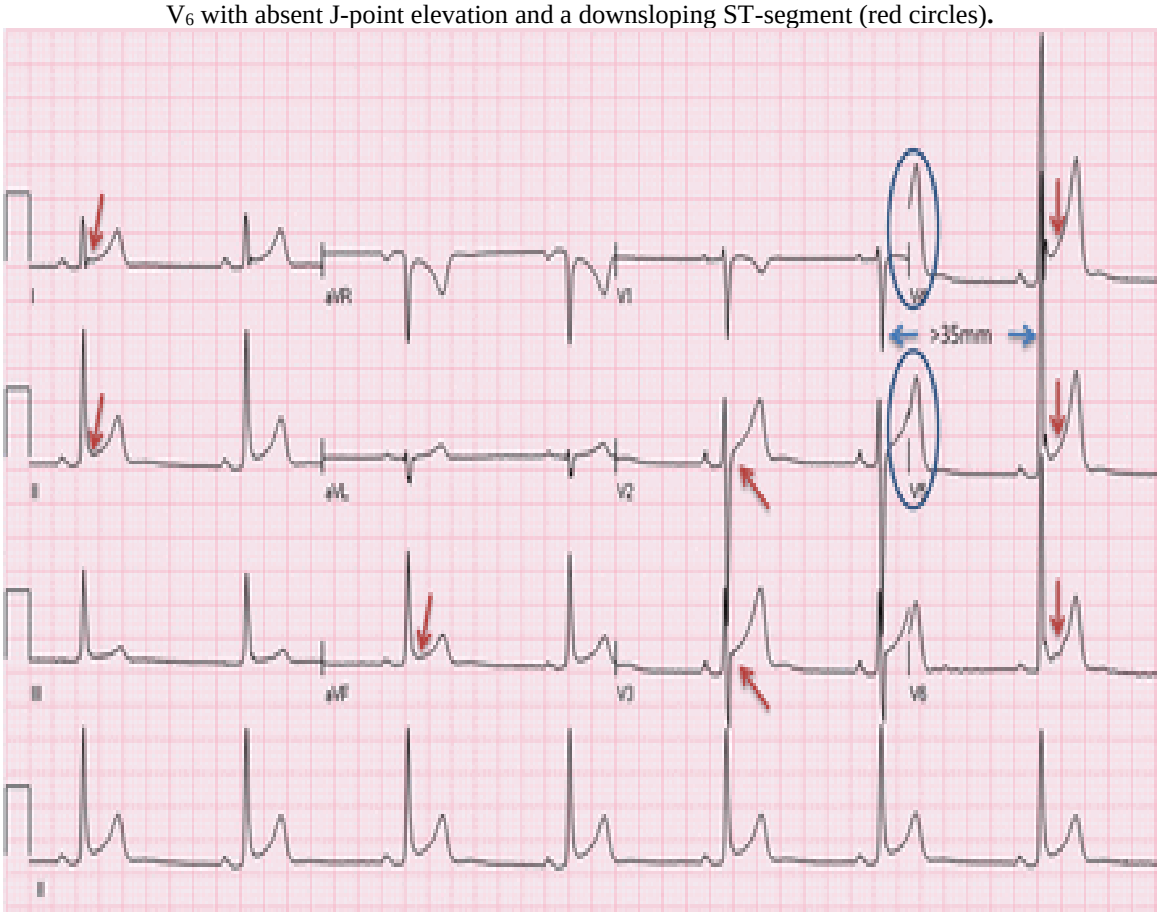


Figure 2. (A) T-wave inversion in V_1 to V_4 preceded by J-point elevation and convex 'domed' ST-segment elevation (green circles)**.** This should not be confused with pathological T-wave inversion (B) which demonstrates T-wave inversion in V_1 to V_6 with absent J-point elevation and a downsloping ST-segment (red circles)**.**



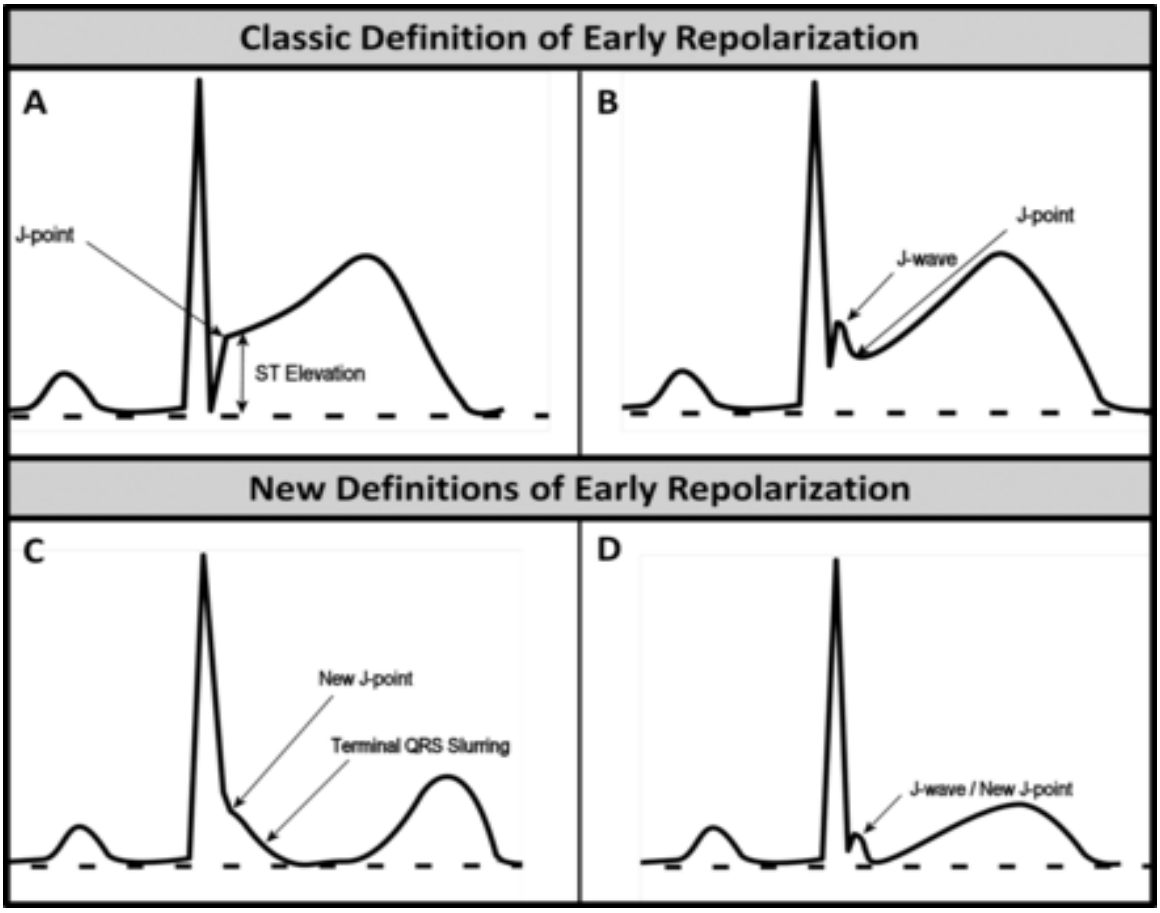


Figure 3. (A and B) Classic definition of early repolarization based on ST elevation at QRS end (J-point). Examples without (A) and with (B) a J wave. (C and D) New definitions of early repolarization showing slurred QRS downstroke (C) and J-wave (D) without ST elevation.

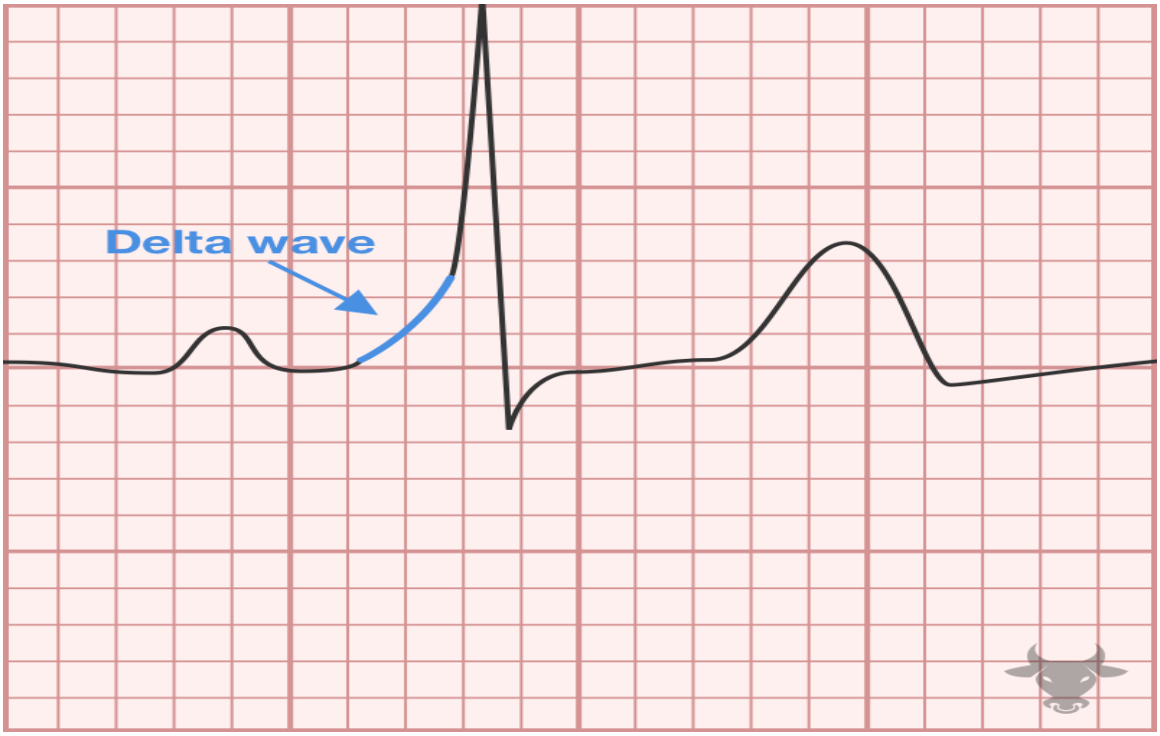


Figure 4. Delta wave pattern of WPW syndrome

Table (1): Important definitions when interpreting resting electrocardiogram in junior athletes

ECG	Definition
Normal ECG findings	
Juvenile T-wave pattern	T-wave inversion V_1–V_3 in athletes <16 years
I degree AV block	PR interval longer than 200 msec but shorter than 300 msec
Increased QRS voltage	Isolated QRS voltage criteria for left (SV_1 + RV_5 or RV_6 > 3 mV<u>a</u>) or right ventricular hypertrophy (RV_1 + SV_5 or SV_6 > 0.8 mV)
Borderline ECG findings	
Incomplete RBBB	rSR′ pattern in lead V_1 and a qRS pattern in lead V_6 with QRS duration <100 ms
Left axis deviation	−9° to −90°
Right axis deviation	>110°
Abnormal ECG findings	
Short PR interval	PR interval ≤90 ms
Complete RBBB	rSR′ pattern in lead V_1 and an S wave wider than R wave in lead V_6 with QRS duration ≥100 ms
Complete LBBB	QRS ≥100 ms, predominantly negative QRS complex in lead V_1 (QS or rS) and upright notched or slurred R wave in leads I and V_6
Prolonged QT interval	QTc ≥470 msec in high-level junior athletes
	QTc ≥460 msec
Profound sinus bradycardia	<40 beats per minute or sinus pauses ≥3 s
Ventricular pre-excitation	PR interval ≤90 msec with a delta wave (slurred upstroke in the QRS complex) and wide QRS (≥90 ms)
Profound, non-specific intraventricular conduction delay	Any QRS duration ≥130 ms
Profound 1° AV block	PR interval ≥300 ms
T-wave inversion	≥1 mm in depth in two or more contiguous leads, excluding leads aVR, III, and V_1
– Anterior	V_2–V_4
	– excludes: black athletes with J-point elevation and convex ST segment elevation followed by TWI in V_2–V_4; athletes < age 16 with TWI in V_1–V_3; and biphasic T waves in only V_3
– Lateral	I and aVL, V_5 and/or V_6 (only one lead of TWI required in V_5 or V_6)
– Inferolateral	II and aVF, V_5–V_6, I and Avl
– Inferior	II and aVF

AV, atrioventricular; LBBB, left bundle branch block; RBBB, right bundle branch block; TWI, T-wave inversion.Reference values may differ according to sex [12].

6.4. Complete LBBB

LBBB is found in less than 1 in 1000 athletes but is common in patients with cardiomyopathy and ischemic heart disease. Thus, complete LBBB always should be considered an abnormal finding and requires a comprehensive evaluation to rule out a pathological cardiac disorder. LBBB is recognized by a QRS ≥120 ms, predominantly negative QRS complex in lead V1 (QS or rS), and upright notched or slurred R wave in leads I and V6. Evaluation Athletes with complete LBBB require a thorough investigation for myocardial disease including echocardiography and a cardiac MRI [23].

6.5. Prolonged QT interval

The hallmark ECG feature of congenital LQTS is QT prolongation, this group of potentially lethal, genetically mediated ventricular arrhythmia. Symptoms if present include arrhythmic syncope, seizures or aborted cardiac arrest/sudden death stemming from torsade de pointes and VF. Pathophysiology of LQTS involves delayed ventricular repolarization originating primarily from loss-of-function mutations in genes encoding voltage gated potassium channels that govern phase 3 repolarization. LQTS is estimated to affect 1 in 2000 individuals, and this may be underestimated given the subpopulation of so-called 'normal QT interval' or 'concealed' LQTS, in which this prolongation is hidden, so correction formula should be used to confirm the calculated Q T interval by computer. Bazett's formula for calculating QT interval (QTc = QT/√RR; provided RR interval is measured in seconds) loses accuracy at slow and fast heart rates; underestimating the inherent QTc at heart rates 90bpm. Another formula to be applied for QTC interval calculation is Fridericia formula QT =QT/ (RR0.33).it is used in addition to Bazett's formula in doubtful cases. Genetic testing for LQTS is recommended for any athlete where there is high index of suspicion for the LQTS, or for an asymptomatic patient with no family history but an incidental the finding with a QTc interval >480ms pre-puberty and >500ms post-puberty that is confirmed on repeat the ECG testing [24-25].

6.6. Ventricular preexcitation

This condition occurs when there is an accessory pathway bypassing AV node resulting in preexcitation of the ventricle, this result in short PR interval and wide QRS complex, a pattern called Wolf Parkinson White in PR<120ms and QRS >120ms and there is delta wave created from early ventricular depolarization. Approximately 1/1000 to 4/1000 athletes exhibit the WPW pattern. The presence of an accessory pathway can predispose an athlete to sudden death because rapid conduction of atrial fibrillation across the accessory pathway can result in VF. Asymptomatic Athletes with WPW pattern should be investigated for the presence of a low-risk or high-risk accessory pathway. Exercise stress test represents the primary non-invasive risk stratification test in which abrupt, complete loss of pre-excitation at higher heart rates suggests a low-risk accessory pathway. If non-invasive testing cannot confirm a low-risk pathway or is inconclusive, electrophysiology testing should be considered to determine the shortest pre-excited RR interval during atrial fibrillation. If the shortest pre-excited RR interval is ≤250 ms (240 bpm), then the accessory pathway is deemed high risk. Young athletes with a shortest pre-excited RR interval of ≤250 ms should proceed with transcatheter pathway ablation. As there is association between WPW and Ebestien anomaly and cardiomyopathy, Echocardiography should be taken in consideration [26].

6.7. Ventricular arrhythmias

If Ventricular couplets, triplets and non-sustained ventricular tachycardia present alone, these are not life-threatening arrhythmias, but can be a marker for underlying cardiac pathology or lead to sustained ventricular tachycardia which may cause SCD. These ventricular arrhythmias may be idiopathic or secondary to an underlying cardiac pathology like cardiomyopathies, ion channelopathies or other diseases such as myocardial infarction. The evaluation of such condition should include a thorough family history, an echocardiogram to evaluate for structural heart disease, cardiac MRI to assess for ARVC or other cardiomyopathies ambulatory ECG monitor and exercise ECG test. Depending on these results, further evaluation may be needed including electrophysiology study or genetic testing [27].

7. Conclusions

Knowledge of the physiological cardiac adaptations in athletes that occur on the ECG is essential to the cardiovascular care of athletes. When appropriately interpreted, the ECG can yield useful information by taking into consideration the structural and electrical alterations that frequently occur as a result of regular exercise. The identification of athletes at risk for (SCD) depends on the ability to distinguish ECG findings related to the athlete's heart from alterations that may indicate an underlying pathological condition. Doubtful conditions should be further assessed and correlations to clinical data and further investigation is required.

References

[1] A. Pelliccia, S. Caselli, S. Sharma, C. Basso, J.J. Bax, D. Corrado, A. D'Andrea, F. D'Ascenzi, F.M. Di Paolo, T. Edvardsen. (2018). European Association of Preventive Cardiology (EAPC) and European Association of Cardiovascular Imaging (EACVI) joint position statement: recommendations for the indication and interpretation of cardiovascular imaging in the evaluation of the athlete's heart. European Heart Journal. 39(21): 1949-1969.

[2] B.D. Levine. (2008). : what do we know, and what do we still need to know? The Journal of physiology. 586(1): 25-34.

[3] N. Ghorayeb. (1995). Coração do Atleta– Modificações fisiológicas X supertreinamento e doenças cardíacas. Arq Bras Cardiol. 64: 161-165.

[4] B.G. Winkel, B. Risgaard, G. Sadjadieh, H. Bundgaard, S. Haunsø, J. Tfelt-Hansen. (2014). Sudden cardiac death in children (1–18 years): symptoms and causes of death in a nationwide setting. European Heart Journal. 35(13): 868-875.

[5] D. Corrado, C. Basso, A. Pavei, P. Michieli, M. Schiavon, G. Thiene. (2006). Trends in sudden cardiovascular death in young competitive athletes after implementation of a preparticipation screening program. Jama. 296: 1593-1601.

[6] S.S. Chugh, K. Reinier, S. Balaji, A. Uy-Evanado, C. Vickers, R. Mariani, K. Gunson, J. Jui. (2009). Population-based analysis of sudden death in children: The Oregon Sudden Unexpected Death Study. Heart Rhythm. 6: 1618-1622.

[7] I.M. Asif, K.G. Harmon. (2017). Incidence and etiology of sudden cardiac death: new updates for athletic departments. Sports Health. 9: 268-279.

[8] I.M. Asif, J.A. Drezner. (2012). Sudden cardiac death and preparticipation screening: the debate continues—in support of electrocardiogram-inclusive preparticipation screening. Progress in cardiovascular diseases. 54: 445-450.

[9] M. Ackerman, D.L. Atkins, J.K. Triedman. (2016). Sudden cardiac death in the young. Circulation. 21: 1566-1574.

[10] L. Diaz-Gonzalez, V. Bruna, P.L. Valenzuela, J. Velásquez-Rodriguez, A. Boraita, A. Lucia, M. Martinez-Sellés. (2021). Sinus bradycardia in paediatric athletes. European Journal of Preventive Cardiology. 28(10): 1142-1144.

[11] M. Albiński, M. Saubade, C. Benaim, A. Menafoglio, P. Meyer, B. Capelli, T. Perrin, L. Trachsel, D. Hagemeyer, D. Casagrande. (2021). Impact of early sports specialisation on paediatric ECG. Scandinavian journal of medicine & science in sports. 31: 1335-1341.

[12] G.L. Ragazzoni, L. Cavigli, E. Cavarretta, S. Maffei, G.E. Mandoli, M.C. Pastore, S. Valente, M. Focardi, M. Cameli, G. Di Salvo. (2023). How to evaluate resting ECG and imaging in children practising sport: a critical review and proposal of an algorithm for ECG interpretation. European Journal of Preventive Cardiology. 30(5): 375-383.

[13] G. McClean, N.R. Riding, C.L. Ardern, A. Farooq, G.E. Pieles, V. Watt, C. Adamuz, K.P. George, D. Oxborough, M.G. Wilson. (2018). Electrical and structural adaptations of the paediatric athlete's heart: a systematic review with meta-analysis. British journal of sports medicine. 52: 230.

[14] M. Papadakis, S. Basavarajaiah, J. Rawlins, C. Edwards, J. Makan, S. Firoozi, L. Carby, S. Sharma. (2009). Prevalence and significance of T-wave inversions in predominantly Caucasian adolescent athletes. European Heart Journal. 30: 1728-35.

[15] J.-B. Langdeau, L. Blier, H. Turcotte, G. O'Hara, L.-P. Boulet. (2001). Electrocardiographic findings in athletes: the prevalence of left ventricular hypertrophy and conduction defects. The Canadian journal of cardiology. 17: 655-9.

[16] M.V. Perez, K. Friday, V. Froelicher. (2012). Semantic confusion: the case of early repolarization and the J point. The American journal of medicine. 125: 843-4.

[17] E.W. Hancock, B.J. Deal, D.M. Mirvis, P. Okin, P. Kligfield, L.S. Gettes. (2009). AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram: part V: electrocardiogram changes associated with cardiac chamber hypertrophy: a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society: endorsed by the International Society for Computerized Electrocardiology. Circulation. 119(10): e251-61.

[18] N.K. Lakdawala, J.J. Thune, B.J. Maron, A.L. Cirino, O. Havndrup, H. Bundgaard, M. Christiansen, C.M. Carlsen, J.-F. Dorval, R.Y. Kwong. (2011). Electrocardiographic features of sarcomere mutation carriers with and without clinically overt hypertrophic cardiomyopathy. The American journal of cardiology. 108: 1606.

[19] P.M. Elliott, A. Anastasakis, M.A. Borger, M. Borggrefe, F. Cecchi, P. Charron, A.A. Hagege, A. Lafont, G. Limongelli, H. Mahrholdt. (2014). 2014 ESC Guidelines on diagnosis and management of hypertrophic cardiomyopathy. Polish Heart Journal (Kardiologia Polska). 35: 2733-79 84.

[20] C. Calore, A. Zorzi, N. Sheikh, A. Nese, M. Facci, A. Malhotra, A. Zaidi, M. Schiavon, A. Pelliccia, S. Sharma. (2016). Electrocardiographic anterior T-wave inversion in athletes of different ethnicities: differential diagnosis between athlete's heart and cardiomyopathy. European Heart Journal. 37: 2515-7.

[21] R.E. Bent, M.T. Wheeler, D. Hadley, J.W. Knowles, A. Pavlovic, G. Finocchiaro, F. Haddad, H. Salisbury, S. Race, Y. Shmargad. (2015). Systematic comparison of digital electrocardiograms from healthy athletes and patients with hypertrophic cardiomyopathy. Journal of the American College of Cardiology. 65: 2462-3.

[22] R.E. Bent, M.T. Wheeler, D. Hadley, V. Froelicher, E. Ashley, M.V. Perez. (2015). Computerized Q wave dimensions in athletes and hypertrophic cardiomyopathy patients. Journal of electrocardiology. 48(3): 362-7.

[23] J.H. Kim, A.L. Baggish. (2015). Electrocardiographic right and left bundle branch block patterns in athletes: prevalence, pathology, and clinical significance. Journal of electrocardiology. 48(3): 380-4.

[24] P.M. Rautaharju, B. Surawicz, L.S. Gettes. (2009). AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram: part IV: the ST segment, T and U waves, and the QT interval: a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society: endorsed by the International Society for Computerized Electrocardiology. Circulation. 2009;119:e241–50. 122

[25] B.J. Maron, D.P. Zipes. (2005). 36th Bethesda Conference: eligibility recommendations for competitive athletes with cardiovascular abnormalities. J Am Coll Cardiol. 45:1312-77..

[26] M. Cohen, J. Triedman, B. Cannon, A. Davis, F. Drago, J. Janousek, G. Klein, I. Law, F. Morady, T. Paul. (2012). Pediatric and Congenital Electrophysiology Society (PACES). Heart Rhythm Society (HRS). 9(6): 1006-24.1.

[27] L. Meyer, B. Stubbs, C. Fahrenbruch, C. Maeda, K. Harmon, M. Eisenberg, J. Drezner. (2012). Incidence, causes, and survival trends from cardiovascular-related sudden cardiac arrest in children and young adults 0 to 35 years of age: a 30-year review. Circulation. 126(11): 1363-72.