International Journal of Research
Available at https://edupediapublications.org/journals
p-ISSN: 2348-6848
e-ISSN: 2348-795X
Volume 02 Issue 03
March 2015

Electrocardiographic Screening and Sports

A Research Paper

By

Sat Pal,

Assistant Professor, Physical Education,

R.G. Govt. College, Saha, Ambala (Haryana)

Introduction:

In this age of competition, Billions of Dollars are being investing on scientific training and built up the sports infrastructure throughout the World. But, here, an extremely important aspect, 'Electrocardiographic screening and sports' is ignoring. When we are talking about to achieve the peak performance in healthy environment then we should not ignore the significance of Electrocardiographic screening in sports.

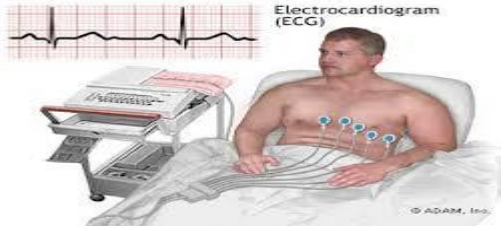
Sports cardiology of the European society of cardiology recommend that a 12-Lead ECG should be a part of the pre-participation screening of young competitive Athletes for prevention of so many cardiac deseases.

The International Olympic Committee has also recommended the routine ECG screening for the sportsmen before the sports activities. Abnormalities, however, may be detected by the utilization of the Electrocardiographic screening. The 12-Lead ECG is an established tool in the evaluation of Athletes Heart.

Although, the physicians are frequently asked to interpret an ECG in the setting of cardiovascular evaluation of the Athletes. But, most of the Athletes , due to the lack of knowledge, lack of facilities are not going for the Electrocardiographic screening before participation.In 1997, a mendatory pre-participation screening program was implemented in Israel. Similarly , there are so many countries where the Electrocardiographic screening is mendatory before going to the sports competition.

What is Electrocardiogram (ECG)

ECG or EKG , is taken from Greek word Kardia which meaning the" Heart". Its a transthoracic interpretation of the electrical activity of Heart over a period of time , as detected by electrodes attached to the surface of the skin and recorded by a device external to the body. The recording produced by this noninvasive procedure is termed an Electrocardiogram.It records the electrical activities of the Heart showing certain Waves called P,Q,R,S,T,U including showing the J point. All the Waves are associated with the depolarization and Repolarization of upper and lower chambers of Heart.

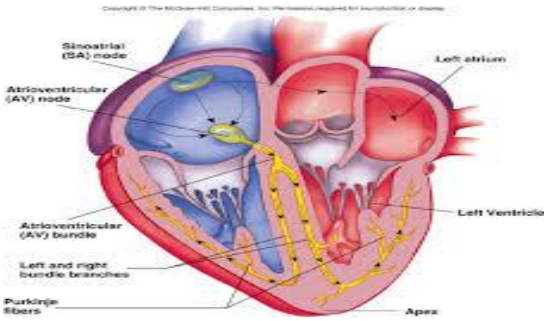


The ECG graph paper:

The output of an ECG recorder is a graph with time represented on the x-axis and voltage represent on y-axis. A dedicated ECG machine would usually print into graph paper which has a background pattern of 1mm square with bold divison every 5mm in both vertical and horizontal direction.

Electrical conduction system of Heart

The normal electrical conduction in the Heart allows the impulses that is generated by the Sinoatrialnode (SA node) of the Heart to be propagated to the cardiac muscle (myocardium) , the myocardium contract after stimulation. It is ordered stimulation of the myocardium that further allows efficient contraction of the Heart, thereby allowing blood to be pumped through the body.



Signals arising in the SA node which is located at the top of the right Atrium that is also supported by the Autonomus nervous system that stimulate the Atria to contract and travel to the AV node which is located in the inter-Atrial septum. After a delay, the stimulus diverges and is conducted through the right and left Bundle of Hiss to the respective Purkinje Fibers for each side of the Heart , as well as to the endocardium at the apex of the Heart, then finally to the Ventricle epicardium

Review of related literature:

Most of the sports related ECG research has been conducted on various fields of sports. P wave dispersion is an independent predictor of Atrial fibrillation. P wave dispersion is associated in inhomogeneous and discontinuous propagation of sinus impulses. DilaverisPE , designed a study to assess the P wave dispersion and duration and in this study the P wave dispersion and duration was assessed in the elite basketball players. The study showed that P wave dispersion have been thought to reflect left Atrial enlargement and altered conduction.

Maximum wave dispersion and duration was increased in the elite basketball players. The Heart rate was also decreased in the players. A study showed the types and frequency of ECG abnormalities in a sample of football players in USA. Resting and exercised 12-Lead ECG recording were analyzed. In this study , frequency of various ECG abnormalities findings in all athletes were: left ventricle hypertrophy=64.5%, interventricular conduction delay=2.6%, sinus bradycardia=9.1%, sinus arrhythmia=15.6%,first degree Atrioventricular block=11.7%, left Atrial enlargement=48.10%, early Repolarization=33.8%. these abnormalities are more frequent in african-american athletes.(2-7).

The evaluation and spectrum of Electrocardiographic changes in 1000 elite athletes, in this study as a result, the athletes had a significantly higher prevalence of sinus bradycardia and sinus arrhythmia. The RR interval , QRS complex and QT duration were prolonged in athletes. ST segment elevation was common in athletes.(8-10) the study

showed the cardiac abnormalities. In this way, so many studies are there which showed the cardiac abnormalities concerned the various kind of sports activities.

Significance of the study:

The Electrocardiographic screening is extremely significant regarding to find out the actual and accurate picture of cardiac electrical conduction of Athletes Heart. These are:

1. To find out the duration and height of atrial depolarization (P wave) in the sportsmen participating in the different categories of sports.
2. To find out the Atrioventricular conduction of heart (PR interval) in the sportsmen participating in the different types of sports.
3. To find out the right and left ventricle hypertrophy (R wave in v5,v6 +Q wave in v1,v2) in the sportsmen participating in the different kind of sports .
4. To find out the duration of ventricular depolarization (QRS complex) in the sportsmen participated in the different kind of sports.
5. To find out the Ventricular depolarization angle (QRS electric axis) in the sportsmen participating in the different kind of sports.
6. To find out the period of ventricular Repolarization (T wave) in the sportsmen participating in the different kind of sports.
7. To find out the ST segment elevation and depression in the sportsmen participating in the different kind of sports.
8. To find out the timing from initial depolarization of ventricles to the end of their Repolarization (QT interval – a risk factor for the ventricles) in the sportsmen participating in different kind of sports.
9. To find out the resting heart rate (RR interval – one cadiac cycle) in the sportsmen participating in the different kind of sports.

As medical providers we all work to prevent death while improving quality of life. Therefore, it is difficult to see a young athlete, allegedly in good health, die suddenly as it contradicts all efforts we work towards. Witnessing this unforeseen event draws out the question, “how?” Young athletes are required to receive routine medical screening, however, it is possible for certain pathologies to be silent until a fatal event. It seems as if there should be something the medical community should do to prevent the unforeseen theft of these lives. Sudden Cardiac Death (SCD) is an uncommon incident, only occurring in 0.61/100,000 persons per year in the U.S.1 By definition SCD is a death in which the patient had stable cardiac function until the event, and usually occurs less than one hour from symptom onset. 2 Corrado et al determined the relative risk of sudden cardiac death was 2.8 times greater for an athlete compared to their non-athlete counterparts.3 In the U.S. the top five cardiac abnormalities that cause SCD are, in order of prevalence, hypertrophic cardiomyopathy, coronary artery anomalies of wrong sinus origin, myocarditis, arrhythmogenic right ventricular cardiomyopathy, and ion channelopathies such

as Brugada Syndrome and Long QT Syndrome1. It is shown that hypertrophic cardiomyopathy accounts for 1/3 of SCD a year. Other causes of SCD, including Wolf-Parkinson-White syndrome, Marfan Syndrome, cardiac sarcoidosis, blunt trauma to the chest, myxomatous mitral valve degeneration, premature atherosclerotic coronary artery disease, and aortic valve stenosis, will not be addressed in
this paper.

Once a provider comes to the diagnosis of a cardiac abnormality, recommendations of activity level must be provided. The Bethesda Conference is a convention, sponsored by the American college of Cardiology Foundation, in which representatives from various organizations, including cardiologists and other providers from the medical community, discusses the latest details of cardiovascular abnormalities in trained athletes. All information is reviewed and recommendations of activity level according to diagnosis are formulated. The U.S. as well as the European medical and athletic communities adheres to these guidelines. It is noted that these recommendations are specific for competitive athletes and not to be utilized with non-competitive recreational athletes. Table 1 outlines the recommendations from the last conference, held in November 2004, for each of the top five diagnoses that have the potential to cause SCD.

Hypertrophic Cardiomyopathy

Hypertrophic Cardiomyopathy is an idiopathic disease of the heart that has a familial link. It is considered an autosomal dominant disease that can also develop due to genetic mutations. The left ventricle and/ or right ventricle develop asymmetrical hypertrophy without an obvious cause, like aortic stenosis. The apical portion of the left ventricle is most often the hypertrophic portion. Some patients will also have left ventricular stiffness and subsequent impaired filling. Patients may be asymptomatic or complain of symptoms such as dyspnea, angina, fatigue, syncope, or palpitations.On physical exam one will find a harsh systolic crescendo-decrescendo murmur between the apex and left sternal border. The murmur increases with the Valsalva maneuver or when standing from squatting, and decreases when squatting from standing, and with passive leg elevation, or handgrip.The murmur of hypertrophic cardiomyopathy is unlike the murmur of aortic stenosis, in that it does not radiate to the carotid arteries. It will, however, radiate to the lower sternal border, axillae, and to the base of the heart. One may also note mild cardiomegaly, apical systolic thrill and heave, brisk carotid upstroke, or an S4 heart sound on physical exam.

Electrocardiogram (EKG) may reveal left ventricular hypertrophy (LVH), ST and T-wave abnormalities, abnormal Q-waves, or atrial and ventricular arrhythmias. EKG is abnormal in 75%-95% of patients with HCM. Echocardiogram is considered the gold standard test to diagnose hypertrophic cardiomyopathy, and will reveal an asymmetrically hypertrophied septum, a narrow left ventricular outflow tract, a small to normal sized left ventricle, or systolic anterior motion of the mitral valve.4 Treatment for hypertrophic cardiomyopathy is medical

symptomatic treatment unless there is obstruction. Alcohol ablation, myectomy, pacemaker, or implantable cardioverter-defibrillator (ICD) would be considered depending on the severity of the obstruction and success of less invasive treatment options. It is recommended that digoxin be avoided unless atrial fibrillation develops or there is systolic dysfunction. 4

Coronary artery anomalies of wrong sinus origin

Coronary artery anomalies of wrong sinus origin are a congenital malformation of one of the coronary arteries. The anomalous artery originates on the opposite side of the aorta than intended. In the anomalous position the artery travels between the aorta and the pulmonary artery trunk. This positioning creates an angling and a slit-like opening of the vessel, which decreases blood flow through the artery.4 Patients may present with exertional syncope, chest pain, dizziness, or symptomatic ventricular arrhythmias. Most often patients are asymptomatic, have normal EKGs, and no significant findings on physical exam. Identification of anomalies can be done using MRI or ultrafast computed tomography imaging, but coronary arteriography is considered the best diagnostic study. 5Treatment for this malformation is surgical bypass.

Myocarditis

Myocarditis occurs most commonly from a viral infection (coxsackeivirus B), causing inflammation and necrosis of the myocardium, which also could implicate the endocardium, the pericardium, and the valves. Myocarditis may also result from adenovirus, parvovirus, drugs, or toxins like cocaine. It ishypothesized that the viral infection triggers an immune response that subsequently injures the myocardium after the virus has cleared. Patients may present with symptoms of congestive heart failure, but most commonly are asymptomatic. Symptoms such as chest pain dizziness, syncope, palpitations, tachyarrhythmias or bradyarrhythmias may appear days to weeks after a febrile illness. On physical exam the patient may exhibit tachycardia, hypotension, fever, murmur of mitral or tricuspid regurgitation, S3 or S4 gallops. EKG abnormalities, such as ST segment and T-wave changes, conduction delays, left bundle branch block, AV block, supraventricular tachycardia, or ventricular ectopy are transient, and most often found in first 2 weeks of febrile illness. An echocardiogram will often reveal regional wall motion abnormalities, mitral or tricuspid regurgitation. Once the diagnosis is determined, serial echos will be closely scrutinized to determine progression of the disease.Erythrocyte sedimentation rate (ESR) will be elevated and may be used to monitor the course of the disease. Creatinine phosphokinase-myocardial band (CPK-MB) and cardiac troponin-I (tn-I) may alsobe elevated. Endomyocardial biopsy is the gold standard diagnostic test. However, biopsy is not required since it does not contribute toward the treatment of myocarditis, unless it becomes necessary to make a more specific diagnosis should the patient fail to respond to standard therapy. Patients with symptomatic myocarditis should be hospitalized for symptom control.2

Arrhythmogenic right ventricular cardiomyopathy

Arrhythmogenic right ventricular cardiomyopathy is another disease that is autosomal dominant. In the disease process myocardial cells of the right ventricle are partially or totally replaced by adipose and fibrous tissue. These changes lead to reentrant ventricular tachycardia which appears in the EKG as a left bundle branch block. The EKG may also show inverted T-waves in right precordial leads and epsilon waves. Further imaging may reveal right ventricular dilation and poor contractility with a normal left ventricle. The diagnostic test of choice is MRI.4 Patients may experience palpitations or syncope, which can be treated with antiarrhymics. However, if medications do not control symptoms, cryo-based or catheter-based radiofrequency ablation, ICD, or transplant must be considered. WPW pattern is defined as a short PR interval (<120 ms), the presence of a delta wave (slurring of the initial QRS) and a wide QRS (>120 ms).55 WPW pattern should be differentiated from a low atrial rhythm with a short PR interval that is a common finding in athletes. The short PR is a result of the impulse being generated by an atrial focus outside the sinus node and closer to the AV node. Due to the proximity to the AV node, atrial conduction time is reduced and the PR interval is shortened. Findings that can help differentiate this from preexcitation

include an atypical P wave axis (negative P wave in the inferior leads) suggesting the atrium is activated from bottom to top rather than top to bottom as in sinus rhythm. No further evaluation is recommended for asymptomatic athletes with only a short PR interval and no other

ECG abnormality.

Brugada type-1 ECG (left) should be distinguished from early repolarisation with ‘convex’ ST-segment elevation in a trained athlete (right). Vertical lines mark the J-point (STJ) and the point 80 ms after the J-point (ST80), where the amplitudes of the ST segment elevation are calculated. The ‘downsloping’ ST segment elevation in Brugada pattern is characterised by a STJ/ST80 ratio >1. Early repolarisation patterns in an athlete show an initial ‘upsloping’ ST segment elevation with STJ/ST80 ratio <1.8 of 17. Evaluation of WPW The diagnostic evaluation of asymptomatic athletes with WPW pattern remains controversial and is conducted usually by an electrophysiologist. Stratification methods for the risk of sudden death include invasive and non-invasive tests. Non-invasive measures of a low-risk accessory pathway include intermittent pre-excitation during sinus rhythm and abrupt, complete loss of pre-excitation during an exercise stress test.56 57 If non-invasive testing is inconclusive, electrophysiology testing should be considered. Characteristics of a high-risk pathway are generally determined during an electrophysiology study by the shortest pre-excited RR interval during induced atrial fibrillation. If the shortest pre-excited RR interval is measured as ≤250 ms [240 beats/min (bpm)] then the pathway is deemed high risk.52 Young athletes with a shortest pre-excited RR interval ≤250 ms should proceed with transcatheter ablation.58 An echocardiogram should also be considered due to the association of WPW with

Ebstein's anomaly and cardiomyopathy. Profound first-degree AV block.

Diagnostic criteria

The high vagal tone in athletes also leads to a slowing of AV nodal conduction, and hence a lengthening of the PR interval. It is not uncommon to see PR intervals longer than 200 ms in

athletes at rest. Even significant PR prolongation ≥300 ms may occur, although this by itself is not necessarily pathological and is usually asymptomatic.

Evaluation

In asymptomatic athletes with a profound first-degree AV block (≥300 ms), the athlete should undergo a minimal exercise load (ie, like climbing a flight of stairs) to increase sympathetic tone. If this results in shortening and normalisation of the PR interval, the PR prolongation is due to functional (vagal) mechanisms and hence benign. If the PR interval does not normalise to ≤200 ms with exercise, a structural cause of AV conduction disturbance (such as Lyme disease or sarcoidosis) should be investigated. Athletes with a profound first-degree AV block (≥300 ms) who have symptoms (ie, syncope, palpitations) or a positivefamily history of cardiac disease or sudden death require additional evaluation to rule out pathological causes of heart block.

Mobitz type II second-degree AV block

Diagnostic criteria

An abrupt loss of P wave conduction (P wave with no ensuing QRS complex), without prior PR prolongation, represents Mobitz type II second-degree AV block. If Mobitz type II or more advanced types of AV block including 2:1 or 3:1 occur during sinus rhythm, it may be indicative of underlying structural heart disease.

Evaluation

Suspected Mobitz type II second-degree AV block or other more advanced types of AV block (2:1 or 3:1 block) should first be differentiated from Wenckebach (Mobitz type I) second-degree AV block. Wenckebach (Mobitz type I) block is present when there is PR prolongation before a blocked P wave and a shorter PR in the first conducted beat after the block. Mobitz type I second-degree AV block is usually a functional block from increased vagal tone and does not constitute pathology in an athlete. Further diagnostic evaluation can be done with an ECG after minor exercise, as a slight increase in sympathetic tone will resolve the conduction disturbance in physiological cases. A Holter monitor (or other form of long-term ECG recording) also can assist in clarifying the type of AV block. Mobitz type II or higher degree (2:1 or 3:1) AV block requires further evaluation for underlying pathological cardiac disease. ECG showing Mobitz type II second-degree AV block. Note the presence of P waves with loss of conduction and no QRS comple (arrows) and without PR prolongation in the beats prior, nor PR shortening in the beats after (which would suggest Mobitz type I). Mobitz type II second-degree AV block in an athlete is not due to increased vagal tone and should prompt evaluation for underlying conduction disease. This is only reproduced in colour in the online version. Drezner JA, et al. Br J Sports Med 2013;47:153–167. doi:10.1136/bjsports-2012-092070 13 of 17

Third-degree AV block/complete heart block

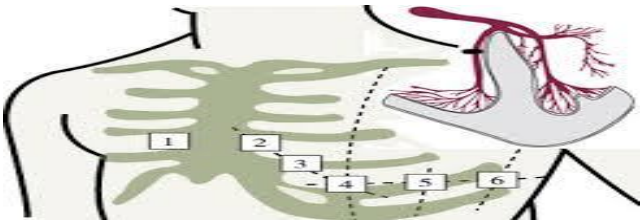
Diagnostic criteria

Complete heart block is not an expression of athlete's heart and should be considered an abnormal finding requiring additional evaluation.

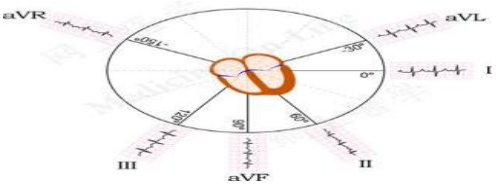
Evaluation With true third-degree AV block, there are more P waves than QRS complexes and the ventricular rhythm is perfectly regular due to an undisturbed junctional or ventricular pacemaker. Complete heart block can easily be Figured. Signal Averaged Eelectro CardioGram (SAECG) is a very specialized type of surface ECG, which involves computerized analysis of small segments of a standard ECG in order to detect abnormalities, termed as ventricular late potentials (VLP) which would otherwise be obscured by skeletal muscle and electrical noise. There are various types of these low amplitude signals which can be detected by SAECG [11], but the most important are VLP and the main clinical use of SAECG is to detect these VLPs. VLP are present in terminal part of QRS complex and these may extend into ST segment; hence called 'late potentials' because these arise late in ventricular activation process. Ventricular late potentials had been the pivotal point of cardiac electro physiologic research for the last twenty-five years and they have been investigated extensively. VLPs arise in an area of myocardium where the conduction velocity of cardiac impulse is slow, e.g., peri-infarct zone. This means when depolarization in healthy myocardium is complete or almost complete this area will still be depolarizing. It will lead to the generation of low voltage, fractionated signals towards the end of QRS complex. The same area will become substrate for the development of ventricular tachyarrhythmias through re-entry mechanism [12]. Therefore VLP may act as noninvasive marker for the development of ventricular tachyarrhythmias. The criteria for their presence and co-relation with ventricular tachyarrhythmias are now well established. In this way, we can say that the pre-participation Electrocardiographic screening is extremely important and highly beneficial to detect any kind of cardiac abnormalities, if any.

Definitions of the important terms:

1. ECG Lead: Lead refer to the electrical cable attaching the electrodes to the ECG recorder.

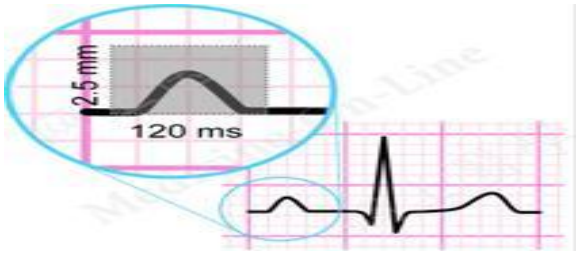


2. Limb Lead: The electrode which are located on the limbs- one on each arm and one on the left leg. These are also known as L.1, L.2 and L.3 .
3. Chest Lead: There are six chest lead also with electrodes positioned horizontally around the left anterior hemi-theorax between the 4thand 5th interspaces, such as : V1,V2,V3,V4,V5,V6.
4. Augmented Lead: Lead aVR, aVL, aVFare the augmented lead. These are derived from the same three electrodes as lead , 1, 2, and 3. However, they view the Heart from different angles.

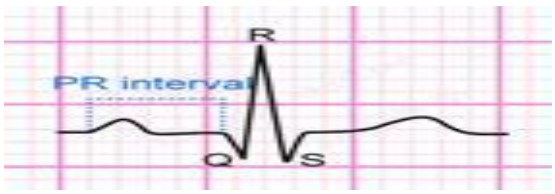


p-ISSN: 2348-6848
e-ISSN: 2348-795X
Volume 02 Issue 03
March 2015

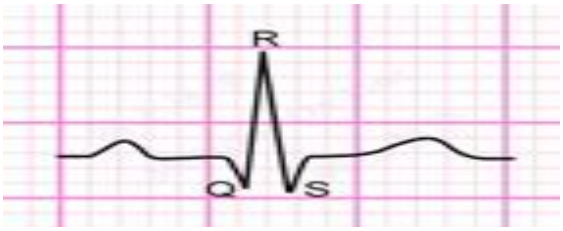
5. Axis: The Heart electrical axis refer to the general direction of the Heart ' depolarization wave front in the frontal plane.
6. Atrial depolarization (P wave) – the first deflection of the P wave associated with right and left Atrial contraction (depolarization) the normal duration of p wave is 80 ms.



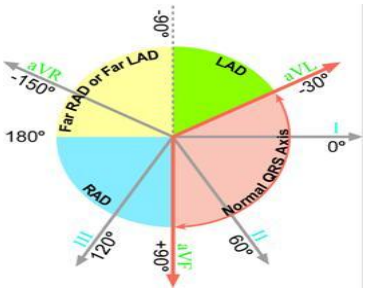
7. Atrioventricular conduction (PR interval): the PR interval reflect the time the electrical impulses take to travel from the SA node through the AV node and entering the ventricles. Normal interval is 120-200 ms.



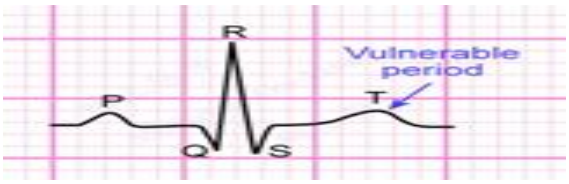
8. Right and left ventricles hypertrophy: the enlargement of the ventricles. The Heart muscle cell became enlarged due to an increased demand for the Heart to work harder.
9. Ventricular depolarization (QRS complex) - : the QRS complex reflect the rapid depolarization of the right and left ventricles. This complex has a series of three deflections that reflect the current associated with right and left ventricle depolarization. The normal duration of QRS complex is 80-120 ms.



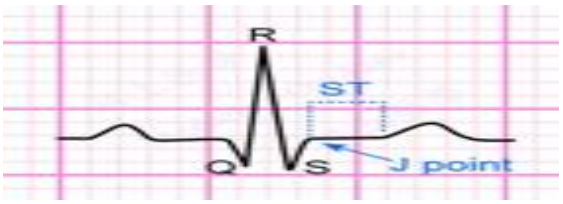
10. Ventricular depolarization angle (QRS electrical axis) : the QRS electrical axis refer to the direction of ventricular depolarization wave front . The normal QRS axis is -30 to +90.



11. Ventricular Repolarization (T wave) - its a Repolarization (recovery period) of the ventricles represented by T wave. It's a vulnerable period. Normal interval is 160 ms.

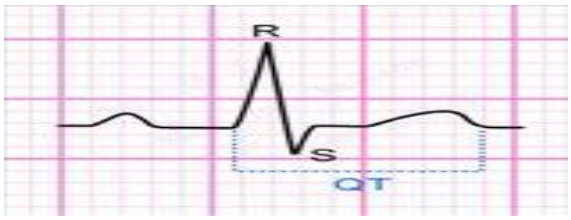


12. ST segment :- It connect the QRS complex and T wave. The normal period of this segment is 80-120 ms.



13. QT interval :- The QT interval is measured from the beginning of QRS complex to the end of T wave. It represent the time in which the ventricles depolarized and repolarized andits also

a measurement of ventricular action potential duration. Normal interval is up to 420 ms in heart rate of 60 bpm.



14. Resting Heart Rate (RR interval) :- The interval between an R wave and the next R wave . its also known as one cardiac cycle. The normal interval is 0.6 to 1.2 sec.

Conclusion:

The chief aim of this paper is to highlight the significance of ECG screening in sports. Electrocardiographic screening in sports cannot be ignoring. When we are investing Billions of Dollars to achieve the best performance in sports then why we are ignoring this kind of extremely significant safety parameter.

Thus, we can say that the importance of Electrocardiographic screening cannot be ignored and avoid. If we want to get the best performance then we must keep in our strategy plan so that we can provide a better platform and a healthy environment to the sportspersons.

References:

1. Dilaveris PE, Gialafos EJ. Simple electrocardiographic marker for the prediction of paraxymal idiopathic Atrial fibrillation. AM heart j 1988,135,733-738
2. BasilicoFC .cardiovascular disease in Athletes . AM j sports med,1999,27,108-121
3. Crouse SF ,Rohack JJ , Jacobsen DJ : cardiac structure and function in women basketball athletes. Res,Qexar sports 1992, 63,393-401
4. Maron BJ: Heart disease and other cause of sudden cardiac death in young athletes. Currproblcardiol 1998,23,477-529
5. MaronBJ ,Pelliccia A : the heart of trained athletes, cardiac remodeling and the risk of sports circulation , 2006,114,1633-1644
6. MorganrothJ ,Maron BJ : the athletes heart syndromes, a new perspectives. Ann nyacadsci 1977,301,931-941
7. PollicciaA ,Di Paolo FM : outcomesin athletes with mark ECG Repolarization abnormalities. N engl j med 2008,358,152-161
8. OakieyCM , the ecg in the highly trained athletes. Casdiolclin 1992,10,295-302
9. Smith WG ,Gullen KJ, electrocardiogram of marathon runners in the 1962 commonwealth games. By heart 1964,26,469-76
10. Oakley DJ,Oakley CM .significance of abnormal electrocardiogram in highly trained athletes.Amcardiol, 1982,50,985-89