Electrocardiography in pre-participation screening and current guidelines for participation in competitive sports

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SUMMARY

Electrocardiography (ECG) is especially significant in pre-participation screening due to its ability to discover or to rise a suspicion for certain cardiovascular diseases and conditions that represent a serious health risk in athletes. Common, conditionally benign and training related ECG changes are sinus bradycardia and sinus arrhythmia, first degree atrioventricular block, incomplete right bundle branch block, benign early repolarization, and isolated QRS voltage criteria for left ventricular enlargement. Uncommon ECG changes, unrelated to training, and some specific syndromes are ST segment depression and/or ≥ 2 mm T wave inversion in two or more adjacent leads, intraventricular conduction disorder, Wolf–Parkinson–White syndrome, long QT interval syndrome, short QT interval syndrome, catecholaminergic polymorphic ventricular tachycardia, monomorphic ventricular extrasystole and benign ventricular tachycardia.

Keywords: examination; athletes; recommendations

INTRODUCTION

In sport, among active competitors, there is a silent fear of sudden cardiac death (SCD). Many are aware that it can be of an abrupt onset and is frequently without alarming symptoms, so the question that remains in the air after a sports exam is, "Is my heart healthy?"

With only history taking and examination, cardiac disease is often underdiagnosed while electrocardiography (ECG), in most cases, can reveal abnormalities. European Society of Cardiology (ESC), International Olympic Committee and, at present, many sports associations, recognize the benefit of ECG in pre-participation screening (PPS) and it is constituted in routine examinations of athletes [1, 2]. American Heart Association (AHA) that determines guidelines for screening in the USA does not involve ECG as an obligatory part of PPS, although numerous papers published in that country speak in favor of ECG screening [3-6]. AHA considers ECG a nonspecific and costly tool for screening a substantial number of athletes [3]. The greatest, non-randomized prospective study on PPS of athletes was performed in Italy on 42,386 athletes over a 26-year period and it resulted in a decrease of the average rate of SCD by 63% from the early to the late screening period [7]. Another thorough study was conducted in Italy in 2003, including 32,652 athletes, where the authors found that the total number of markedly abnormal ECGs that would require additional testing was less than 5% [8]. Guidelines for PPS evolved greatly over the last several years. The ESC recommen-

dations from 2010 were upgraded in 2011 by Dr. Froelicher's group of the Stanford University, called the Stanford criteria. Additionally, the ECG criteria were further refined in 2012 at the setting of a conference held in Seattle, hence the name the Seattle criteria. Improvement that was introduced in 2011 referred to a better way of defining and clearer cut-off values for long QT interval and intra-ventricular conduction delay. Also, a T wave inversion associated with ST segment elevation in precordial V1–V4 leads in black athletes was classified under "common and training-related" ECG changes [9]. The subsequent Seattle criteria provided more detailed descriptions of possible adaptational and pathological findings with reference values suggestive of abnormalities and instructions for further management of athletes with abnormal ECGs [10]. Practical application and comparison of ESC 2010, the Stanford and the Seattle criteria showed that the latter demonstrates much higher specificity with unchanged sensitivity in detection of ECG abnormalities in athletes and, conversely, a high degree of common, training-related ECG changes [11, 12, 13]. The main cause for improved specificity are comprehensive recommendations in regard to interpretation of T wave inversion, right atrial enlargement, right ventricular hypertrophy, intra-ventricular conduction delay, and QTc interval prolongation and shortening [14, 15]. The newest, refined ECG criteria offered by Riding et al. [16] confirmed a lower rate of false positives in Arabic, black and Caucasian male athletes then the aforementioned ones, while remaining 100%

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sensitive in identifying all athletes with cardiac pathology associated with SCDs.

Athletes around the world perform sports examinations at various settings, from modern centers for sports medicine to general practitioner's offices. In our country, according to current legislation, examinations of all athletes should be done by sports medicine specialists [17]. In lack thereof, examinations of athletes can be made by internal medicine specialists, occupational medicine specialists, general practitioners, and pediatricians. There is a slowly marching world trend that these examinations should be done by sports cardiologists. Sports cardiology is a branch that slowly evolves into an independent specialty, but in our country it does not exist uniformly yet. By the time it does, the necessity of correct ECG diagnostics in athletes needs to be taken seriously and an effort to make it more efficient and available must be made by highlighting the guidelines through education seminars.

The purpose of this article was to itemize specific ECG markings that can be seen in athletes and to discriminate "similarities" in physiological and pathological findings.

Most studies agree that the commonest, conditionally benign abnormalities, seen in athletes' ECGs are sinus bradycardia, sinus arrhythmia, first degree atrioventricular (AV) block, incomplete right bundle branch block, benign early repolarization, and isolated QRS voltage criteria for left ventricular hypertrophy [1, 18, 19].

COMMON AND TRAINING RELATED ECG CHANGES

Sinus bradycardia represents heart frequency below 60 beats per minute, of sinus node origin. It is inversely proportional to the level of training and, in exceptional athletes, values less than 30 beats per minute with asymptomatic sinus pauses during sleep are often observed. Langdeau et al. [20] tested 100 elite athletes and found medium heart frequency to be 52 beats per minute. Recommendations for marked sinus bradycardia (less than 30 beats per minute) and sinus arrhythmia with pauses longer than three seconds in awake state, are to perform additional tests (24-hour ECG, echocardiography, stress tolerance test) in order to exclude the "sick sinus" syndrome. It is a good sign if the competitor is without symptoms and heart frequency rises directly proportional to physical exertion. Then, all competitive sports are allowed with yearly check-ups. If there are no structural changes of the heart but there are symptoms like dizziness and syncope which subside after three months without training, practicing sports is allowed with check-ups every one to six months. Lately, there are many articles in favor of existence of intrinsic changes in sinoatrial node that are, besides high parasympathetic tone, possible cause for resting bradycardia in highly trained athletes [1, 21-24].

First and second (Mobitz type I) degree AV block are frequently seen in basketball, volleyball and football players. If there are no symptoms, the heart is without structural changes and QRS complexes are not deformed, further evaluation is not necessary, and practicing all va-

rieties of sports is allowed with yearly check-ups. Presence of abnormal QRS complexes and PR interval in duration of 0.3 seconds and longer that does not resolve during exercise test requires detailed diagnostics. Implantation of a pacemaker is most often considered or, sometimes, the athlete is re-evaluated after one to two months of being excluded from sports. Absence of symptoms will enable him to be engaged in less intensive activities with control examinations every one to three months. Persistence of symptoms or presence of structural heart abnormalities will require implantation of a pacemaker with exclusion from all kinds of contact sports [1, 9, 10, 18, 22, 23, 25].

Second (Mobitz type II) and third degree AV block are findings that demand more complex examinations with permanent pacing before any sports activity. However, according to AHA guidelines, those who are asymptomatic, have structurally normal heart, do not have history of pre-syncope and syncope, QRS complexes are narrow with ventricular rhythm of 40–50 beats per minute at rest and adequately rise with exertion, with or without occasional ventricular extrasystole (VES) and without ventricular tachycardia in exertion, can participate in all kinds of competitive sports [1, 22, 23].

Incomplete right bundle branch block (IRBBB) is present in about one third of athletes [11, 25, 26], predominately in male sex. R-R' pattern (rsR') in V1 lead is a characteristic finding and QRS complex duration is 0.08–0.12 milliseconds (SI: ms). Also a wide terminal S wave in leads I and V6 is present. It can be of reversible morphology with the cessation of training. Negative family and personal history and normal physical examination are enough for evaluation [25].

Arrhythmogenic right ventricular dysplasia/cardiomyopathy (ARVD/C) affects 0.6–1 / 100,000 persons in a population and is the most common cause of SCD in Europe. It is considered that individuals with ARVD/C have five times higher risk of sudden death if they engage in sports. If ECG shows an IRBBB pattern with presence of chest pain and rapid heartbeat, other ECG markers should be looked for. IRBBB paired with inverted T wave that is not seen only in V1 and V2 but involves V3 and V4 with the presence of VES (frequent, more than 1,000 in 24 hours) and morphology of left bundle branch block (LBBB) are highly suspicious of ARVD/C. A recent study compared diagnostic findings in athletes and patients with ARVC and the authors noticed that the majority of athletes with inverted T waves exhibited biphasic T wave morphology preceded with convex ST segment elevation, opposed to ARVC patents that had T wave inversion preceded with isoelectric ST segment [27]. Another unique characteristic of this disease is the presence of epsilon wave, a discrete, sharp deflection at the end of QRS complex, most visible at leads V1–V3. Individuals with ARVD/C are banned from all competitive sports but are sometimes allowed to practice low static and dynamic sports [1, 21, 28-31].

Brugada syndrome is autosomal dominant channelopathy involving sodium channels that can escalate in monomorphic or polymorphic ventricular tachycardia, or even fatal ventricular fibrillation. Incidence is one in

every 2,000–5,000 persons. In many cases first symptom of a disease was sudden death. Patients report characteristic symptoms like pre-syncope, syncope and palpitations. Typical ECG consists of R-R' morphology of IRBBB with high origin of ST segment ($\geq$ 2 mm [SI: mm]) that slopes down (J wave) resulting in negative, deeply concave or "saddle-back" form of T wave, seen in leads V1–V3. There are three types. Type 1 is diagnostic and is characterized by ST segment $\geq$ 2 mm which rushes into deeply concave T wave without isoelectric separation. Types 2 and 3 are characterized by "saddle-back" T wave, both have high origin of ST segment ($\geq$ 2 mm) and at the point of $\geq$ 1 mm it becomes followed with biphasic or positive T wave (type 2) or at the point of < 1 mm it is followed with positive "saddle-back" T wave (type 3). Most deaths occur during rest, but strenuous exercises may potentiate bradycardia or rise core temperature to and above 40°C, which can precipitate malignant arrhythmias. Diagnostic criteria that measure relation of amplitudes of J-point and at the 80 ms following J point (STJ and ST80) are used. In athletes it should be $\leq$ 1 and in Brugada syndrome it is > 1. But definitive diagnosis of type 1 Brugada syndrome is when ST segment elevation is observed either spontaneously or after intravenous administration of sodium channel blocking agent (ajmaline, flecainide, pilsicainide or procainamide) in at least one precordial lead (V1 and V2) when ECG electrodes are placed in standard or superior position. Athletes diagnosed with Brugada syndrome, according to AHA guidelines, are proved for competitive sports with low cardiovascular demands and with avoiding hyperthermia and electrolyte imbalance, provided that they have control examinations every year. ESC orders exclusion of these athletes from all competitive sports [1, 21, 28, 32-36].

Atrial septal defect (ASD) frequently goes with IRBBB and coupling of second heart tone should be excluded. If there is a small ASD (< 6 mm) or six months post closure, with normal pulmonary pressure and no significant arrhythmia or ventricular dysfunction, practicing all sports is allowed [1, 37].

Isolated QRS complex voltage augmentation for left ventricular hypertrophy (LVH), according to Sokolow–Lyon and Cornell criteria, does not require echocardiography, unless there are symptoms, positive family history and/or non-voltage criteria for LVH. This finding is not rare in sports like cycling, cross-country skiing and rowing [25, 34, 38].

Hypertrophic cardiomyopathy (HCM) can have similar pattern on ECG and close attention is required during interpretation of the results. Isolated QRS complex voltage augmentation for LVH is seen in only 2% of those with the disease. The biggest difference in ECG is presence of prominent RS or even Rs waves in right precordial leads (V1–V3) and qR waves in lateral or inferior leads. The newest recommendation is that Q waves that are > 3 mm in depth and/or > 40 ms in duration in any lead except III and aVR should be further evaluated. Suspicion of HCM should be made if there is a T wave inversion more than 1 mm in depth in two or more leads V2–V6, II and aVF, or I and aVL. Excepted from this rule are juvenile

T waves that may be inverted in leads V1–V3 until early adolescence and domed ST segment elevation followed by inverted T waves in V1–V4 in Afro/Caribbean athletes. In HCM, inverted T waves can be accompanied with ST segment depression in two or more adjacent leads. LBBB with left axis deviation and left atrial enlargement are also frequently seen. Upper echocardiographic limit for LV wall thickness in athletes is considered to be $\leq$ 12 mm, but in small amount of healthy sportsmen it may go up to 14 mm. Endurance sports lead to increase in LV diastolic chamber size and eccentric LV hypertrophy with normal wall thickness. Isometric or strength-based exercises are characterized by a mild increase in LV wall thickness resulting in concentric LV hypertrophy. This form of hypertrophy may mimic HCM because in fewer number of cases HCM can be symmetrical. This is why the greatest diagnostic problem in echocardiography is represented by the "grey zone," where LV wall thickness is between 13 mm and 15 mm. LV cavity dimensions in athletes are 40–60 mm, compared to HCM patents, where dimensions rarely exceed 50 mm. One Italian study showed that LV cavity size < 54 mm had 100% specificity and sensitivity in distinguishing HCM from athlete's heart. Cardiopulmonary exercise testing may be helpful in diagnosing HCM because these athletes usually have low peak oxygen consumption, but such tests can be performed only in fully equipped laboratories with experienced staff. After cessation of training stimulus, athlete's heart returns to normal dimensions following several years of detraining in eccentric LVH and after six months in concentric LVH. In HCM the reduction in myocardial diameters following detraining may be seen in minimal extent but it is never complete. AHA and ESC are unanimous that ones with probable or definite HCM should be excluded from competitive sports. The difference is that AHA allows competing for those who are genetically positive but phenotypically negative [1, 9, 10, 19, 21, 28, 38-46].

Early repolarization is frequent, benign finding, seen in 50–80% of athletes. It is characterized by ST segment elevation starting from J point (juncture of QRS complex and ST segment) for at least 0.1 millivolt (SI: mV). It is often present in precordial leads, usually V3 and V4, but it can also be seen in V5, V6, I, avL, as in II, III, avF or V2. In Caucasians, this elevation is concave upwards, ending with positive and frequently prominent T wave, whereas in black race it can be convex upwards and ending with negative T wave in leads V2–V4. It is assumed that ST elevation in inferior and lateral leads with J point elevation and QRS slurring can be associated with the development of polymorphic ventricular tachycardia or ventricular fibrillation. Having that in mind, presence of symptoms requires additional diagnostics. In the work of Cappato et al. [47] it is noted that QRS slurring without ST elevation was a frequent finding in electrocardiograms of athletes that survived cardiac arrest or have died from sudden cardiac death, while ST elevation alone was a frequent finding in the control group of healthy individuals. Nevertheless, all patterns of early repolarization, including ST elevation, J point elevation, J wave, and QRS slurring, should be considered normal variants in athletes [1, 9, 10, 21, 47, 48].

UNUSUAL ECG CHANGES NOT RELATED TO TRAINING AND SOME SPECIFIC SYNDROMES

ST segment depression and/or T wave inversion ≥ 2 mm in two or more adjacent leads are markers of high cardiovascular risk and should be further examined. ST segment depression ≥ 0.5 mm in two or more leads and/or T wave inversion in precordial (V2–V6), inferior (II and avF) and lateral (I and avL) leads should rise a suspicion for heart disease. Flattened or minimally inverted T wave (< 2 mm) may be a benign phenomenon subversive to reversion after a light physical activity. It is rarely seen in healthy individuals, but it is frequent in cardiomyopathies and should not be neglected. Post-pubertal presence of inverted T wave, beside V1, may be pathological as well. Pelliccia et al. [37] analyzed occurrence of cardiovascular complications in athletes without structural anomalies that initially had inverted T wave more than 2 mm in depth in three or more leads, predominately precordial, through multiple-year follow-up. From 12,000 analyzed cases, initial ECGs showed repolarization anomalies in 1%, but only 81 athletes were included in the study. After the follow-up (which lasted from one to 27 years, SD 9 ± 7) cardiomyopathies (three cases of HCM, one case of ARVD/C, and one case of dilated cardiomyopathy) were determined in 6% (five athletes) [1, 10, 18, 49].

Congenital coronary artery anomalies are among the most frequent causes of sudden death in athletes. Origination of left coronary artery from right Valsalva sinus is not rare. SCD results from ventricular arrhythmia triggered by ischemia during exercise due to impaired coronary blood flow coming from compression of anomalous vessel as it courses between pulmonary artery and ascending aorta. This condition is hard to diagnose without magnetic resonant angiography or computed tomography coronary angiography. ECGs of these individuals rarely show signs of abnormalities. Prohibition from sports goes for all competitive sports [1, 18, 28].

Myocarditis should be suspected in otherwise healthy person that gives information about recent viral illness, newly occurred physical intolerance, clinical signs of cardiac insufficiency with presence of repolarization abnormalities on ECG and/or regional dyskinesia of ventricle wall. In addition, during infection with cardiotropic viruses there is a rise in minimal and fall of maximal heart frequency, hence heart rate variability can be a marker for autonomic nervous system state in a diseased myocardium. Active disease demands exclusion from all kinds of competitive sports. After six months from the occurrence of first symptoms it is necessary to determine resolution of the disease with personal history and examination, ECG, echocardiography, stress tolerance test, and Holter ECG, and if the findings are negative, return to sport is warranted [30, 50].

Intraventricular conduction delay – any QRS complex of ≥ 140 ms in duration requires stress testing, 24-hour ECG, and imaging methods. Complete LBBB morphology with QRS ≥ 120 ms, predominantly negative QRS complex in lead V1 (QS or rS), and upright monophasic R wave in leads I and V6 also requires further diagnostics [10, 40].

Lenegre's disease is an autosomal dominant disease that leads to progressive destruction of a conduction system of the heart and can be lethal in athletes [34, 51].

Wolf–Parkinson–White syndrome is a ventricular pre-excitation resulted from anterograde conduction of impulses via accessory atrioventricular pathway. Prevalence is similar in athletes and general population (0.1–0.3%). Majority of affected people are asymptomatic, but palpitations may be reported. Characteristic ECG finding is the presence of a delta wave, shortened PR interval and widened QRS complex. Individuals who have adjacent atrial fibrillation are at much greater risk of exercise induced ventricular fibrillation. Individuals that do not have any symptoms, positive family history or structural changes of the heart are allowed to compete in all sports. Although, younger athletes in particular, regardless of being without symptoms or structural heart disease, especially if the refractory period of the accessory pathway is ≤ 240 ms (six small boxes on an ECG), should undergo in-depth evaluation, including electrophysiological testing, before permission to participate in moderate to high-intensity competitive sports. Those who experience symptoms can participate three months after corrective ablation. Long refractory period is linked with low risk of sudden cardiac death; hence, the ablation is not necessary, but it is strongly advised to stop physical activity in the presence of palpitations, with regular check-ups as well. If there is a shortened PR interval without a delta wave, other ventricular pre-excitation syndrome – Lown–Ganong–Levine syndrome, or even structural disease of the heart, like Fabry's disease – should be considered [1, 2, 22, 23, 28].

Long QT interval syndrome (LQTS) can be acquired (medications, bradycardia, changes in metabolism, electrolyte imbalance), or inborn, which has similar prevalence as Brugada syndrome, that is 1 / 2,000–5,000 in general population. According to USA data, mortality by this syndrome is 2% in athletes. It is interesting that there are five variants due to mutations in five different genes, three of them code information for potassium channels (LQT1, LQT2, LQT5) and two code information for sodium channels (LQT3, LQT4). Those who have LQT1, the most frequent one, are prone to syncope or cardiac arrest during exertion, especially while swimming or diving. Because of myoclonic seizures in syncope it is often mistaken for epilepsy. Death with LQT2 occurs during emotional arousal or acoustic stimulation, whereas in LQT3 it often happens while sleeping. QT interval is dependent of heart frequency. Consequently, the corrected QT interval (QTc) is being used for diagnostics, ideally when heart rate is 60–90 beats per minute. It is calculated when QT interval becomes divided with square root of R-R interval, expressed in ms. QT interval measuring should be done in leads III, V3 or V5. Lesser number of highly trained athletes (0.4–0.7%) has a slightly longer QT interval in relation to general population, > 440–460 ms. QTc values of ≥ 470 ms for men, or ≥ 480 ms for women, up to the value of 500 ms, are thought to be the "grey zone." If there are no other explanations, 500 ms or more is considered to be definitive LQTS. In both cases, ESC recommends

exclusion from all competitive sports for those that are genotype/phenotype positive. It is also advised to restrain from sudden intense activities and specific triggers. AHA criteria says that definite exclusion from competitive sports is demanded in athletes that survived cardiac arrest or have had syncope. Those without symptoms may compete in low intensity sports, whereas individuals that are genotype positive but phenotype negative can compete in all sports [1, 10, 22, 28, 52, 53, 54].

Short QT interval syndrome implies the QTc values of ≤ 320 ms (≤ 310 ms in children). Causes like hypercalcemia, hyperkalemia, hyperthermia, acidosis, digitalis toxicity or anabolic steroid abuse should be excluded. Engagement in sports with low static and dynamic demands is allowed [1, 22, 23].

Atrial tachyarrhythmias, ventricular arrhythmias and premature ventricular contractions are, according to the Seattle criteria, ECG findings that require thorough diagnostic management. Findings of supraventricular tachycardia, atrial flutter, atrial fibrillation, two or more premature ventricular contractions in 10 second tracings or couplets, triplets, and non-sustained ventricular tachycardia may suggest cardiovascular pathology [10].

In the case of paroxysmal supraventricular tachycardia (atrioventricular nodal reentry tachycardia, atrioventricular reentry tachycardia over a concealed accessory pathway, atrial tachycardia) ablation is recommended for competitive athletes, and if there are no recurrences after three months, competitive sports are allowed, but individual risk of arrhythmia relapse during exertion is to be considered. Athletes with atrial fibrillation can participate in all sports when there is proven rate control, no structural heart disease, and no hemodynamic impairment. For individuals that require ablation and/or drug therapy, engagement in sports is debated on individual basis. European guidelines allow all competitive sports in athletes with atrial flutter if they have no symptoms three or more months after mandatory ablation therapy. USA recommendations demonstrate more loose standards for sports participation in athletes with atrial tachyarrhythmias [22, 23].

Athletes without structural heart disease having premature ventricular contractions that happen during rest, exercise or stress testing, can participate in all competitive sports if VES are not frequent (< 2,000 in 24 hours), and if physical strain does not increase the occurrence of VES or induce symptoms. If symptoms appear in these circumstances or if an athlete is diagnosed with structural heart disease, then only allowance in low static and low dynamic sports participation can be advised. The difference between USA and European recommendations is that ESC guidelines permit only recreational types of activities whilst USA's allow competing in class IA competitive sports [22, 32, 55].

ESC and AHA agreed that in athletes who suffer from nonsustained ventricular tachycardia but have no symptoms or positive family history for SCD, have a structurally and electrophysiologically healthy heart, there are no polymorphic or coupled VES and their incidence in 24-hour monitoring is low (< 2,000), plus occurrence of VES does not increase while exercising, all competitive sports are allowed with yearly control examinations. If there is a cardiovascular disease, then only recreational type of exercises are permitted by ESC recommendations, while AHA permits practicing low-intensity competitive sports [22, 32].

For athletes with sustained ventricular tachycardia that stay asymptomatic during exercise, who have no structural heart disease, who had a successful ablation of one automatic focus or if there is a typical automatism originating from right ventricle outflow tract or left ventricle fascicles or there is yet an automatic focus but with frequency of < 150 beats per minute and < 8–10 successive ventricular beats, participation in all sports is allowed but it demands careful surveillance at least during the first year and more frequent check-ups [22, 32].

The first manifestation of many abovementioned diseases and conditions is, frequently, sudden cardiac arrest, which may often lead to sudden cardiac death. Sports participants are conditionally healthy population that rarely see physicians. By honoring current guidelines and performing routine electrocardiography we can further primary prevention in sports and lower the mortality rate.

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Електрокардиографија у препартиципационом скринингу и важеће препоруке за учествовање у такмичарским спортовима

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КРАТАК САДРЖАЈ

Електрокардиографија (ЕКГ) заузима посебан значај у препартиципационом скринингу јер се њоме могу открити или се може посумњати на извесна кардиоваскуларна обољења и стања која су здравствени ризик за спортисте. Честе, условно безазлене и тренингом условљене ЕКГ промене су синусна брадикардија и синусна аритмија, атриовентрикуларни блок првог степена, непотпуни блок десне гране, бенигна рана реполаризација и изоловани *QRS* волтажни критеријум за хипертрофију леве коморе. Неуобичајене ЕКГ промене које нису у вези с тренингом и неки специфични синдроми су депресија *ST*-сегмента и/или инверзија Т-таласа ≥2 *mm* у два или више суседних одвода, интравентрикуларни поремећај спровођења, Волф–Паркинсон–Вајтов синдром, синдром дугачког *QT* интервала, синдром скраћеног *QT* интервала, катехоламинергичка полиморфна вентрикуларна тахикардија, мономорфне вентрикуларне екстрасистоле и бенигна вентрикуларна тахикардија.

Кључне речи: преглед; спортисти; смернице