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Signs of athletic adaptation on the electrocardiogram (ECG)

Az edzettség szív-adaptációnak jelei nyugalmi EKG-n

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Absztrakt: A 12-elvezetéses elektrokardiogram (EKG) fontos információt szolgáltat a szív elektromos aktivitásáról, amely egyben képet adhat a szívizom átalakulásáról (remodeláció), beleértve a szívbetegségeket is. A szívizom átalakulása azonban nem csak kóros lehet, hanem fiziológiás folyamat is. Ebbe tartozik a szív sporttal kapcsolatos átalakulása a sportszív kialakulása is. Azon egyénekben, akik magas intenzitású, rendszeres fizikai aktivitást végeznek, olyan EKG jelek kialakulására is lehet számítani, amik hasonlítanak a kóros EKG jelekhez. Ezek a "sport-EKG" jelek a következők lehetnek: sinus bradycardia, bizonyos atrioventrikulárisblokkok, inkomplett jobbTawara szárblokk, bal kamra megvastagodás izolált EKG kritériumai, valamint a korai repolarizáció (amit gyakran akut szívinfarktusként értékelnek). Vannak azonban olyan EKG jelek, amelyek betegségekre utalhatnak, ezért azonnali további kivizsgálást igényelnek. Ezek közül a legfontosabbak: jelentős ST depresszió, kóros T hullám inverzió és kóros Q hullám, interventrikuláris elektromos vezetési zavar, Wolf-Parkinson-Whiteszindróma, hosszú, vagy rövid QT, Brugadaszindróma, valamint pitvar tachiaritmiák és kamrai aritmiák és extraszisztolék. Európában az EKG értékelés része a sportolást megelőző szűrővizsgálati protokolloknak, ami viszont az Egyesült Államokban nem így van. A különbség oka az, hogy mivel a fals-pozitív EKG-k aránya sportolókban magas, az USA szűrővizsgálati protokolljaiban nem alkalmazzák az EKG-t. Ez a tény tehát rávilágít arra a problémára, hogy a sportorvosi gyakorlatban az EKG értékelése jelentős sport-kardiológiai jártasságot igényel. A normál, fiziológiás sport-adaptációs EKG jelek tehát az adaptáció mértékére utalnak, míg a potenciálisan kóros EKG jelek felismerés megelőzheti a hirtelen szívhalált. A 12-elvezetéses EKG pontos értékelése tehát nagyon fontos része a mindennapi sportorvosi gyakorlatnak.
Kulcsszavak: szívizomátépülés, sport-EKG, EKG értékelés, koronaria ischemia, szívbetegségek, hirtelen szívhalál

Abstract: Twelve-lead electrocardiogram (12-lead ECG) provides important information on the cardiac electric signals, which reflects on the myocardial remodelling in different cardiac pathologies. Remodelling, however does not only refer to a pathologic process in the heart, but may also mean physiological adaptation, including athletic heart. Individuals with regular and high intensity sport activity develop ECG signals, which may be similar to ECG patterns of cardiac patients. These "athletic ECG signals" include sinus bradycardia, certain atrioventricular blocks, incomplete right bundle branch blocks, isolated voltage criteria of left ventricular hypertrophy and early repolarisation (it's false interpretation includes acute myocardial infarction). There are, however, certain ECG signs, which may refer to cardiac disease and need immediate further investigations. These include significant ST segment depression, pathologic T-wave inversion and pathologic Q-waves, interventricular conduction abnormality, Wolf-Parkinson-White syndrome, long and short QT, Brugada syndrome, atrial tachyarrhythmia, ventricular arrhythmias and extra systole. In Europe ECG is part of pre-participation screening protocols, which is not the case in the United States. The reason of this difference and absence of ECG from the US protocols is the high number of false positive ECG-s among athletes. This fact refers to a real problem in sport medicine practice: precise interpretation of an athletic ECG needs practice in sport-cardiology. Thus, normal physiological signs on an athletic ECG refer to the extent of

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sport-adaptation and the recognition of potentially pathological signs may prevent sudden cardiac death. Therefore exact interpretations of a 12-lead ECG are important in daily sport medicine practice.
Keywords: cardiac remodeling, sport-ECG, ECG interpretation, coronary ischemia, heart diseases, sudden cardiac death

Athletic heart develops in all individual doing intensive and regular physical exercise. There are different cardiac patterns of athletic remodeling, depending on the athlete's gender, the disciple and the intensity of sport activity. Current diagnostic modalities are able to monitor the rate of cardiac adaptation. Since this remodeling develops during a slow process, annual screening of athletes gives the possibility to detect step-by-step development - or lack of development - of the different signs of athletic heart. This information may be useful for trainers in the evaluation of the athletes, especially in young age groups. Sport-cardiology examinations, including resting 12-leads ECG and transthoracic echocardiography are able to detect signs of cardiac adaptation. Although, adaptation is a physiologic process, but its signs on ECG and echocardiography may be considered as pathologic variants in non-athlete individuals. Therefore, these signs of physiologic athletic remodeling are called as "grey zones" in the literature and they need precise interpretation to prevent misleading diagnosis.

Athletic ECG

Athletic screening programs, approved by the European Society of Cardiology and the International Olympic Committee include 12-leads ECG and its precise interpretation. How did it happen, that the American Heart Association (AHA) protocol does not contain ECG? The answer is based on the high number of false positive ECGs, which are the results of wrong interpretations of the ECG-signs (Figure 1) of athletic adaptation. According to European guidelines, in Hungary ECG is part of the athletic pre-participation screening protocols. The guideline for athletic ECG interpretation is described by the Stanford criteria, which were further specified by the Seattle criteria (*Drezner* et al., 2013) and its recent revaluation (*Sheikh* et al., 2014), which became useful in the everyday practice (*Wasfy* et al., 2015). These criteria distinguish ECG signs associated with sport and signs not associated with athletic activity.

ECG signs associated with sport

Sinus bradycardia in athletes is accepted as normal in case of >30/min. In athletes, especially in those with highly intensive endurance training components, even lower heart rate may be present. Heart rate with ≤30/min or sinus arrhythmia with RR distance higher, than 3.00 milliseconds are candidates for further evaluation to exclude sick sinus syndrome (*Corrado* et al., 2010).

Atrioventricular block (Degree I and degree II Mobitz Type I) are also frequent ECG signs associated with athletic activity. Different diagnostics however, include stress test, during which sport-related blocks disappear. In athletes, with higher degree AV blocks (AV Ib. and AV degree III) immediate further evaluations are needed. (*Heidbuchel* et al., 2006). In those symptomless athletes with higher degree AV blocks, according to the AHA recommendations sport activity is allowed in the following cases: A) lack of structural heart disease, B) lack of pre-syncope or syncope, C) narrow QRS and >40/min ventricular rate with increasing heart rate at stress.

Incomplete right bundle branch block (IRBBB) is present in approximately one third of all athletes (*Drezner* et al., 2013; *Kiss* et al., 2015). Frequent signs are the R-R' morphology (rsR') in V1 lead and wide S wave in leads I and V6. QRS duration in normal athletic adaptation is 0.08-0.12 milliseconds in these cases and the presence of IRBBB does not represent cardiac pathology. However, in some disease with higher risk of sudden cardiac death, IRBBB pattern may be present and associated with other diagnostic ECG and imaging signs (e.g.: arrhytmogenic right ventricular cardiomyopathy, Brugada syndrome).

Isolated QRS voltage criteria of left ventricular hypertrophy (Cornell and Sokolow-Lyon) are not indications for further examinations (*Georgijevic* & *Andric*, 2016). In athletes however, with symptoms or positive family history of sudden cardiac death, a more detailed examination may be indicated. ECG signs of hypertrophic cardiomyopathy (HCM) are similar to those of non-pathologic left ventricular hypertrophy. Further detailed medical examinations are indicated in the following cases: A) presence of deep (>3mm) and/or >40 milliseconds q wave, except in leads III and aVR, B) presence of deep (>1mm) inverted T wave in at least two of V2-6, aVF, I, aVL leads. In children and adolescents inverted T waves are often present in leads V1-3 and in African and Caribbean athletes in leads V1-4. In patients with HCM pathologic inverted T waves are associated with ST segment depression in at least two concomitant leads. Signs on the resting 12-leads ECG, are obviously interpreted with results detected by the cardiac imaging modalities (most frequently echocardiography). In patients with an inter-ventricular septum (IVS) >15mm, HCM is likely present. Grey zone (possible athletic adaptation, but HCM cannot be excluded) is represented by the IVS thickness between 13-15mm. Left ventricular cavity size are also important in the different diagnostics between physiologic adaptation and HCM. Athletic adaptation is more likely in patients with end-diastolic left ventricular diameter of 40-60mm. In HCM patients left ventricular end-diastolic diameter does not exceed 54mm. Aerobic stress test is also a differential diagnostic tool, since low maximal oxygen uptake is present in case of HCM. In case HCM is strongly suspected, cardiac MRI or deconditioning (transient suspension of athletic activity) for 4-6 months is recommended. The images provided by the cardiac MRI provide tissue-level details of possible myocardial damage in HCM patients, while deconditioning results in a reverse remodeling in case of physiologic sport adaptation (with decreasing cavity size and myocardial thickness), but not in HCM.

Early-repolarization represents the most common signs of sport-adaptation, which is present in 50-80% of elite athletes (*Cappato* et al., 2010). This appears mostly by ST segment

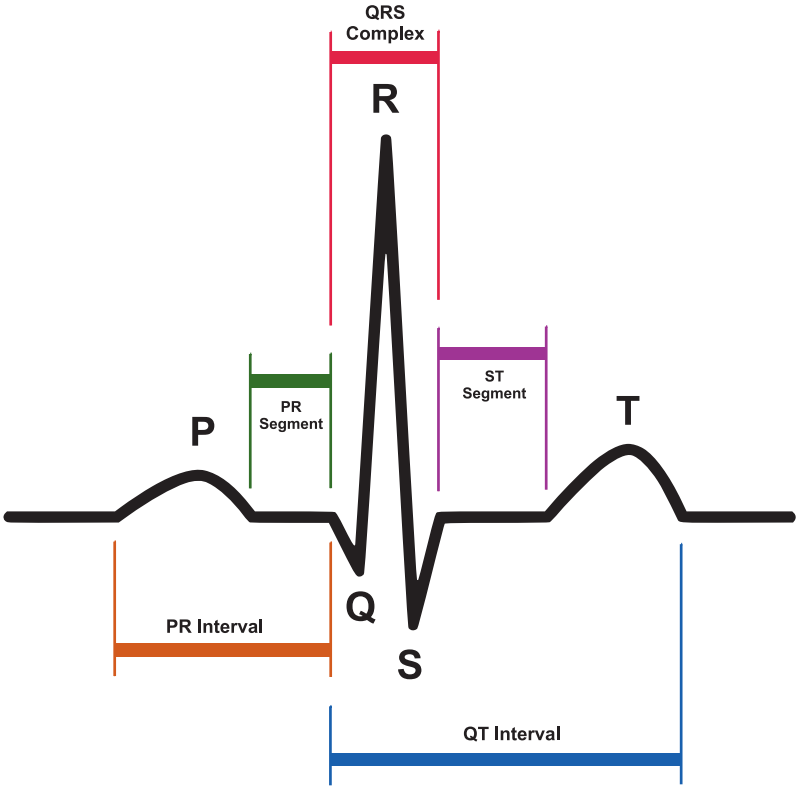

Figure 1. Schematic representation of a normal ECG curve
Arrows represent duration of the cardiac electric signals in time. Standard recording speed is 25mm/second.

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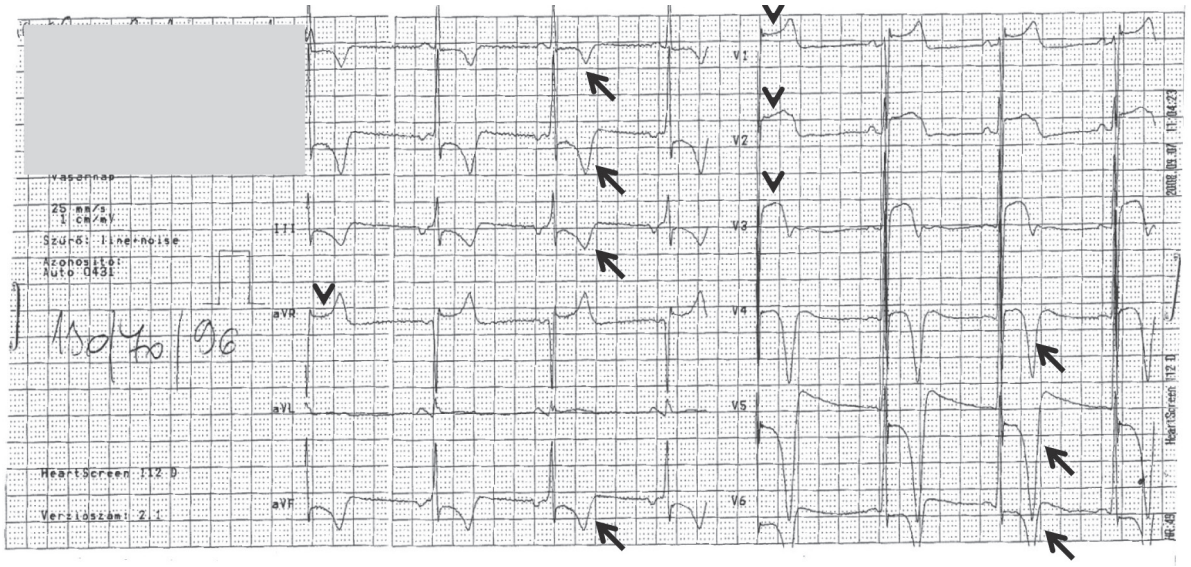

Figure 2. ECG of an African-origin 25 years old elite soccer player
J point elevation may reflect on early repolarization (arrowhead). Deep inverted T waves (arrows), however suggest cardiac pathology, or even structural heart disease. Despite the bizarre ECG, only myocardial hypertrophy, without pathology was confirmed in this athlete.

elevation (J point elevation, ascending ST segment elevation), mainly in pre-cordial leads (Figure 2). It is most common in leads V3-4, but it is frequently seen in leads V2, V5, V6, I, aVL, II and III. In healthy Caucasian athletes ST segment elevation is followed by positive T wave, but in black athletes ST segment elevation, followed by negative T wave is often present in leads V2-4. In athletes without symptoms these signs do not require further examinations, but at the presence of symptoms (palpitation, angina) further diagnostics are needed. Broadening of the terminal part of the QRS complex is also often seen in elite athletes. This sign is more often detected in athletes following resuscitation, therefore this sign, in contrast to "simple ST segment elevation" may reflect on cardiac pathology, especially in athletes with symptoms.

ECG signs without sport-relation

ST segment depression (>0.5mm), T wave inversion (≥2mm) or pathologic Q waves in at least two concomitant leads always suggest cardiac pathology and further examinations are definitely needed (Figure 3). Inverted, but not deep (<2mm) T waves are rather frequent signs and may disappear at deconditioning. Inverted T waves in adults, in all leads, except V1 may suggest cardiac pathology. ***Inter-ventricular conduction abnormality*** (broad QRS) in case of >140 milliseconds need further evaluation (stress-test, 24 hours ECG, imaging modalities). Left bundle branch block (LBBB) morphology with a duration of >120 milliseconds QRS, especially in association with inverted QRS complex in lead V1 need further evaluation. The prevalence of ***Wolf-Parkinson-White syndrome*** in athletes is similar to that in the total population (0.1-0.3%), but the susceptibility of the development of malignant ventricular tachycardia is higher in athletes. Its sign on the resting ECG include delta wave, short PR interval and wide QRS complex. Short PR interval without delta wave may also be pathologic (Lown-Ganong-Levine syndrome, or structural heart disease). In athletes with symptoms, but even in symptomless athletes further evaluation tests and catheter-ablation therapy is recommended. Following successful ablation therapy competitive sport activity is not limited by cardiac reasons. ***Long QT*** on ECG may be acquired (bradycardia, metabolic or electrolyte deficiencies), or congenital. Congenital long QT syndrome is resulted by mutations in five different genes. In case of LQ1, LQ2 and LQ5 mutations in a potassium channel protein gene, in LQ3 and LQ4 in a sodium channel protein gene represent the etiology. Clinical symptoms and genetic background shows a strong association. In cases with potassium channel mutations (these are

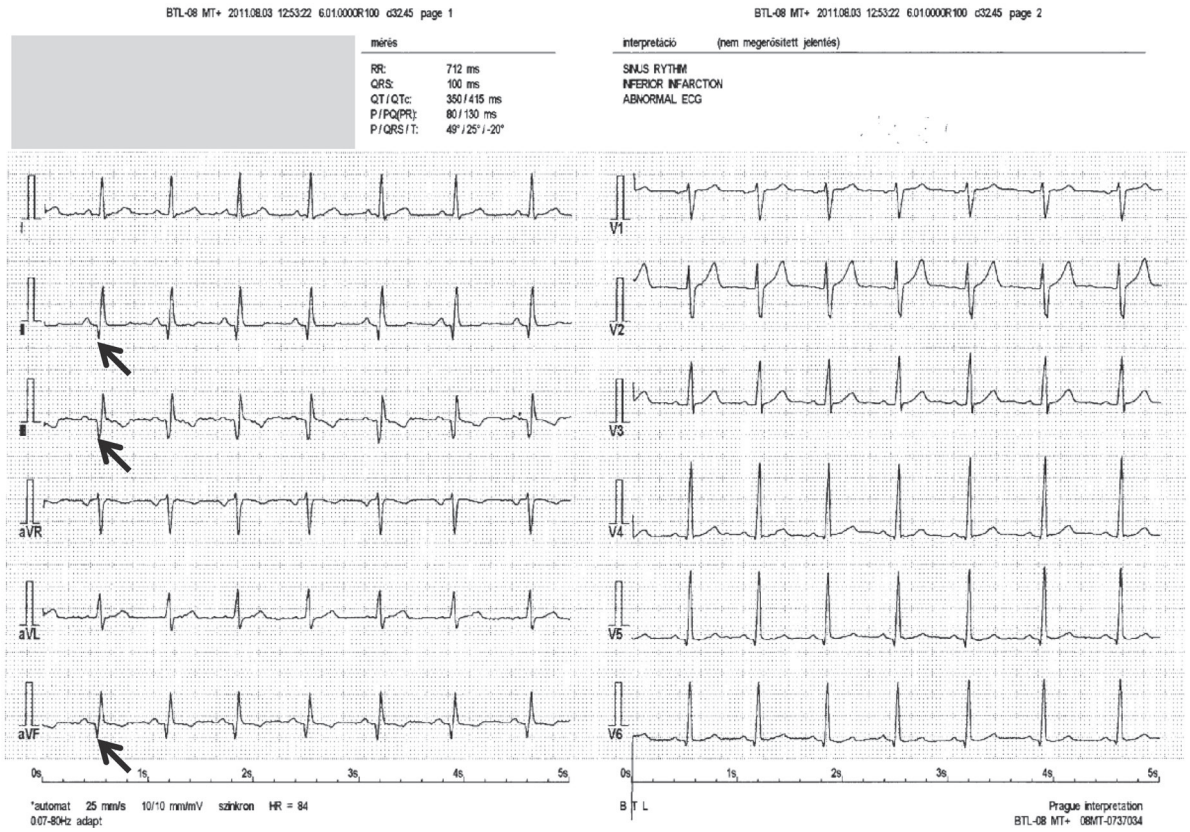

Figure 3. ECG of a Caucasian-origin 36 years old handball player. Athlete does not have any symptom

Patient was referred to the sport-cardiology unit because of his age and strongly positive family history for sudden death. Q waves (arrows) detected in the inferior leads indicated invasive coronary angiography examination. Severe 2-vessel disease has been confirmed, followed by percutaneous intervention.

more frequent) syncope or sudden cardiac death results following physical or emotional stress, but in cases with sodium channel mutations adverse events and malignant arrhythmias occur during rest (mainly during sleep). Mutations in the same sodium channel genes results in Brugada syndrome (see later), which may result in sudden death similarly at rest. The diagnosis of long QT duration is based on the corrected QT interval. In men a corrected QT duration of 470-500 milliseconds and in women 480-500 milliseconds represent a "grey zone" and may suspect pathology. In case corrected QT is >500 milliseconds, long T syndrome is strongly suspected, further evaluations are needed and competitive sport is forbidden until the final diagnosis. ***Short QT interval*** (≤320 milliseconds) is mainly the result of metabolic and electrolyte disturbances (e.g. hyperthermia, acidosis, use of anabolic steroids) and its therapy is based on the correction of the etiology factor.

Brugada syndrome is an autosomal dominant channelopathy, which may result in monomorphic or polymorphic ventricular tachycardia, ventricular fibrillation and concomitant sudden death. ECG presentation of the disease include IRBBB, elevated (>2mm), followed by descending pattern of the ST segment and inverted T waves in leads V1-3. These are the clear Type I signs of the disease, in Type 2 and 3 ECG patterns biphasic or positive T wave may be present. Definite diagnosis of the disease is based on resting ECG, symptoms (sudden death, syncope, palpitations) and a positive ajmalin-test. Ajmalin is a sodium channel blocker and in Brugada patients its administration results in the appearance of Type 1 ECG signs and/or ventricular tachycardia. Although Brugada syndrome is a rather rare disease (incidence: 1: 2.000-5.000), its importance is emphasized by the fact, that in elite athlete IRBBB and early repolarization may be present with Brugada-like

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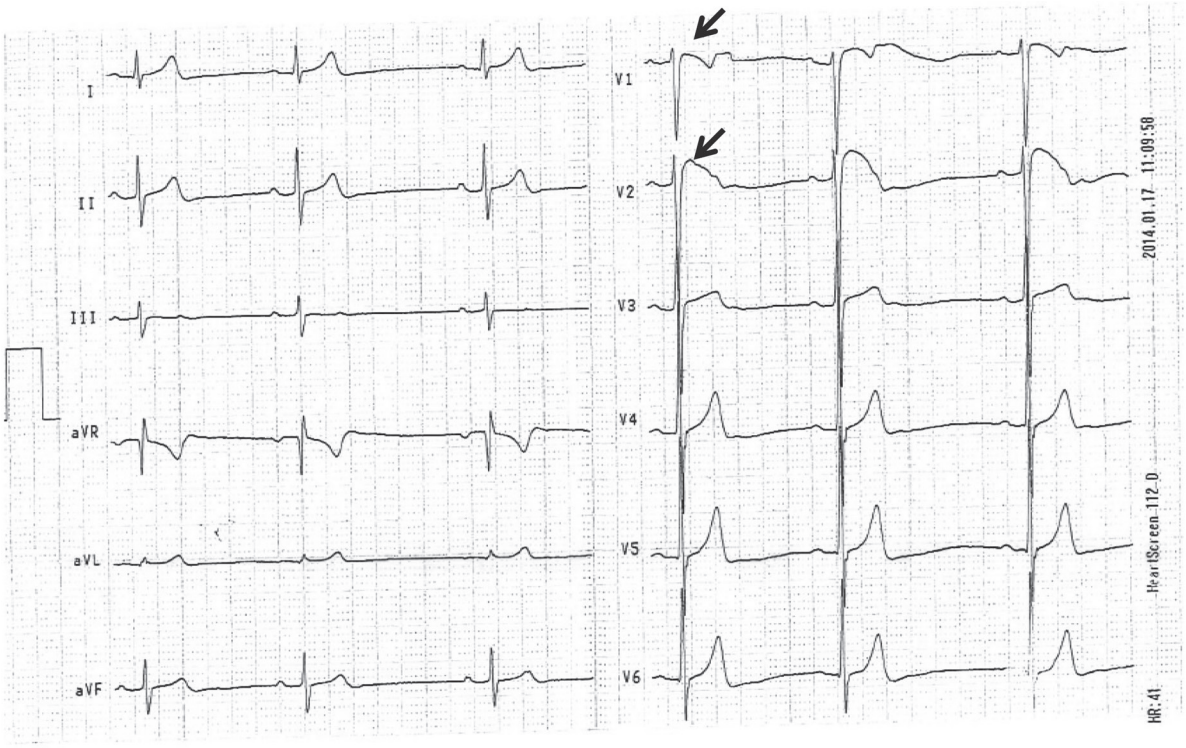

Figure 4. Latin-origin 22 years old symptomless polo player
Brugada-like ECG pattern in leads V1-2 (arrows) reflect on early repolarization. This ECG pattern, however indicate further evaluation, including ajmalin-test.

ECG pattern (Figure 4) (*Visser* et al., 2016). ***Atrial tachyarrhytmias, ventricular arrhythmias and extrasystole*** definitely need further medical tests in athletes (*Kiss* et al., 2015). In paroxysmal supraventricular tachycardia cases (AVNRT, AVRT, atrial tachycardia) ablation therapy is needed and if it is followed by a three month symptomless period, elite athletic activity is not limited from medical reasons. In atrial fibrillation with normal ventricular rate, without structural heart disease or heart failure, elite sport activity can be licensed. Its limitation is the type of sport, since contact-sports are not recommended for anticoagulated patients. In patients with sporadic ventricular extrasystole, without structural heart disease, if A) ventricural extrasystole is rare (<2.000 extrasytole/ 24 hours) and B) it is not getting more frequent or does not cause symptoms at stress, competitive sport can be licensed. In athletes with sustained ventricular tachycardia or symptoms, ablation therapy is recommended.

Borderline variants are described in the recently refined athletic ECG recommendation (*Sheikh* et al., 2014). The borderline variants may reflect on pathology, but only in cases, when at least two of them are present. These include: left atrial enlargement, right atrial enlargement, left axis deviation, right axis deviation, right ventricular hypertrophy, T wave inversion up to V4 in black athletes.

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