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CASE REPORT

ST domed Elevation – an Atypical Variant of Early Repolarization in the White Athlete after the COVID-19 Pandemic. A Case Report.

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ABSTRACT

Background: Interpreting the electrocardiography (ECG) recording in young athletes is challenging for the sports doctor. Changes in intraventricular conduction and early ventricular repolarization observed during sports training are not always confirmed in the literature. We test healthy people and look for irregularities in their ECG records. The study aims to draw attention to ST-Elevation (STE) as a variant of the White Athlete's heart norm, with a frequency lower than an inverted T wave (TWI), but also as an effect of early repolarization of the ventricles in young athletes. Is it physiology?

Case report: 14-year-old football player, examined for five years at a sports clinic, cardiology checked every year. Changes like symptomatic adult ECGs with STE and myocardial necrosis were suspected during our observation. Echocardiography, stress test good. A simple response to exercise is to adapt the athlete's heart by ischemia, preconditioning for the heart muscle, and a reversible remodelling that disappears after the sports period. We focus on changes in ECG, which are observed in athletes under 14 years of age, while most of the ECG classifications in an athlete were described above 16 years of age.

Conclusion: the variant observed in our White Athlete is an example of several similar repolarization changes in ECG not yet classified as physiological or resulting from intensive training.

Key words: ST-Elevation; early repolarization; ECG criteria; athletes

Introduction

Playing sports professionally increases the risk of sudden death by 2.5 times in individual age groups.[1] Each interpretation of heart conduction disturbances in young athletes is challenging for the doctor examining the ECG before admission to competitive sport.[2,3,4] As a standard, each time an athlete visits, an assessment for sudden arrhythmic death syndrome (SADS) is performed.[5] However, the ECG record and its interpretation are several times more valuable in diagnosing pathology than other data on the history of the disease of most asymptomatic young athletes.[6] For ten years, the recognition of training-dependent (physiological) changes in the ECG, along with differences in the record (changes not related to training), enables effective prevention of sudden death in sports.[3,4,7] It is assumed that 90% of changes in ECG are associated with adaptive *remodelling* of the heart muscle, e.g. sinus bradycardia, atrioventricular block stage I, incomplete right bundle branch block (IRBBB), early repolarization (ER), isolated signs of hypertrophy left ventricle (LVH).[8]

Even simple computer software that supports ECG find irregularities in the record and presents them in the study printout has a massive advantage over other studies.[1,6] What if the test does not detect anything? Furthermore, can the change visible in the patient's ECG be considered a borderline abnormality (the intermediate zone between the normal and abnormal ECG, the so-called minor abnormalities) or a missing element in the cardiological criteria allowing to play sports?

Revision of the Seattle criteria from 2012,[4] which are the basis for the assessment of ECG in athletes, based on a comparison of both indices (Seattle and cardiological ESC), among others, notes the RSR syndrome (QRS > 120 ms) in the anterior precordial leads as a unique feature of abnormalities but not entirely pathological.[2] Moreover, in the case of T wave inversions (TWI), the most common ECG abnormalities in athletes, he suggests returning to the Seattle criteria, i.e. extending TWI to other leads such as V2-V6, II and aVF or I and aVL (excludes III, aVR and V1).[9] Specific criteria appearing individually in both the Seattle criteria and the recommendations of the European Cardiac Society (ESC) are not a significant risk factor: right ventricular hypertrophy (RVH), the pathological axis of the right (RAD) and left (LAD) heart or enlargement of both atria.[2] They were proposed in the Refined Criteria[4] as minor importance, typical for an athlete's heart. The diagnosis of the RSR pattern QRS complex in V1 is justified due to the possibility of diagnosing the Brugada syndrome.[10] Departing from the top of the R` wave (RSR`) of the isoelectric line with β (beta) angle open to the ST segment, we recognize the type 1 Brugada pattern. The clinical syndrome described with this QRS pattern is characterized by sudden cardiac death.[11] For V2 and V3, similarly, the presence of β departure angle of over 45 degrees in the RSr` or rSR` syndrome may speak for the Brugada syndrome type 2. Brugada syndrome type 2 is sporadically seen in adolescent athletes. However, the β angle is < 30%, a safety marker.[10] The most practical tests confirming (safely) the disease (Brugada syndrome) also include re-recording the ECG from leads V1 and V2 parasternal, two intercostal spaces higher, i.e. in the second intercostal space. The lack of the same changes in ECG suggests a variant of the norm in clinical observation.[12]

However, in clinical practice, in most cases, changes are observed that are not indexed in ESC and Seattle in the ECG record, such as various types of intraventricular conduction disturbances (e.g., rSR, RSr, rSr` etc.) and disturbances of early repolarization of the V2-V6 ventricles: concave (smiling) but also domed (convex) as a variant of the STE norm.[7] I omit findings: *notching* and *slurring*, as typical for benign early repolarization in people trained over 16 years in the lateral and inferior walls.[13] In the Seattle criteria, the absent ST depression appeared in the review by Sheikh et al.[4] as ST depression ≥ 0.5 mm in two adjacent leads. It is believed that the current indexed criteria for diagnosing abnormalities in ECG in athletes apply to people over 16 years of age, while in the case of TWI changes, the most common (mild variant) is even from the age of 14.[2]

In adults, ECG changes, considered mild in athletes, raise a well-founded concern about cardiovascular disease.[1] STE domed elevation (frequent V2, V3-shaped variables) usually in precordial V1-V4 leads with TWI are a variant of the pathology diagnosed as proximal occlusion of the left anterior descending (LAD) branch, suggesting a threatening anterior myocardial infarction.[14] They are called Wellens syndromes types 1 and 2.[15] However, a prevalent benign lesion is the concave ST elevations (so-called smiling) in leads V2-V6 with arched high T wave, suggesting benign early repolarization of the ventricular muscle in athletes (standard variant).[16,17] In 2012, Smith introduced the criteria for the diagnosis/calculation based on 3 or 4 parameters (Smith Index) of V2-V4 leads of the LAD stenosis risk price (cellular application: ECG_SMITH), which helped me many times in the hospital emergency department on call.[18]

A typical feature of benign early ventricular repolarization is a high J-point departure in precordial, anterior (V2-V4), lateral (V5-V6, I, aVL), and lower (II, aVF) leads in the Afro-American race (Black Athletes).[17] They were not included in the Seattle and ESC criteria, although it was once suggested that they might constitute a risk of sudden death (Haissaguerre's syndrome).[19,20] On the other hand, a high J-point departure and a high T point are also considered an early pathology of the coronary vessels – de Winter pattern.[21] However, here, an explicit criterion for diagnosing this variant is the location of the J-point, which is significantly below the isoelectric line in the case of typical pathology. In the case of the standard variant for early ventricular repolarization in athletes, the J-point is elevated by 1-3 mm in precordial leads as a tooth of the descending QRS arm, mentioned earlier (notching) or as a slurring, which is J-wave syndrome.[22,23] In children and adolescents, there is no correlation between early repolarization features and the duration of exercise or the type of sports discipline.[23,24]

The described case is a boy, 14-years-old, a football player since 2015, who is routinely inspected annually at a sports clinic. Each visit consists of a subjective assessment (interview from the child and parent/trainer) and physical examination (anthropometric tests, general evaluation of fitness and flexibility, clinical tests, e.g., physical fitness, such as exercise grading (Harvard tests), additional tests (blood, urine)). A training interview was conducted, and an assessment of functioning outside of training suggests problems with various organs and physiological systems. The patient was under the control of a cardiologist for at least five years due to innocent heart murmurs (2/6 of Levine). In the examination, the echocardiography (echo) of the heart – the left ventricle pseudo-chord. Heart dimensions and flows are standard in the heart, with no additional abnormalities. They are permitted annually to practice sports without any reservations. The survey collects a family history of the burden of acute and chronic diseases. A critical component is the evaluation of the ECG series, especially in the case of the evolution of changes in subsequent records in the patient's documentation, year after year.

The local bioethics committee agreed to the research. The consent of the patient/examined person and his guardian to participate in the research was obtained.

Case Report

The analysis covered the entire period of observation in the clinic for several years. For four consecutive years, the ECG evaluation showed no pathology. In the previous study, attention was paid to developing domed STE in leads V2-V3, suggesting abnormalities. The patient's excellent clinical condition made it clear that there were no obstacles to practising sports.

Figures 1-4 show the ECG changes' evolution (2018, 2019, 2021, 2022). In 2020, there was no study due to the SARS-CoV-2 (COVID-19) virus pandemic. We see the transition o f early repolarization ECG changes during five years of athlete's observation, from 9 to 14. V1 lead is not essential in this case (RSr`/RS pattern).

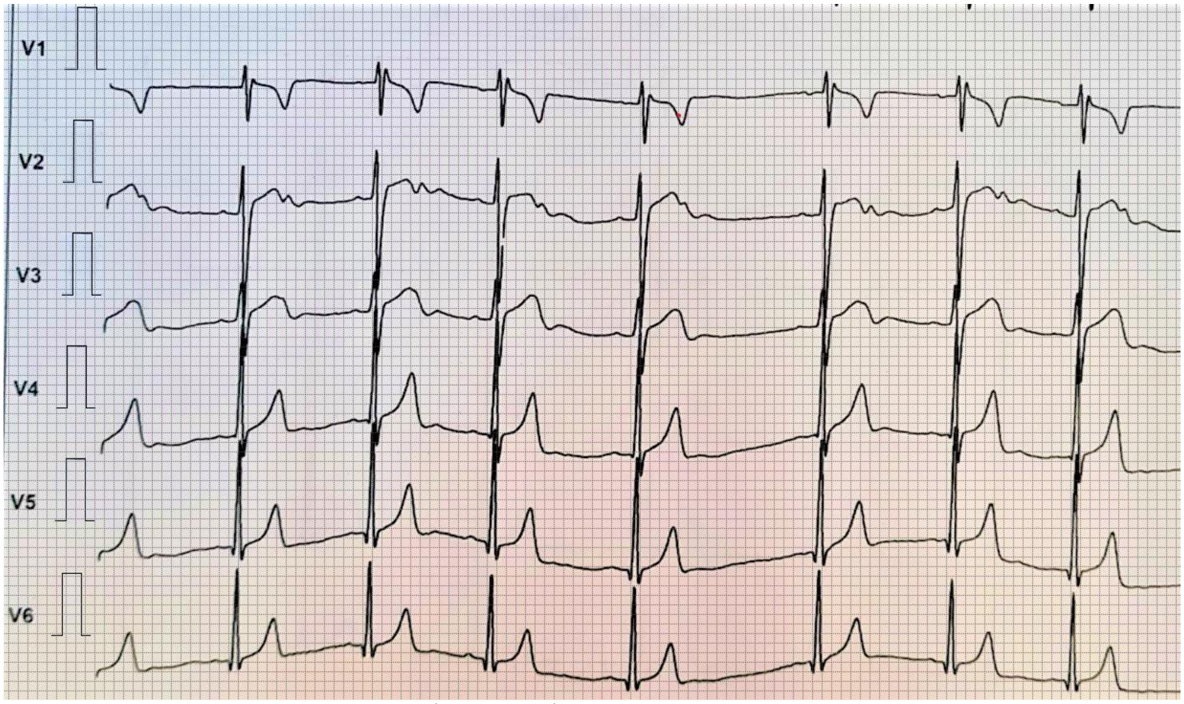


Figure 1. ECG leads V1-V6; 25 mm/s, 10 mm/mV; **2018**. STE convex J-shape in V2 and V3 leads with TWI (+TWI). STE concave in V4 lead.

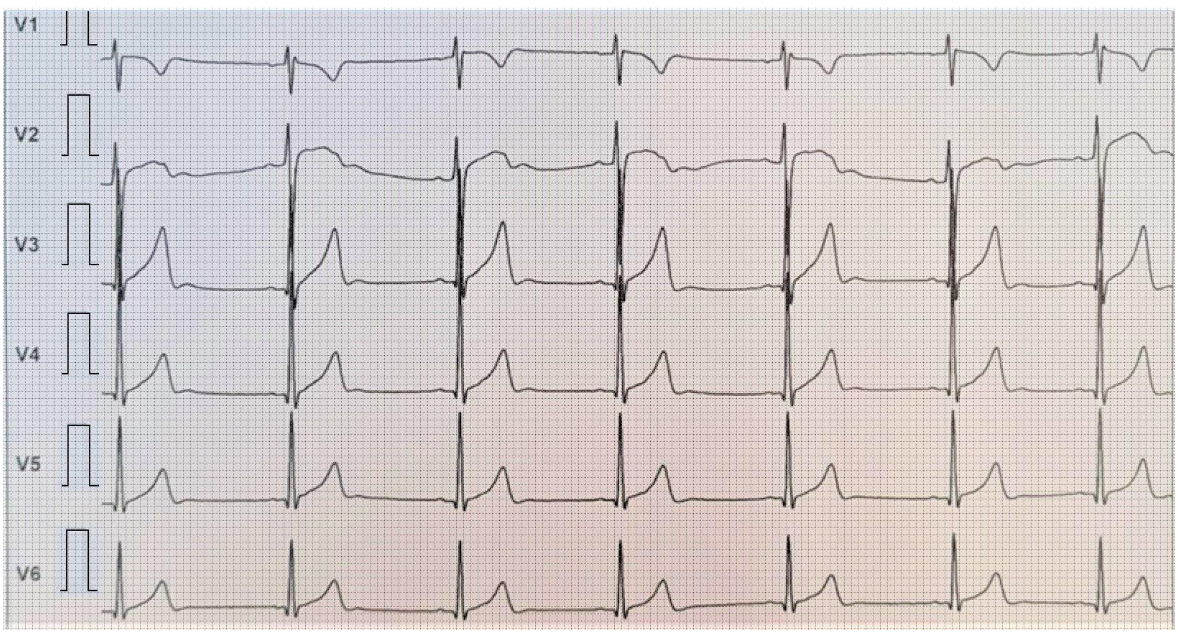


Figure 2. ECG leads V1-V6; 25 mm/s, 10 mm/mV; **2019.** STE convex J-shape in V2 lead with TWI (+TWI). STE concave in V3 lead with the smiling sign V3-V6 with J-point elevation.

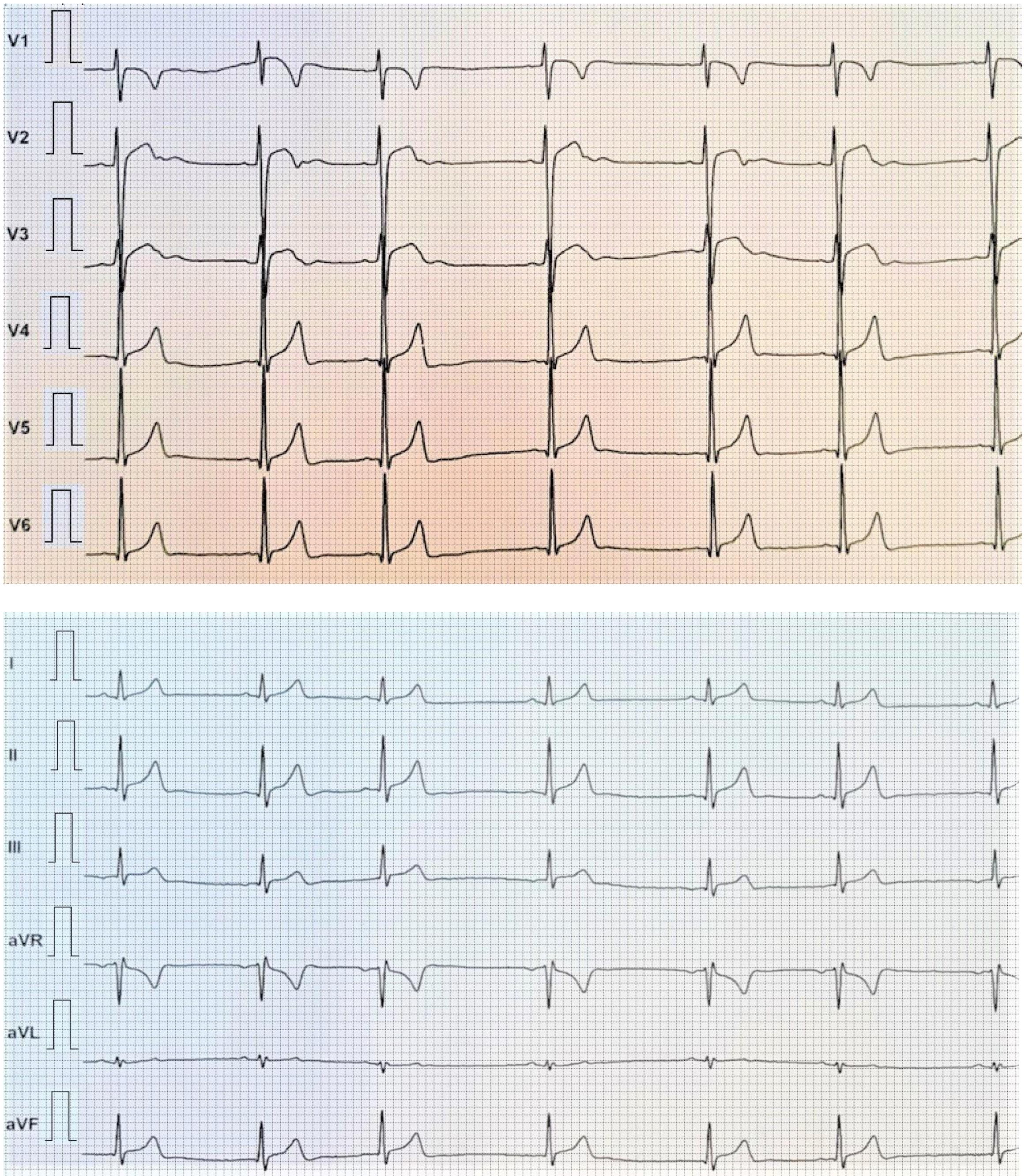


Figure 3. ECG leads V1-V6; 25 mm/s, 10 mm/mV; **2021**. STE convex J-shape in V2 and V3 leads with TWI (+TWI). STE concave in V4-V6 leads with the smiling sign.

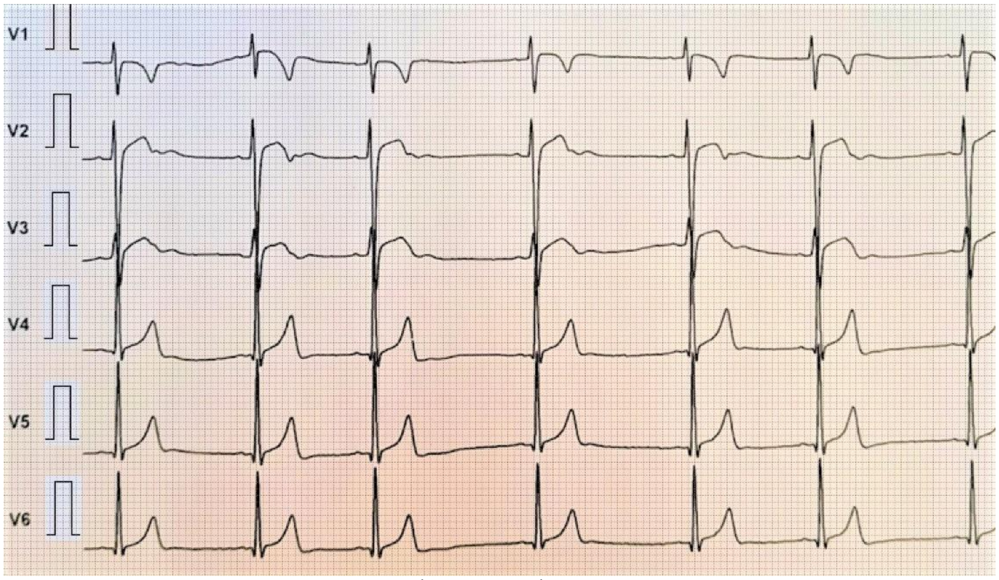
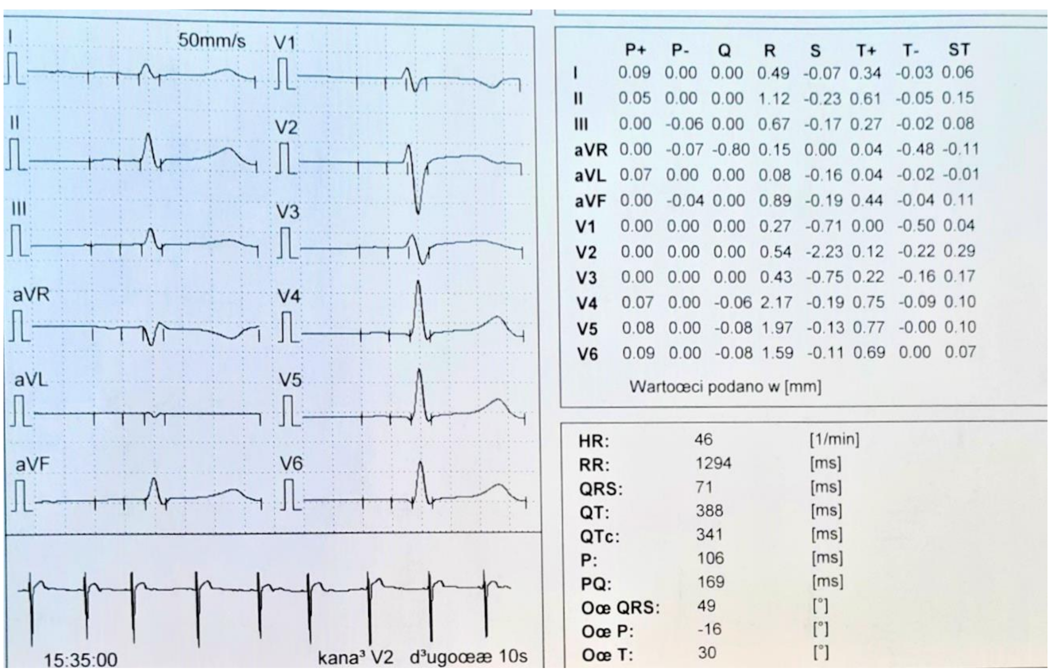


Figure 4. A ECG 12 leads; 25 mm/s, 10 mm/mV; **2022**. STE convex J-shape in V2 and V3 leads with TWI (+TWI). STE concave in I, II, III, aVF, V4-V6 leads with the smiling sign. A heart rate of 46/min is typical for extensive training on athletes` hearts.



	P+	P-	Q	R	S	T+	T-	ST
I	0.09	0.00	0.00	0.49	-0.07	0.34	-0.03	0.06
II	0.05	0.00	0.00	1.12	-0.23	0.61	-0.05	0.15
III	0.00	-0.06	0.00	0.67	-0.17	0.27	-0.02	0.08
aVR	0.00	-0.07	-0.80	0.15	0.00	0.04	-0.48	-0.11
aVL	0.07	0.00	0.00	0.08	-0.16	0.04	-0.02	-0.01
aVF	0.00	-0.04	0.00	0.89	-0.19	0.44	-0.04	0.11
V1	0.00	0.00	0.00	0.27	-0.71	0.00	-0.50	0.04
V2	0.00	0.00	0.00	0.54	-2.23	0.12	-0.22	0.29
V3	0.00	0.00	0.00	0.43	-0.75	0.22	-0.16	0.17
V4	0.07	0.00	-0.06	2.17	-0.19	0.75	-0.09	0.10
V5	0.08	0.00	-0.08	1.97	-0.13	0.77	-0.00	0.10
V6	0.09	0.00	-0.08	1.59	-0.11	0.69	0.00	0.07

Wartoœci podano w [mm]

HR:	46	[1/min]
RR:	1294	[ms]
QRS:	71	[ms]
QT:	388	[ms]
QTc:	341	[ms]
P:	106	[ms]
PQ:	169	[ms]
Oœ QRS:	49	[°]
Oœ P:	-16	[°]
Oœ T:	30	[°]

Figure 4. B Morphological analysis parameters of 12 lead patient ECGs by a computer program in a sports clinic.

It should remember that the high risk of an athlete's death before age 35 is caused primarily by cardiac arrhythmia or cardiomyopathy, which most often reveals congenital disabilities of the conductive system, which is rarely the cause of comorbid disease—the analysis of conduction disturbances observed in V1-V4 in people before 14 years. Side effects during competitive sports can be significantly reduced.

We will not achieve such commitment and burden in a young athlete in clinical tests as in competitions. What we see in the clinic is an example of adaptation to physical exertion. During sports, the heart physiologically outgrows and adjusts to the *preload* and *afterload*. We only see a simple answer to the exercise:

- Athlete's heart adaptation to exercises.
- Preparation of the heart muscle to intensive stress training (*preconditioning*).
- Reversible rebuild that disappears after sports training (*remodelling*).

Discussion

These changes in ECG, which we focus on, are observed in athletes younger than 14 years of age, while most of the ECG classifications in an athlete are described above 16 years.

Although the variation of the J-point curve with the STE in V2-V4 is significant, the three teams can be separated from each other: convex, concave, and smiling sign. As mentioned earlier, ST elevations and convexities suggest an anterior wall infarction, Prinzmetal's angina, or left ventricular aneurysm. In the case of pathologic concave elevation, it is often SADS risk, a picture of the early repolarization syndrome (ERS), but it can also be a picture of myocarditis.

Our clinical case is the need to draw attention to the lack of guidelines for the interpretation of ECG in young people training sports and a request to supplement the data on the variants of STE in the ECG criteria both in the Seattle revision and the new European guidelines (ESC).

So far, the benign early repolarization (BER) criteria ("high take-off" or "J-point elevation") have been concave STE at least 1 mm in two adjacent V2-V5 but not always together with elevation of the inferior and lateral walls. Our case concerns the anterior wall of the heart. Undoubtedly, it can also be classified as an early repolarization after excluding other causes of STE on the anterior wall. As can be seen in the record from the previous individual years of the athlete, the formation of a convex STE (domed) occurs over the front wall in one lead.

Moreover, as mentioned above, the ST convex elevation co-occurring with the inversion of the T wave (+TWI) V1-V4 is an overall picture of the Black race. It is a concern variant of the norm in the Seattle criteria,[2] although it also occurs with a lower frequency in the White race, but is not included in the standard.[17] Ilodibia et al.[25] pointed out that 77% of all athletes are ER and STE with TWI (+TWI), and almost 30% are West Africans (Nigeria, Ghana, Algeria).

The Black race most often presents ER, LVH, TWI, and STE, in contrast to the White[17]. STE is 11-85% in Black meta-analyzes and 1-61% in White.[17,25] On this basis, we propose to include the anterior precordial leads (summarized in Table 1).

Abnormalities	+TWI	Leads	J-shape	Type	Ethnicity
STE ≥1 mm	common	V1-V4	convex	normal	Black Athletes
	rare	V2-V6	concave	normal	White Athletes
	atypical	V2-V3	convex	borderline	

Table 1. STE early repolarization evaluation proposition in the anterior precordial leads for White Athletes. A rare variant, typical concave STE in V2-V6 (with the smiling sign). Atypical variant convex (domed) STE in V2-V3 as a borderline. +TWI: with T wave inversion in precordial leads.

Conclusion

A revision of the ECG criteria for admission to competitive sports, known as the Seattle criteria,[4] has revealed that there is no reference to the STE section of the White Athlete guidelines.[26] The literature analysis describing similar cases previously observed in other patients in the clinic suggests a mechanism of early ventricular repolarization[16]. It is a physiological mechanism of the heart's adaptation to exercise load[27]. Changes in ECG are visible during training, and after quitting the sport, the ECG recording usually normalizes, which seems to be related to myocardial *remodelling*[23].

The purpose of the short report is to draw attention to the dynamic changes in ECG records in athletes, sometimes difficult to interpret as a result of training. The lack of correlation with the clinical condition, cardiac echo examination, and clinical tests in the patient raises the question of including another change in the Seattle criteria suggesting the physiological stress on the myocardium during exercise in the Caucasian race.

Informed Consent Statement

Written informed consent has been obtained from the patient(s) parents to publish this paper.

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Conflict of interest

None declared.

Data availability statement

The data supporting this study's findings are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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