https://doi.org/10.47183/mes.2025-345

SIGNIFICANCE OF QT INTERVAL PROLONGATION IN YOUTH AND ADOLESCENT SPORTS (LITERATURE REVIEW)

Leonid M. Makarov✉, Vera N. Komoliatova, Anush G. Akopyan, Dmitryi A. Besportochnyi, Alena V. Dmitrieva, Irina I. Kiseleva, Ilya V. Zyabkin

Federal Scientific and Clinical Center for Children and Adolescents of the Federal Medical and Biological Agency, Moscow, Russia

Introduction. Up to 42% of young athletes who die suddenly show no signs of known cardiac diseases at autopsy (such as cardiomyopathies, myocarditis, or congenital heart defects). However, molecular genetic analysis in young sudden death victims identifies mutations in genes responsible for Long QT Syndrome (LQTS) in 17–23% of cases. During comprehensive medical examinations (CME) of young athletes in Russian national teams within the FMBA of Russia system, up to 24% of athletes are disqualified from sports due to detected QT interval prolongation for further diagnosis, with the diagnosis of LQTS being confirmed in 0.24% of cases.
Objective. Analysis of diagnostic methods for LQTS in young athletes and criteria for their clearance for sports training.
Discussion. The pathogenesis of LQTS is based on genetically determined impairment of cardiac ion channel function, which causes myocardial electrical instability predisposing to cardiac events. Such events include Torsades de Pointes (TdP) ventricular tachycardia, syncope, cardiac arrest, and sudden cardiac death (SCD). Diagnosis of LQTS is based on the Schwartz criteria, which incorporate data from standard ECG (QTc > 450 ms), Holter monitoring (HM), stress tests, clinical presentation, and family history. A score of more than three points based on these criteria makes the diagnosis of LQTS highly probable. According to international criteria for QT interval assessment in adult athletes, the proposed upper limits of normal QTc duration are up to 470 ms for males and up to 480 ms for females. Some authors suggest that QTc values up to 500 ms may be acceptable in athletes; however, according to the Schwartz criteria, this value is sufficient to confirm the diagnosis of LQTS. Bradycardia, typical of trained athletes, is another LQTS criterion in pediatric ECG assessment. Methods for QT assessment during bradycardia in young athletes are not specified. Intense training may increase QT interval duration; conversely, temporary detraining may lead to its decrease. The Schwartz criteria based on HM results include only T-wave alternans and TdP tachycardia. QT interval assessment during HM remains a subject of debate. To date, at least 17 pathogenic genes responsible for LQTS have been identified. Detection of Class IV–V pathogenic mutations is sufficient for diagnosing LQTS, regardless of QT duration. The issues of clearance/return to sport-specific training for athletes with LQTS remain controversial, being addressed differently across countries. There are known cases of athletes with LQTS who have achieved significant success in sport competitions, as well as regular occurrences of SCD in young individuals with this condition. Current Russian and international guidelines state that competitive sports are contraindicated for patients with confirmed LQTS.
Conclusions. The assessment of the QT interval in young athletes involves numerous methodological and clinical peculiarities distinct from those in non-athletic individuals. Underestimating these peculiarities can lead to over- or under-diagnosis of LQTS, thereby potentially creating a life-threatening situation for the athlete. Individual risks in different categories of LQTS patients are composed of multiple components. Disqualification from sports does not eliminate the risk of cardiac events in LQTS; at the same time, the extent to which sports activity itself increases these risks remains unknown today. This underscores the relevance of actively studying and clarifying these unresolved issues in young athletes with QT interval prolongation and LQTS.

Keywords: youth and adolescent sports; QT interval; long QT syndrome; sudden cardiac death

For citation: Makarov L.M., Komoliatova V.N., Akopyan A.G., Besportochnyi D.A., Dmitrieva A.V., Kiseleva I.I., Zyabkin I.V. Significance of QT interval prolongation in youth and adolescent sports (literature review). *Extreme Medicine*. 2026;28(1):79–87. https://doi.org/10.47183/mes.2025-345

Funding: the study was carried out without sponsorship.

Potential conflict of interest: the authors declare no conflict of interest.

✉ Leonid M. Makarov dr.leonidmakarov@mail.ru

Received: 8 July 2025 **Revised:** 17 Sep. 2025 **Accepted:** 9 Oct. 2025 **Online first:** 29 Oct. 2025

УДК 616.1

ЗНАЧЕНИЕ УДЛИНЕНИЯ ИНТЕРВАЛА QT В ДЕТСКО-ЮНОШЕСКОМ СПОРТЕ (ОБЗОР ЛИТЕРАТУРЫ)

Л.М. Макаров✉, В.Н. Комолятова, А.Г. Акопян, Д.А. Беспорточный, А.В. Дмитриева, И.И. Киселева, И.В. Зябкин

Федеральный научно-клинический центр детей и подростков Федерального медико-биологического агентства, Москва, Россия

Введение. До 42% юных спортсменов, умерших внезапно, не имели каких-либо признаков известных болезней сердца при аутопсии (кардиомиопатий, миокардитов или пороков сердца), а молекулярно-генетический анализ, проведенный у молодых внезапно погибших лиц, выявляет в 17–23% случаев мутации в генах, отвечающих за синдром удлиненного интервала QT (СУИQT). При проведении углубленных медицинских обследований (УМО) у юных спортсменов сборных РФ в системе ФМБА России до 24% атлетов отводятся от спорта из-за выявленного удлинения интервала QT для уточнения диагноза и в 0,24% случаев диагноз СУИQT подтверждается.
Цель. Анализ методов диагностики удлинения интервала QT у юных спортсменов и критериев их допуска к спортивным тренировкам.
Обсуждение. В основе развития СУИQT лежит генетически детерминированное поражение функции ионных каналов кардиомиоцитов, вызывающее электрическую нестабильность миокарда, которая предрасполагает к возникновению сердечных событий: желудочковой тахикардии типа «пируэт» (Torsade de Pointes — TdP), синкопе, остановке сердца и внезапной сердечной смерти (ВСС).

Для диагностики СУИQT используются критерии Шварца, в которые входят данные стандартной ЭКГ (QTc более 450 мс), холтеровского мониторирования (ХМ), стресс-тестов, клинической картины, семейного анамнеза. Выявление более 3 баллов по этим критериям делает диагноз СУИQT высоковероятным. Согласно международным критериям по оценке интервала QT у взрослых спортсменов, нормой продолжительности интервала QTc предлагают считать у мужчин значения QTc до 470 мс, а у женщин — до 480 мс. Некоторые авторы предлагают считать допустимыми значения QTc у спортсменов до 500 мс, но по критериям Шварца этого достаточно для подтверждения диагноза СУИQT. Брадикардия, типичная для тренированных спортсменов, также является одним из критериев СУИQT при оценке ЭКГ у детей. Методы оценки QT при брадикардии у юных спортсменов не уточнены. Интенсивные тренировки могут увеличивать продолжительность интервала QT, а временный детренинг, наоборот, уменьшать. В критерии Шварца по результатам ХМ включены только альтернация зубца Т и тахикардии TdP. Оценка интервала QT при ХМ является предметом дискуссий. На сегодняшний день известно не менее 17 патогенных генов, отвечающих за формирование СУИQT. Выявление патогенных мутаций IV–V класса достаточно для постановки диагноза СУИQT, независимо от продолжительности QT. Вопросы допуска/возвращения в спорт спортсменов с СУИQT остаются дискуссионными и решаются в разных странах неодинаково. Известны как спортсмены с СУИQT, добившиеся значительных успехов в спорте, так и регулярные случаи ВСС у молодых людей с этим заболеванием. Существующие на сегодняшний день отечественные и международные рекомендации говорят о противопоказании для занятий соревновательным спортом больным с доказанным СУИQT.

Выводы. В оценке интервала QT у юных спортсменов существует много методических и клинических особенностей, в отличие от лиц, не занимающихся спортом. Их недооценка может привести к гипер- или гиподиагностике СУИQT и создать угрозу для жизни спортсмена. Индивидуальные риски у разных категорий больных СУИQT слагаются из многих компонентов. Отвод от спорта не устраняет риск развития сердечных событий при СУИQT, но насколько сам спорт увеличивает риски, сегодня неизвестно. Это определяет актуальность активного изучения и уточнения данных неопределенных вопросов у юных спортсменов с удлиненным интервалом QT и СУИQT.

Ключевые слова: детско-юношеский спорт; интервал QT; синдром удлиненного интервала QT; внезапная сердечная смерть

Для цитирования: Макаров Л.М., Комолятова В.Н., Акопян А.Г., Беспорточный Д.А., Дмитриева А.В., Киселева И.И., Зябкин И.В. Значение удлинения интервала QT в детско-юношеском спорте (обзор литературы). *Медицина экстремальных ситуаций*. 2026;28(1):79–87. https://doi.org/10.47183/mes.2025-345

Финансирование: исследование проведено без спонсорской поддержки.

Потенциальный конфликт интересов: авторы заявляют об отсутствии конфликта интересов.

✉ Макаров Леонид Михайлович dr.leonidmakarov@mail.ru

Статья поступила: 08.07.2025 **После доработки:** 17.09.2025 **Принята к публикации:** 09.10.2025 **Online first:** 29.10.2025

INTRODUCTION

Electrocardiographic (ECG) changes constituting the “athlete’s heart” phenotype are predominantly studied in adult athletes. A limited number of studies are dedicated to ECG findings in adolescent athletes engaged in competitive sports [1]. The QT interval is one of the most critical parameters in the ECG assessment of young athletes, since its pathological prolongation serves as a marker for the risk of dangerous ventricular arrhythmias (“red flag”) necessitating mandatory further investigation and clarification of its underlying causes [2, 3].

The clinical significance of QT interval prolongation in athletes is determined primarily by its association with the problem of sudden cardiac death (SCD). Up to 42% of young athletes who experience a sudden death show no signs of known cardiac diseases at autopsy (such as cardiomyopathies, myocarditis, or congenital heart defects) [4]. Molecular genetic analysis performed in young sudden death victims without obvious cardiac disease identified at autopsy or in their relatives reveals mutations in genes responsible for long QT syndrome in 17–23% of cases [5, 6].

Long QT syndrome (LQTS) is a condition with a high risk of life-threatening cardiac events. These include polymorphic Torsade de Pointes (TdP) ventricular tachycardia, syncope, cardiac arrest, and sudden cardiac death (SCD). The disease is based on a genetically determined impairment of cardiac ion channel function (a hereditary or congenital channelopathy), which causes electrical instability of the cardiac cell and the entire myocardium, predisposing to the occurrence of cardiac events [7]. QT interval prolongation on ECG is detected in 24% of young athletes during comprehensive medical examinations (CME) at the elite sports mastery level within the FMBA of Russia system, leading to temporary or permanent disqualification from sports to clarify its nature and rule out LQTS [8]. In 0.24% of cases, the LQTS diagnosis is confirmed based on clinical electrocardiographic criteria and molecular genetic studies [9], which raises new questions regarding potential risks, clearance for sports participation, and the necessity of treatment.

This article carries out a review of methods for diagnosing QT interval prolongation in young athletes and the criteria for their clearance for sports training.

MATERIALS AND METHODS

The literature search was conducted in electronic bibliographic databases in the Russian (eLibrary, CyberLeninka) and English (Web of Science, Scopus, PubMed) languages. The search depth was 21 years. Articles published in medical scientific journals and specialized medical monographs relevant to the research topic were analyzed. The criteria for source selection

and inclusion in the review were based on search queries using the following keywords: QT interval, sports, young elite athletes, long QT syndrome, acquired long QT syndrome, clearance for sports.

RESULTS AND DISCUSSION

LQTS was first described in 1957 by Norwegian physicians A. Jervell and F. Lange-Nielsen [10]. They observed a family with six children, four of whom had congenital sensorineural deafness and episodes of loss of consciousness; subsequently, three of them died suddenly. The current prevalence of LQTS is estimated to be one in 2000–2500 individuals, making it one of the most common arrhythmogenic channelopathies [11].

The Schwartz criteria [12, 13] are used worldwide for diagnosing LQTS (Table 1). These criteria incorporate data from standard ECG, Holter monitoring (HM), stress tests, clinical presentation, family history, and examination of first-degree relatives. A score of more than three points on these criteria, in the absence of secondary causes for QT prolongation, makes the diagnosis of LQTS highly probable. The identification of a pathogenic gene with pathogenicity Class IV–V is also sufficient for establishing the diagnosis of LQTS, regardless of the QT interval duration [13].

According to the clinical interpretation based on [12, 13], a total score of ≤ 1 points indicates a low probability of LQTS; a total score of 1–3 points indicates an intermediate probability of LQTS; a total score of ≥ 3 points indicates a high probability of LQTS.

The primary criterion for diagnosing the condition remains the detection of a prolonged QT interval on a standard 12-lead resting ECG. Methodologically, the QT interval measurement is recommended to be performed in standard lead II or precordial lead V5, where it is considered most representative [14]. Since the QT interval duration is dependent on heart rate (HR), several formulas exist for heart rate correction of the QT interval.

Clinical medicine, including sports cardiology, primarily uses the Bazett formula [15]:

$$QTc\ (ms) = QT\ (ms) / \sqrt{RR\ (s)}, \quad (1)$$

where QTc — corrected QT interval value in milliseconds (ms); QT — measured QT interval value in milliseconds (ms); RR — RR interval value in seconds (s), preceding the measured QT interval.

For children, a normal QTc interval value on a standard ECG is considered to be less than 440 ms [16]. For adult males, a QTc value of less than 450 ms is proposed as the norm, while for females, it is less than 460 ms [17]. Several studies suggest the preferential use of the Fridericia formula for QTc assessment in athletes due to employing a cubic root rather than a square root [18]. In the study [19] involving a large cohort of 1473 adolescents aged 7–15 years engaged in sports, the QTc interval calculated using the Bazett formula was 412 ± 25 ms, whereas the QT interval corrected using the Fridericia formula was 387 ± 21 ms. The authors concluded that the Fridericia formula is a more accurate method for assessing the QT interval duration in young athletes. However, despite the criticism of QTc assessment using the Bazett formula, precise reference values for QTc calculated using alternative formulas have not been established for young athletes, thus requiring further research [20].

According to current international criteria for assessing the QT interval in adult elite athletes, the proposed upper limits of the Bazett-corrected normal QTc interval are 470 ms for males and 480 ms for females [15]. Some studies in athletes suggest that QTc values up to 500 ms may be considered acceptable and non-hazardous [21, 22]. However, according to the Schwartz criteria (Table 1), the detection of a QTc interval in the range of 460–480 ms already contributes three points to the LQTS diagnostic score and is assessed as indicating an

Table 1. Schwartz criteria for diagnosis of long QT syndrome

Electrocardiographic Criteria	Score
QTc > 480 ms	3
QTc > 460–479 ms	2
QTc > 450–459 ms (males)	1
QTc ≥ 480 ms at the 4th min of recovery during stress testing (cycle ergometry, treadmill)	1
Torsade de Pointes tachycardia	1
Macroscopic T-Wave Alternans	1
Bifid T Wave in at least three leads	1
Bradycardia (age-appropriate)	0.5
Clinical criteria	
Stress-induced syncope	2
Syncope without stress	1
Congenital deafness	0.5
Documented family history of LQTS	1
Family history of unexplained sudden death in relatives under 30 years of age	0.5

Table compiled by the authors based on data from the sources [12, 13]

Note: QTc — corrected QT interval value in milliseconds (ms), calculated by the formula QTc (ms) = QT (ms)/√RR (s); QT — measured QT interval value in milliseconds (ms); RR — RR interval value in seconds (s), preceding the measured QT interval.

intermediate probability of LQTS (Table 1). This discrepancy in interval assessment between clinical and sports cardiologists creates difficulties in reaching a diagnostic conclusion. In addition, as studies have shown, fewer than 50% of cardiologists and fewer than 40% of other physicians who interpret ECGs are capable of correctly measuring and assessing the QT interval [23].

Expert opinions on the impact of athletic training on the QT interval duration are equivocal. In a study by Małek et al. following ECG screening of 600 healthy Caucasian children (aged 5–17 years) [24], only the development of bradycardia, sinus arrhythmia, incomplete right bundle branch block, and early repolarization pattern were considered. However, Caramoci [25] reviewed 20 published studies and found that several of them confirmed a longer QT interval in young athletes compared to non-athletic individuals at the same heart rate. In another large-scale study of automated ECG data from 672 young athletes aged 17–22 years compared to 6534 non-athletes, a lower heart rate and a longer QT interval were observed. However, these athletes had a significantly shorter QTc interval = 409 (384–426) ms, calculated using the Bazett formula, versus 428 (411–445) ms in non-athletes [26]. This result may be attributable to the lower QTc values produced by this specific formula in the context of bradycardia [27]. Clinical observations have shown that intense training may increase the QT interval, whereas temporary detraining may shorten its duration [25, 28]. All researchers emphasize the need for heightened vigilance in cases of QT prolongation in athletes and the necessity of ruling out LQTS [29].

The Schwartz diagnostic criteria for LQTS include QTc assessment at the 4th minute of recovery after a stress test (Table 1). Other criteria for QT evaluation have been proposed in [30–32]; however, these criteria have not been validated in athlete cohorts. Holter monitoring (HM) is also used in the diagnosis of LQTS according to the Schwartz criteria. However, based on HM results, the Schwartz criteria include only the documentation of Torsades de Pointes tachycardia and macroscopic T-wave alternans. The measurement and interpretation of the QT interval itself during HM remain a subject of debate. Some authors recommend measuring the QT interval at a stable heart rate of 60 bpm without correction [31], while others propose QT assessment at minimum and maximum heart rates [32, 33]. In addition, some approaches suggest using an average daily QTc greater than 450–460 ms as a diagnostic criterion [33–35]. Such studies have not thus far been conducted in athletes.

Evaluation of QT dynamics based on calculating the linear regression coefficient between RR and QT intervals (Slope QT/RR) is an additional method for QT interval assessment during HM in athletes [36]. QT dynamics reflects the adaptation of the QT interval to changes in heart rate. In healthy individuals, the normal linear regression coefficient (Slope QT/RR) ranges 0.16 ± 0.02 for men and 0.20 ± 0.04 for women [37]. Trained healthy athletes are characterized by lower Slope QT/RR values: 0.13 ± 0.02 for men and 0.16 ± 0.03 for women [37]. In physiological terms, lower Slope QT/RR values are defined as QT hypo-adaptation. This implies that under an increase in heart rate, the corrected QT interval (QTc) prolongs beyond the normal range; under a decrease in heart rate, it fails to shorten to normal values [36]. Conversely, QT hyper-adaptation (high Slope QT/RR values) leads to significant QT interval shortening at higher heart rate values and excessive prolongation under lower heart rate values [38]. QT hyper-adaptation is typical under conditions of increased sympathetic influence on heart rhythm in newborns [39], in patients with type 3 long QT syndrome (LQT3), and in chronic heart failure [39], where cardiac events occur at rest, during sleep, or immediately after physical exertion in athletes [27]. This suggests possible gene-specific individual risks associated with sports participation in patients with LQTS.

Bradycardia, typical of trained athletes, is one of the LQTS criteria included in the Schwartz score with a specific diagnostic weight of 0.5 points. This item is considered only in children and adolescents, where heart rate norms have stricter age-specific limits [16]. On the other hand, bradycardia is common in elite young athletes; as a result, when calculating QTc using the Bazett formula, it can yield a false-negative result, showing normal QTc values despite actual QT prolongation [27]. The most recent international recommendations for ECG interpretation in athletes [15] suggest performing an ECG after mild exercise in cases of bradycardia below 60 bpm, although not mentioning specific types of exercise. Viskin [40] proposed recording an ECG in the orthostatic position to increase heart rate and assess QTc during bradycardia. This study was conducted in adult non-athlete patients, and its methodology is often criticized [30].

To date, at least 17 pathogenic genes responsible for LQTS have been identified [11–13] (Table 2).

The three most frequently occurring molecular genetic variants of LQTS are: Type 1 (LQT1) and Type 2 (LQT2), which are associated with mutations in the *KCNQ1* and *KCNH2* genes, respectively, governing potassium channel function. Type 3 (LQT3) is associated with mutations in the *SCN5A* gene, which regulates sodium channel function in cardiomyocytes (Table 2). These three variants account for approximately 80% of all LQTS patients [11–13].

The genetic variant of LQTS can often be suspected based on a characteristic ECG pattern. For patients with LQT1, the ECG typically shows a broad T-wave. In this variant, the primary triggers for cardiac events are physical exertion, water contact, and swimming. For individuals with LQT2, the ECG is characterized by a bifid T-wave, and a typical trigger for cardiac events in this case is a sudden loud sound. In women, the first episodes often occur after childbirth. Finally, for the LQT3 variant, the ECG typically shows a late-onset T-wave

Table 2. Genetic variants of long QT syndrome

Clinical syndrome	Molecular genetic variant of LQTSe	Inheritance pattern	Locus	Ion channel	Defective gene	Lethal arrhythmia
LQTS (RW)	LQT1	AD	11p15	‘KS	*KCNQ1, KvLQTI*	TdP
	LQT2		7q35	I Kr	*KCNH2, HERG*	
	LQT3		3p21	‘Na	*SCN5A, Nav1.5*	
	LQT4		4q25		*ANKB, ANK2*	
	LQT5		21q22	‘KS	*KCNE1, minK*	
	LQT6		21q22	I Kr	*KCNE2, MiRP 1*	
Anderson–Tawil Syndrome	LQT7		17q23	IK1	*KCNJ2, Kir 2.1*	
Timothy Syndrome	LQT8		6q8A	‘ca-L	*CACNA1C, Cav1.2*	
	LQT9		3p25	‘Na	*CAV3, Caveolin-3*	
	LQT10		11q23.3	‘Na	*SCN4B. Navb4*	
	LQT11		7q21-q22	K	*ARAP9*	
	LQT12		3p25	Na	*Cav3a*	
LQTS (JLN)		AR	11p15	‘KS	*KCNQ1, KvLQTI*	
			21q22	‘KS	*KCNE1, minK*	

Table compiled by the authors based on data from the sources [11–13]

Note: LQTS — long QT syndrome; RW — Romano–Ward syndrome; JLN — Jervell and Lange-Nielsen syndrome; AD — autosomal dominant inheritance; AR — autosomal recessive inheritance; TdP — Torsades de Pointes polymorphic ventricular tachycardia.

with a prolonged ST segment [12]. Cardiac events in LQT3 occur more frequently during sleep, at rest, or immediately after cessation of physical exercise [11–13].

In 25–50% of individuals with an identified pathogenic mutation causing LQTS, a normal QT interval duration is observed on the baseline ECG [11–13]. In addition to congenital forms of LQTS, the QT interval duration can be prolonged by the use of certain medications, various metabolic disturbances, and the adoption of various diets (which is often the case in sports) [41, 42]. Secondary QT prolongation in athletes can be no less dangerous than congenital forms and cause life-threatening cardiac events as well.

The first step in the management of patients with QT interval prolongation is the exclusion of secondary causes (discontinuation of QT-prolonging medications, correction of potential electrolyte imbalances, etc.). In patients with confirmed LQTS, the current guidelines recommend avoiding genotype-specific potential triggers for cardiac events (e.g., physical exertion and sports, supervising children around water for Type 1, avoiding sudden loud noises for Type 2, etc.) [11–13].

The cornerstone of pharmacological therapy for all LQTS patients (even asymptomatic ones) is beta-blocker administration. The most effective agents in preventing arrhythmic risk are non-selective beta-blockers lacking intrinsic sympathomimetic activity, such as nadolol and propranolol [11–13]. In Russian clinical practice, atenolol is widely used [43, 44]. When prescribing these medications to athletes, it is crucial to remember that they are prohibited in certain sports.[1] For patients with the Type 3 molecular genetic variant of LQTS (mutation in the *SCN5A* gene), sodium channel blockers (such as mexiletine, flecainide, and allapinin) can be used [11–13, 43].

The second stage of treatment (or in cases where beta-blockers are contraindicated) for patients with symptomatic LQTS involves considering the possibility of implantable cardioverter-defibrillator (ICD) placement [11–13]. ICD implantation is recommended as a method for secondary prevention of SCD, in addition to beta-blocker therapy, for patients who have experienced cardiac arrest [11–13]. The issue of eligibility for competitive sports participation in patients with implanted antiarrhythmic devices has not yet been definitively resolved [45]. Left cardiac sympathetic denervation (LCSD) is another therapeutic option under consideration [46]. This procedure is performed to eliminate asymmetric

[1] Independent National Anti-Doping Organization RUSADA. https://rusada.ru/substances/prohibited-list/

sympathetic innervation of the heart, which is a proven arrhythmogenic factor in these patients.

The issues of clearance/return to sports activities for athletes with LQTS remain controversial and are addressed differently across countries. Leading UK sports cardiologists Basavarajaiah and Sharma [47] published a case report of a 16-year-old female long-distance runner, a member of the national team. Her training volume was 18 h per week. During pre-participation screening (which had apparently not been conducted previously), her resting ECG revealed sinus bradycardia of 38 bpm, a QT interval prolonged to 620 ms, and a QTc of 530 ms. Molecular genetic analysis identified a mutation typical of Type 1 LQTS (LQT1). Her 13-year-old brother, who was actively engaged in football, was also evaluated as part of family screening; his ECG showed a QTc prolonged to 520 ms and the same genetic mutation. Both athletes were disqualified from sports participation with a recommendation to initiate beta-blocker therapy.

In Type 1 LQTS, the primary trigger for fatal arrhythmias is physical exertion, and one of the key criteria in risk stratification is a QTc prolongation >500 ms. Both factors were present in the siblings, yet no cardiac events had occurred prior to their evaluation. These and similar observations have likely shaped the following perspective on clearance for competitive sports in athletes with LQTS, being supported by leading global sports cardiologists [48]. For individuals with a positive phenotype and a QTc interval prolonged beyond 490 ms, competitive sports are prohibited; only routine daily activities (leisure-time activity) are permitted, with avoidance of genotype-specific cardiac event triggers (e.g., swimming, sudden loud noises). For asymptomatic carriers of LQT1 mutations, competitive sports may be possible provided swimming/diving is avoided. For carriers of LQTS mutations who lack the disease phenotype (i.e., no ECG changes or clinical manifestations), competitive sports are permissible. The required scope of specific evaluation includes a thorough history, ECG, exercise stress testing, 24-h HM, genetic testing, and regular annual follow-up [48]. However, reaching a final decision regarding sports participation in LQTS often remains challenging in practice.

Thus, during an international consultation regarding the management of a 16-year-old asymptomatic professional athlete (hockey) with confirmed Type 3 Long QT Syndrome (LQT3) and a QTc interval prolongation greater than 500 ms, the opinions of the aforementioned experts on continuing the athletic career were divided [49]. S. Sharma, Head of the Sports Cardiology Clinic at St. George's University (London, UK), expressed an opinion in favor of clearing the athlete for competitive sports. His argument was based on the fact that the QT interval in LQT3 shortens during physical exertion. However, A. Pelliccia, Head of the Sports Cardiology Clinic at the Institute of Sports Medicine (Rome, Italy), provided a conclusion stating that the athlete was at high risk for cardiac events, with a verdict contraindicating participation in competitive sports [49]. A. Moss, Founder and Director of the International Long QT Syndrome Registry (Rochester, USA), also expressed the opinion that this athlete was at high risk for cardiac events and required an implantable cardioverter-defibrillator (ICD). He stated that the decision regarding the young athlete's participation in competitions should be made individually with his parents after they have been fully informed of all potential risks [49].

On the other hand, there are known athletes with LQTS who have achieved significant success in sports. For instance, Dana Vollmer, a member of the U.S. swimming team with a confirmed diagnosis of LQTS, won two gold medals at the 2012 London Olympic Games (and has a total of 32 medals from various major international competitions).[2] The details of her medical history and the specific variant of the syndrome are unknown; however, this case underscores the importance of a highly individualized approach to clearance of elite athletes for sports activity.

Johnson and Ackerman from the Mayo Clinic (USA) published the results of a follow-up study of a cohort comprising 130 young patients with LQTS who participated in competitive sports at various levels [50]. The patients received standard LQTS therapy with beta-blockers; some underwent implantable cardioverter-defibrillator (ICD) placement and/or left cardiac sympathetic denervation (LCSD). Not a single patient died during the seven-year follow-up period.

In the study by Aziz [51], 103 patients with confirmed LQTS who were engaged in sports (predominantly recreational) were observed. No episodes of syncope or SCD were recorded during the follow-up period. Two patients experienced ICD discharges, but these were not associated with athletic activity. On the other hand, despite some optimism from these observations, the number of athletes with this condition who have died suddenly also remains significant [52, 53]. Consequently, the current Russian official guidelines state that competitive sports are unequivocally contraindicated for patients with confirmed LQTS [54]. However, the actual risks for different categories of LQTS patients are highly individual and depend on numerous components, including clinical presentation, sex, ECG markers, specific genetic mutation characteristics, and other, still poorly understood, factors [55].

CONCLUSION

We conducted a review of studies focusing on QT interval assessment methods in young athletes, peculiarities of LQTS diagnosis, and issues of clearance for sports training and established that evaluation of the QT interval in athletes has a number of methodological

[2] Dalessio J. Olympian Dana Vollmer swims through heart risk. *Huffpost*. 2012. https://www.huffpost.com/entry/dana-vollmer-heart-condition_n_1711515

and clinical specific features, distinct from ECG interpretation in individuals not engaged in regular competitive sports. Underestimation of these features can lead to both overdiagnosis and unjustified disqualification from sports, as well as underdiagnosis of LQTS, which may result in a failure to identify a dangerous condition for the athlete's life.

These issues become particularly critical when making decisions about disqualifying young athletes at the elite performance level. Achieving this level has consumed virtually the athlete's entire prior life; sports represent their life's choice and profession. Disqualification from sports does not eliminate the risk of cardiac events in LQTS; at the same time, the extent to which sports activity itself increases these risks remains unknown. This uncertainty underscores the urgency of studying and clarifying the unresolved questions regarding the evaluation, management, and permissible level of athletic activity in young athletes with QT interval prolongation and LQTS.

References

1. Ragazzoni GL, Cavigli L, Cavarretta E, Maffei S, Mandoli GE, Pastore MC, et al. How to evaluate resting ECG and imaging in children practising sport: a critical review and proposal of an algorithm for ECG interpretation. *European Journal of Preventive Cardiology.* 2023;30(5):375–83. https://doi.org/10.1093/eurjpc/zwac218
2. Corrado D, Pelliccia A, Bjørnstad HH, Vanhees L, Biffi A, Borjesson M, et al. Cardiovascular pre-participation screening of young competitive athletes for prevention of sudden death: proposal for a common European protocol. Consensus Statement of the Study Group of Sport Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology. *European Heart Journal*. 2005;26(5):516–24. https://doi.org/10.1093/eurheartj/ehi108
3. Zeppilli P, Biffi A, Cammarano M, Corrado D, Cavarretta E, Cecchi F, et al. Italian Cardiological Guidelines (COCIS) for Competitive Sport Eligibility in athletes with heart disease: update 2024. *Minerva Medica.* 2024;115(5):533–64. https://doi.org/10.23736/s0026-4806.24.09519-3
4. Finocchiaro G, Papadakis M, Robertus JL, Dhutia H, Steriotis AK, Tome M, et al. Etiology of Sudden Death in Sports: Insights from a United Kingdom Regional Registry. *JACC.* 2016;67:2108–15. https://doi.org/10.1016/j.jacc.2016.02.062
5. Wong LC, Roses-Noguer F, Till JA, Behr ER. Cardiac evaluation of pediatric relatives in sudden arrhythmic death syndrome: a 2-center experience. *Circulation. Arrhythmia and Electrophysiology.* 2014;7(5):800–6. https://doi.org/10.1161/CIRCEP.114.001818
6. Behr ER, Dalageorgou C, Christiansen M, Syrris P, Hughes S, Tome Esteban MT, et al. Sudden arrhythmic death syndrome: familial evaluation identifies inheritable heart disease in the majority of families. *European Heart Journal.* 2008;29(13):1670–80. https://doi.org/10.1093/eurheartj/ehn219
7. Schwartz PJ, Crotti L, Insolia R. Long-QT syndrome: from genetics to management. *Circulation. Arrhythmia and Electrophysiology.* 2012;5(4):868–77. https://doi.org/10.1161/circep.111.962019
8. Makarov LM, Komoliatova VN, Aksenova NV. Analysis of the reasons for exemptions from sports among young elite athletes. *Russian Bulletin of Perinatology and Pediatrics.* 2020;65(6):65–71 (In Russ.). https://doi.org/10.21508/1027-4065-2020-65-6-65-71
9. Makarov LM, Komoliatova VN, Kiseleva II, Aksenova NV, Besportochny DA, Balykova LA, et al. Epidemiology of syncope in children and adolescents in elite sports (study EPISODE-S). *Russian Bulletin of Perinatology and Pediatrics.* 2019;64(6):62–7 (In Russ.). https://doi.org/10.21508/1027-4065-2019-64-6-62-67
10. Jervell A, Lange-Nielsen F. Congenital deaf-mutism, functional heart disease with prolongation of Q-T interval and sudden death. *American Heart Journal.* 1957;54(1):59–68. https://doi.org/10.1016/0002-8703(57)90079-0
11. Schwartz P, Stramba-Badiale M, Crotti L, Pedrazzini M, Besana A, Tome Esteban MT, et al. Prevalence of the congenital long-QT syndrome. *Circulation.* 2009;120(18):1761–7. https://doi.org/10.1161/circulationaha.109.863209
12. Wilde AAM, Amin AS, Postema PG. Diagnosis, management and therapeutic strategies for congenital long QT syndrome. *Heart.* 2022;108(5):332–8. https://doi.org/10.1136/heartjnl-2020-318259
13. Priori SG, Wilde AA, Horie M, Cho Y, Behr ER, Berul C, et al. HRS/EHRA/APHRS expert consensus statement on the diagnosis and management of patients with inherited primary arrhythmia syndromes: document endorsed by HRS, EHRA, and APHRS in May 2013 and by ACCF, AHA, PACES, and AEPC in June 2013. *Heart Rhythm.* 2013;10:1932–63. https://doi.org/10.1016/j.hrthm.2013.05.014
14. Lepeschkin E, Surawicz B. The measurement of the QT interval of the electrocardiogram. *Circulation*. 1952;6;378–88. https://doi.org/10.1161/01.CIR.6.3.378
15. Sharma S, Drezner JA, Baggish A, Papadakis M, Wilson MG, Prutkin JM, et al. International recommendations for electrocardiographic interpretation in athletes. *European Heart Journal.* 2017;39:1466–80. https://doi.org/10.1093/eurheartj/ehw631
16. Makarov LM, Kiseleva II, Komoliatova VN, Fedina NN. New norms and interpretations of children's electrocardiogram. *Pediatrics named after G.N. Speransky.* 2015;94(2):63–8 (In Russ.). EDN: TUFXDJ
17. Viskin S. The QT interval: too long. too short or just right. *Heart Rhythm.* 2009;6:711–5. https://doi.org/10.1016/j.hrthm.2009.02.044
18. Orchard JJ, Orchard JW, Raju H, La Gerche A, Puranik R, Davis A, et al. Analysis of athlete QT intervals by age: Fridericia and Hodges heart rate corrections outperform Bazett for athlete ECG screening. *Journal of Electrocardiology.* 2022;74:59–64. https://doi.org/10.1016/j.hlc.2022.06.145
19. D'Ascenzi F, Anselmi F, Graziano F, Berti B, Franchini A, Bacci E, et al. Normal and abnormal QT interval duration and its changes in preadolescents and adolescents practicing sport. *Europace.* 2019;21(10):1566–74. https://doi.org/10.1093/europace/euz198
20. Gupta I, Mahendran S, Davis J, Davis A, Orchard J. A Comparison of QT Interval Correction Methods on Electrocardiograms (ECG) of Athletes and Young People: A Systematic Review. *Heart, Lung and Circulation*.

2022;31(3):S106–7. https://doi.org/10.1016/j.hlc.2022.06.142
21. Hoyte C, Terhune E, Carry P, McCanta A, Coel R. Borderline and Long QT Syndrome in Adolescent Athletes Taking Medications. *Journal of Adolescent Health.* 2015;57(2):218–21. https://doi.org/10.1016/j.jadohealth.2015.04.003
22. Basavarajaiah S, Wilson M, Whyte G, Shah A, Behr E, Sharma S. Prevalence and significance of an isolated long QT interval in elite athletes. *European Heart Journal.* 2007;28:2944–9. https://doi.org/10.1093/eurheartj/ehm404
23. Viskin S, Rosovski U, Sands AJ, Chen E, Kistler PM, Kalman JM, et al. Inaccurate electrocardiographic interpretation of long QT: the majority of physicians cannot recognize a long QT when they see one. *Heart Rhythm.* 2005;2(6):569–74. https://doi.org/10.1016/j.hrthm.2005.02.011
24. Małek ŁA, Dotka M, Brzezińska K, Pyndryk P, Materkowski M, Wiecha S. Electrocardiographic Changes in Juvenile Athletes in Relation to Sex, Age, and Sport Category. *Pediatric Cardiology*. 2025. https://doi.org/10.1007/s00246-025-03945-y
25. Caramoci A, Smaranda AM, Drăgoiu TS, Bădărău IA. ECG Screening in Athletes: A Systematic Review of Sport, Age, and Gender Variations. *Reviews in Cardiovascular Medicine.* 2025;26(5):38209. https://doi.org/10.31083/RCM38209
26. Park J, Kimata C, Young J, Perry JC, Bratincsak A. Fine Tuning ECG Interpretation for Young Athletes: ECG Screening Using Z-score-based Analysis. *Sports Medicine — Open.* 2024;10:114. https://doi.org/10.1186/s40798-024-00775-9
27. Schnell F, Behar N, Carré F. Long-QT Syndrome and Competitive Sports. *Arrhythmia and Electrophysiology Reviews.* 2018;7(3):187–92. https://doi.org/10.15420/aer.2018.39.3
28. Dagradi F, Spazzolini C, Castelletti S, Pedrazzini M, Kotta MC, Crotti L, et al. Exercise Training-Induced Repolarization Abnormalities Masquerading as Congenital Long QT Syndrome. *Circulation.* 2020;142(25):2405–15. https://doi.org/10.1161/CIRCULATIONAHA.120.048916
29. Yanık H, Değirmenci E, Büyükakılı B. A comprehensive electrocardiographic analysis for young athletes. *Medical and Biological Engineering and Computing.* 2021;59(9):1865–76. https://doi.org/10.1007/s11517-021-02401-2
30. Zareba W. Challenges of diagnosing long QT syndrome in patients with nondiagnostic resting QTc. *JACC.* 2010;55(18):1962–4. https://doi.org/10.1016/j.jacc.2010.02.018
31. Garson A. How to measure the QT interval–what is normal? *American Journal of Cardiology.* 1993;72(6):14B–16B. https://doi.org/10.1016/0002-9149(93)90034-a
32. Eggeling T, Osterhues HH, Kochs M, Beyer M, Höher M, Hombach V. Diagnostische Wertigkeit von Standard-EKG-Methoden, Kälte-Druck-Test und Valsalva-Manöver beim idiopathischen QT-Syndrom. *Zeitschrift gur Kardiologie*. 1993;82(1):1–7.
33. Makarov L, Komoliatova V, Zaklyazminskaya E, Dmitrieva A. Ambulatory ECG Monitoring in Patients with Long QT Syndrome. *Conference: Scientific Session AHA.* Chicago; 2018. https://doi.org/10.13140/RG.2.2.32275.69921
34. Page A, Aktas MK, Soyata T, Zareba W, Couderc JP. "QT clock" to improve detection of QT prolongation in long QT syndrome patients. *Heart Rhythm.* 2016;13(1):190–8. https://doi.org/10.1016/j.hrthm.2015.08.037
35. Follansbee CW, Beerman L, Arora G. Automated QT analysis on Holter monitors in pediatric patients can differentiate long QT syndrome from controls. *PACE.* 2018;41(1):50–6. https://doi.org/10.1111/pace.13244
36. Makarov LM, Komoliatova VN, Miroshnikova EN, Kazantseva MA. Physiological significance and normative parameters of rate adaptation of QT-interval during Holter monitoring in healthy persons of young age. *Kardiologiia.* 2008;48(4):54–8 (In Russ.).
37. Genovesi S, Zaccaria D, Rossi E, Valsecchi MG, Stella A, Stamba-Badiale M. Effects of exercise training on heart rate and QT interval in healthy young individuals: are there gender differences? *Europace.* 2007;9(1):55–60. https://doi.org/10.1093/europace/eul145
38. Makarov L, Komoliatova V, Zevald S, Schmidt G, Muller CA, Serebruany V. QT dynamicity, microvolt T-wave alternans, and heart rate variability during 24-hour ambulatory electrocardiogram monitoring in the healthy newborn of first to fourth day of life. *Journal of Electrocardiology.* 2010;43(1):8–14. https://doi.org/10.1016/j.jelectrocard.2009.11.001
39. Makarov L. The component of pathogenesis of sudden nocturnal death in patients with heart failure. *Extreme Medicine.* 2022;24(3):74–6. https://doi.org/10.47183/mes.2022.026
40. Viskin S, Postema PG, Bhuiyan ZA, Rosso R, Kalman JM, Vohra JK, et al. The response of the QT interval to the brief tachycardia provoked by standing: a bedside test for diagnosing long QT syndrome. *JACC.* 2010;55(18):1955–61. https://doi.org/10.1016/j.jacc.2009.12.015
41. Hoyte C, Terhune E, Carry P, McCanta A, Coel R. Borderline and Long QT Syndrome in Adolescent Athletes Taking Medications. *Journal of Adolescent Health.* 2015;57(2):218–21. https://doi.org/10.1016/j.jadohealth.2015.04.003
42. Yunisova AS, Smolensky AV. Acquired long QT interval in athletes. *Sports Medicine: Research and Practice.* 2021;11(4):17–25 (In Russ.). https://doi.org/10.47529/2223-2524.2021.4.3
43. Makarov LM, Komoliatova VN, Kiseleva II, Besportochnyi DA, Dmitrieva AV, Akopyan AG, et al. *Long QT syndrome is a disease with a high risk of sudden death.* Moscow: PH "Medpraktika-M"; 2018 (In Russ.).
44. Makarov LM, Komoliatova VN, Zaklyazminskaya EV, Besportochnyi DA, Akopyan AG, Islanov IO, et al. 2 Cases of New Electrocardiographic Patterns in Patients with Long QT Syndrome. *JACC: Case Reports.* 2025;30(15):103753. https://doi.org/10.1016/j.jaccas.2025.103753
45. Pelliccia A. Long QT syndrome, implantable cardioverter defibrillator (ICD) and competitive sport participation: when science overcomes ethics. *British Journal of Sports Medicine.* 2014;48(15):1135–6. https://doi.org/10.1136/bjsports-2013-092441
46. Schwartz PJ, Priori SG, Cerrone M, Spazzolini C, Odero A, Napolitano C, et al. Left cardiac sympathetic denervation in the management of high-risk patients affected by the long-QT syndrome. *Circulation.* 2004;109(15):1826–33. https://doi.org/10.1161/01.CIR.0000125523.14403.1E
47. Basavarajaiah S, Sharma S. (2009). A 16-Year-Old Female Runner with Prolonged QT Interval. *Sports Cardiology Casebook.* London: Springer; 2009. https://doi.org/10.1007/978-1-84882-042-5_13
48. Pellicia A, Heidbuchel H, Corrado D, Sharma S, Borjesson M. Criteria and considerations relative to safe participation in sport for athletes with cardiac abnormalities. *The ESC Textbook of Sport Cardiology.* Oxford: Oxford university press; 2019. https://doi.org/10.1093/med/9780198779742.003.0041
49. Makarov LM. *Holter monitoring.* 4-th edition. Moscow: "Medpraktika-M"; 2017 (In Russ.).
50. Johnson JN, Ackerman MJ. Return to play? Athletes with

congenital long QT syndrome. *British Journal of Sports Medicine.* 2013;47(1):28–33. https://doi.org/10.1136/bjsports-2012-091751
51. Aziz PF, Sweeten T, Vogel RL, Bonney WJ, Henderson J, Patel AR, et al. Sports Participation in Genotype Positive Children with Long QT Syndrome. *JACC Clinical Electrophysiology.* 2015;1(1–2):62–70. https://doi.org/10.1016/j.jacep.2015.03.006
52. Longo UG, Risi Ambrogioni L, Ciuffreda M, Maffulli N, Denaro V. Sudden cardiac death in young athletes with long QT syndrome: the role of genetic testing and cardiovascular screening. *British Medical Bulletin.* 2018;127(1):43–53. https://doi.org/10.1093/bmb/ldy017
53. Corrado D, Trovato D, Zorzi A. Long QT Syndrome: A Preventable Cause of Exercise-Induced Sudden Cardiac Death. *JACC Case Reports.* 2023;22:101991. https://doi.org/10.1016/j.jaccas.2023.101991
54. Boytsov SA, Kolos IP, Lidov PI, Smolenskyi AV, Ardashev AV, Balykova LA, et al. National recommendations for admission of athletes with cardiovascular disorders to the training and competition process. *Rational Pharmacotherapy in Cardiology.* 2011;7(6S) (In Russ.). EDN: OPRUET
55. Mazzanti A, Trancuccio A, Kukavica D, Pagan E, Wang M, Mohsin M, et al. Independent validation and clinical implications of the risk prediction model for long QT syndrome (1-2-3-LQTS-Risk). *Europace.* 2022;24(4):614–9. https://doi.org/10.1093/europace/euab238

Authors' contributions. All authors confirm that their authorship complies with the ICMJE criteria. The primary contributions are distributed as follows: Leonid M. Makarov — conceptualization, review structuring, literature search and analysis, drafting of the initial manuscript, approval of the final manuscript; Vera N. Komoliatova — literature search and analysis regarding the impact of athletic training on the heart in children and adolescents; Anush G. Akopyan — literature search and analysis on the topic of ECG characteristics in young athletes; Dmitryi A. Besportochnyi — literature search and analysis on the topic of exercise testing for QT interval assessment in athletes; Alena V. Dmitrieva — literature search and analysis on the assessment of autonomic influences on QT interval formation in athletes; Irina I. Kiseleva — literature search and analysis on the topic of sports clearance with QT interval prolongation; Ilya V. Zyabkin — final discussion, general editing.

AUTHORS

Leonid M. Makarov, Dr. Sci. (Med.)
https://orcid.org/0000-0002-0111-3643
dr.leonidmakarov@mail.ru

Vera N. Komoliatova, Dr. Sci. (Med.)
https://orcid.org/0000-0002-3691-7449
verakom@list.ru

Anush G. Akopyan
https://orcid.org/0000-0002-4867-0594
anushik.a.g@mail.ru

Dmitryi A. Besportochnyi
https://orcid.org/0000-0002-3699-2289
dr.blad@mail.ru

Alena V. Dmitrieva
https://orcid.org/0000-0002-0314-5235
gerasimchuk_alena@mail.ru

Irina I. Kiseleva, Cand. Sci. (Med.)
https://orcid.org/0000-0003-3285-3211
vkis2@yandex.ru

Ilya V. Zyabkin, Dr. Sci. (Med.)
https://orcid.org/0000-0002-9717-5872
adm@kidsfmba.ru