DOI: https://doi.org/10.17816/cardar321415

Research article

Long *QT* Syndrome in Young Athletes

Svetlana N. Chuprova[1, 2], Irina Yu. Melnikova[1]

[1] North-Western State Medical University named after I.I. Mechnikov, Saint Petersburg, Russia;

[2] Federal State-Financed Institution Pediatric Research and Clinical Center for Infectious Diseases under the Federal Medical Biological Agency, Saint Petersburg, Russia

Long *QT* syndrome is a disease associated with a high risk of sudden cardiac (arrhythmic) death. The frequency of sudden cardiac death is approximately 1: 100,000 young athletes, while autopsies often do not detect changes, which indicates a primary arrhythmogenic death. The article describes two clinical cases of young athletes with prolongation of the *QT* interval. The possible causes of the long *QT* syndrome and the difficulties of diagnosing this syndrome in children and adolescents involved in sports are discussed. Regardless of the reasons leading to the prolongation of the *QT* interval, there is a risk of arrhythmic events. Timely diagnosis of long *QT* syndrome is the way to the primary prevention of sudden cardiac death in young athletes.

Keywords: sports; young athletes; congenital long *QT* syndrome; secondary causes of *QT* interval prolongation; sudden cardiac death.

To cite this article:
Chuprova SN, Melnikova IYu. Long *QT* syndrome in young athletes. *Cardiac Arrhythmias*. 2023;3(1):41–48. DOI: https://doi.org/10.17816/cardar321415

Received: 16.02.2023 **Accepted:** 19.03.2023 **Published:** 31.03.2022

УДК 616.12-008.318-07-08

DOI: https://doi.org/10.17816/cardar321415

Научная статья

Синдром удлиненного интервала *QT* у юных спортсменов

С.Н. Чупрова[1, 2], И.Ю. Мельникова[1]

[1] Северо-Западный государственный медицинский университет им. И.И. Мечникова, Санкт-Петербург, Россия;

[2] Детский научно-клинический центр инфекционных болезней Федерального медико-биологического агентства, Санкт-Петербург, Россия

Синдром удлиненного интервала *QT* — заболевание, ассоциированное с высоким риском внезапной сердечной (аритмической) смерти. Частота внезапной сердечной смерти составляет примерно 1 : 100 000 юных спортсменов, при этом на вскрытии зачастую не обнаруживают изменений, что указывает на первично аритмогенную смерть. В статье приводится описание двух клинических случаев юных спортсменов с удлинением интервала *QT*. Обсуждаются возможные причины синдрома удлиненного интервала *QT*, трудности диагностики данного синдрома у детей и подростков, занимающихся спортом. Независимо от причин, приводящих к удлинению интервала *QT*, существует риск развития аритмических событий. Своевременная диагностика синдрома удлиненного интервала *QT* — путь к первичной профилактике внезапной сердечной смерти у юных спортсменов.

Ключевые слова: спорт; юные спортсмены; врожденный синдром удлиненного интервала *QT*; вторичные причины удлинения интервала *QT*; внезапная сердечная смерть.

Как цитировать:
Чупрова С.Н., Мельникова И.Ю. Синдром удлиненного интервала *QT* у юных спортсменов // Cardiac Arrhythmias. 2023. Т. 3, № 1. С. 41–48. DOI: https://doi.org/10.17816/cardar321415

Рукопись получена: 16.02.2023 **Рукопись одобрена:** 19.03.2023 **Опубликована:** 31.03.2023

Sudden cardiac death (SCD) is a tragic event, particularly for athletes who undergo routine medical examinations and are considered healthy. The incidence of SCD is approximately 1:100,000 in young athletes, with autopsy often showing no abnormalities indicating primary arrhythmogenic death [1]. The causes of SCD can be acquired or genetic. Acquired causes include myocarditis and coronary artery disease, and genetic causes include genetically determined structural heart diseases and channelopathies [2]. An example of channelopathies is the hereditary (congenital) long *QT* syndrome (LQTS). The prevalence of congenital LQTS is estimated at 1:2000 [3], and for its diagnostics, the criteria developed by Schwartz in 1985 [4] and supplemented by him in 2011 [5, 6] are currently used. Over the past 25 years, 17 genes have been associated with LQTS; however, a recent analysis based on a gene- and disease-specific approach developed by the Clinical Genome Resource classified several of these genes as limited or controversial. This analysis selected seven genes with definitive or conclusive evidence of a causal relationship [7]. All of these remaining genes, i.e., *KCNQ1*, *KCNH2, SCN5A, CALM1, CALM2, CALM3, and TRDN*, encode ion channels involved in cardiac repolarization, their modulating subunits, or proteins that regulate or modulate ion channel function [7]. The LQTS genotype can be identified in 75% of sick people with a clear phenotype, and this is important because it determines the approach to their treatment [8]. In addition to congenital LQTS, various causes (conditions) lead to secondary prolongation of the *QT* interval. One of them is an electrolyte imbalance, such as hypokalemia, hypocalcemia, and hypomagnesemia, which can occur under the influence of many triggers, for example, with long-term intake of diuretics, especially loop diuretics (furosemide).

CLINICAL CASES

Young female athlete A, aged 16 years, was referred for a consultation with a cardiologist because of identified changes in the electrocardiogram (ECG). The history revealed that the patient has been doing rhythmic gymnastics since the age of 3.5 years. At the time of the consultation, the duration of training was 3.5 h a day 5–6 times a week, and she was a sub-master athlete. She coped with exertions, and syncope conditions were not registered. She underwent a planned thorough medical examination (TME). During the regular TME, pathological changes in the ECG, such as prolongation of the *QT* interval, were detected. In the analysis of the family history, cases of SCD and syncope conditions did not occur in the next of kin. The *QT* interval duration in the parents was normal.

On examination, the condition was satisfactory. Her height, weight, and body mass index were 163 cm, 42 kg, and 15.8 kg/m^2, respectively. The skin color was normal. Breath sounds were heard in all lung fields, there were no rales, and the respiratory rate was 20 per min. Visually, the region of the heart was not abnormal. Percussion borders of relative cardiac dullness were within the age norm. The heart sounds were clear, and her heart rate (HR) was 64 per minute and regular in a lying position. Her blood pressure and oxygen saturation were 100/60 mm Hg and 99%, respectively. The abdomen was soft and nontender on palpation. The liver was not enlarged. There was no peripheral edema. The pulse on the femoral arteries was determined on both sides. Bowel and bladder habits were normal.

On a surface 12-channel ECG (computer electrocardiograph CARDI, Medical Computer Systems, Moscow, Zelenograd, Russia), with a recording speed of 50 mm/s (Fig. 1), moderate sinus bradycardia was recorded with an HR of 54–57 beats/min, *QT* interval (V5) of 460 ms, and corrected *QT* (QTc) interval of 438–451 ms.

When taking an ECG in an upright position (Fig. 2) in the presence of an increase in sinus rhythm up to 82 beats/min, the duration of the *QT* interval (V5) was 540 ms and *QT*c was 635 ms (pronounced prolongation of the *QT* interval).

Given the *QT* interval prolongation, Holter ECG monitoring (ECG HM) was performed using the Poly-Spectrum-SM system, Neurosoft, Ivanovo, Russia.

During ECG HM, a prolongation of the *QT* interval up to 680 ms was registered (Fig. 3).

The *QT* interval prolongation persisted regardless of the HR, namely, 680 ms in sinus rhythm with an HR of 53 beats/min, 580 ms with an HR of 85 beats/min (Fig. 4), and with a norm according to ECG HM findings up to 480 ms [9].

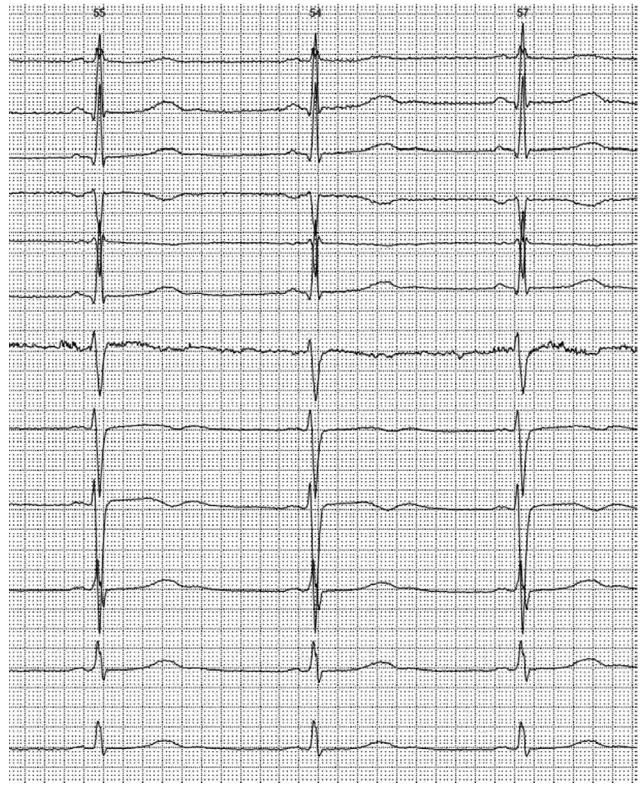

Fig. 1. 12-lead electrocardiogram in young athlete A., 16 years old

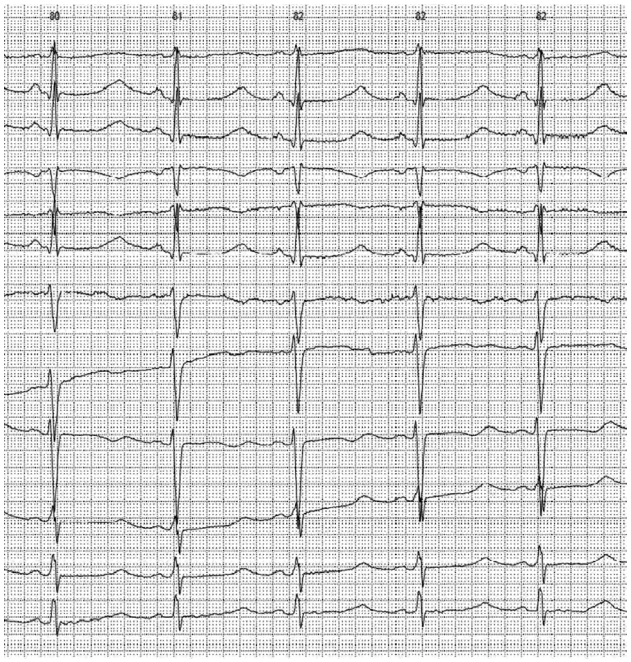

Fig. 2. 12-lead electrocardiogram (standing) in young athlete A., 16 years old

According to the automatic analysis of *QT* (the norms are presented according to the National Russian recommendations for the use of the HM technique in clinical practice), an elongation of this interval was also revealed, where the average *QT* interval was 471 (normal 342–401) ms, *QT*c by Bazett was 500 (normal 396–447) ms, and QTc by Fridericia was 490 (normal 384–421) ms.

Rare (total 354, extrasystole density 0.4%) single ventricular extrasystoles and episodes (3) of trigeminy (Fig. 5) were registered.

Transthoracic echocardiography (EPIQ 5 Ultrasound Device, Philips, Netherlands) showed normal biventricular systolic function. The valvular apparatus had no pathological abnormalities, and the transvalvular flow was normal. No enlargement/hypertrophy of the heart chambers was detected. To rule out possible secondary causes of *QT* interval prolongation in a young athlete, the level of electrolytes in the blood serum was determined. The analysis revealed hypokalemia (potassium 2.2 mmol/L; normal, 3.6–5.6 mmol/L). During a conversation with the patient, it was established that she had been taking furosemide without control for 1.5 years to reduce weight. After drug correction of the potassium level, the *QT* interval was normalized according to the results of ECG (Fig. 6) and ECG HM, ventricular extrasystoles were not recorded.

Bicycle ergometry (Corival Lode, Netherlands) at all stages of the load, with 4 min of recovery, did not reveal an elongation of the *QT* interval.

During the follow-up period (catamnesis 2 years), the athlete fulfilled the standard of mastery of sports in rhythmic gymnastics, and her blood level of potassium and the duration of the *QT* interval remained within the normal range.

Young female athlete E, aged 10 years, was examined due to acute respiratory disease at the Children's Scientific and Clinical Center for Infectious Diseases of the Federal Medical and Biological Agency. The prolongation of the *QT* interval was revealed by ECG with a recording speed of 50 mm/s. ECG (Fig. 7) recorded sinus arrhythmia, episodes of bradycardia (HR 63–86 bpm), *QT* (V5) of 420 ms, and QTc of 429–506 ms (increase in QTc interval with an increase in HR). The two-humped T wave morphology and notched T wave were revealed in leads V4–V6, which is characteristic

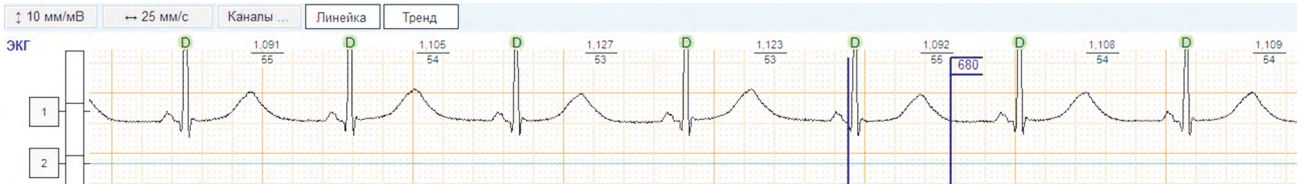

Fig. 3. Fragment of Holter ECG monitoring in young athlete A. The maximum duration of the QT interval

Fig. 4. Fragment of Holter ECG monitoring in young athlete A. Prolongation of the QT interval

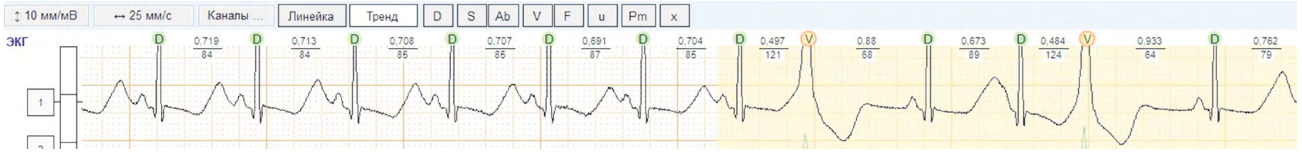

Fig. 5. Fragment of Holter ECG monitoring in young athlete A. Ventricular extrasystoles, trigeminy

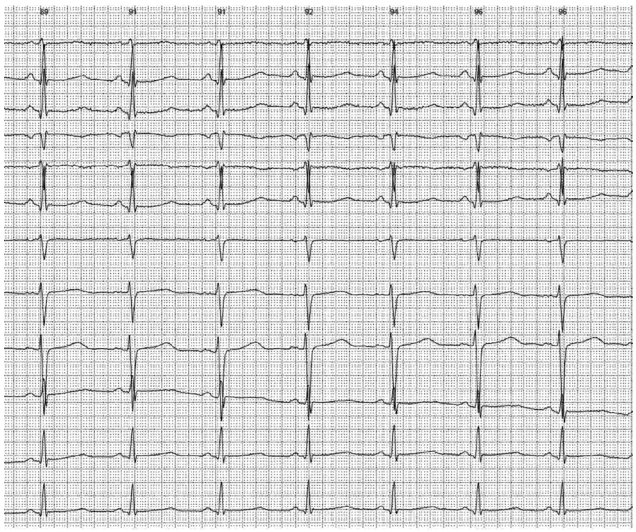

Fig. 6. 12-lead electrocardiogram (standing) in young athlete A., 16 years old, after normalization of potassium concentration in blood serum. *QT* (V5) = 350 ms (A heart rate of 96 beats per min), QTc = 443 ms.

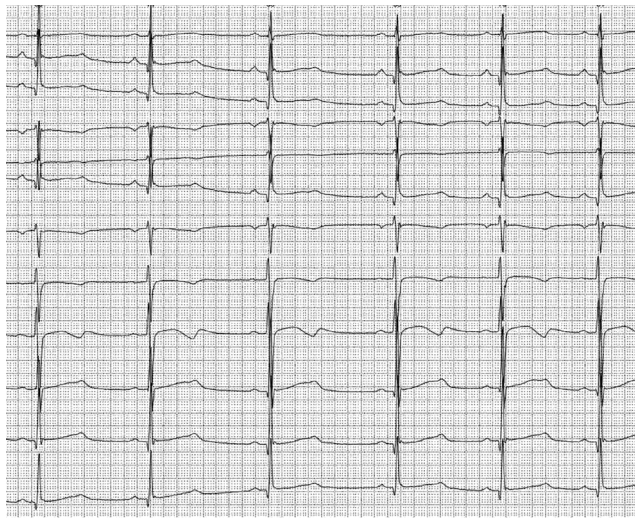

Fig. 7. 12-lead electrocardiogram in young athlete E., 10 years old

of the molecular genetic variant 2 (LQT2) of the hereditary LQTS [10].

During ECG HM (Cardioline System, Italy), the *QT* interval was prolonged up to 540 ms (recording channel 3) at a minimum HR of 46 beats/min (Fig. 8) at a rate of up to 480 ms. Serum electrolytes were normal. Transthoracic echocardiography revealed no pathological changes.

The anamnesis revealed that the patient has been doing rhythmic gymnastics since the age of 4, with training 5–6 times a week for 3 h. No pathological changes in the ECG were detected during the scheduled TME. She had no episodes of loss of consciousness. As regards heredity, the girl's mother had two episodes of loss of consciousness (at the age of 33 and 35 years). The first attack was related to stress, and the other had no apparent cause. The attacks were accompanied by convulsions and involuntary urination. Regarding fainting, the patient was examined in one of the clinics in St. Petersburg and consulted by a neurologist, and a diagnosis of autonomic dysfunction syndrome was made. The ECG of the mother (Fig. 9) revealed sinus rhythm with an HR of 70–80 beats/min and a significant prolongation of the *QT* interval, where the *QT* (V5) at an HR of 76 beats/min was 500 ms and QTc was 562 ms. The T morphology was also characteristic of the molecular genetic variant 2 of the hereditary LQTS.

Based on the generally accepted diagnostic criteria proposed by Schwartz [4, 6], patient E was diagnosed with hereditary LQTS, familial variant (inheritance on the mother's side), and molecular genetic variant 2. Whole-genome DNA sequencing was performed at the EVOGEN Medical Genetic Laboratory (Moscow), which revealed a previously undescribed variant p. Met554ValfsTer100 (leading to the formation of a premature stop codon) in the heterozygous

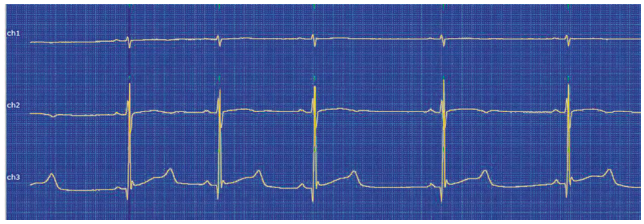

Fig. 8. Fragment of Holter ECG monitoring in young athlete E. Prolongation of the QT interval

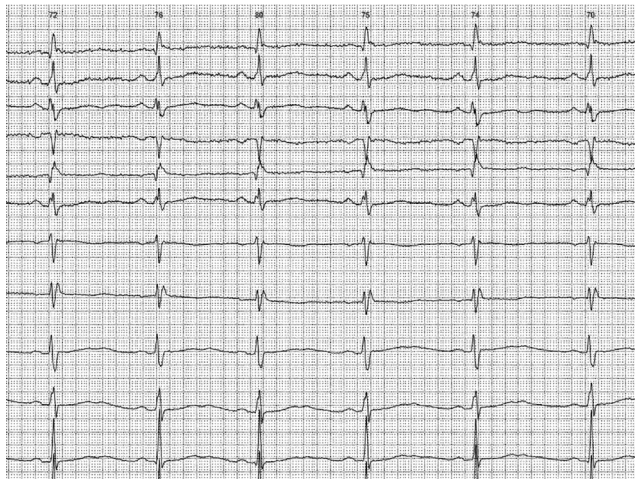

Fig. 9. Prolongation of the *QT* interval in the girl's mother.

state in exon 7 of 15 exons of the KCNH2 gene responsible for the development of the molecular genetic variant 2 of LQTS (LQT2).

Atenolol was recommended at a daily dose of 1 mg/kg. Training at the sports school has been discontinued.

DISCUSSION

LQTS is a disease associated with a high risk of SCD due to the development of torsade de pointes polymorphic ventricular tachycardia [11]. Regardless of the causes of LQTS (such as hypokalemia due to long-term intake of furosemide in clinical case 1 and a mutation in the KCNH2 gene in clinical case 2, which caused the development of LQT2), patients with LQTS are at risk of life-threatening arrhythmic events. In a study conducted in the USA, LQTS causes SCD in 2% of athletes [2], and 0.4% of Olympic athletes may experience ventricular tachyarrhythmias associated with this syndrome [12]. Before diagnosing young athletes with congenital LQTS, acquired causes of *QT* prolongation must be ruled out. The most common causes include medications that prolong *QT*, metabolic changes, and electrolyte disorders. One of the routine methods to detect changes typical for LQTS is a standard 12-lead ECG. The interpretation of the ECG in athletes includes an estimate of the length of the QTc, calculated using the Bazett equation. However, difficulties may be encountered in assessing the *QT* interval in young athletes. Approximately 25%–35% of patients with a genetically confirmed hereditary LQTS may have normal *QT* interval on the ECG at rest [2]. Determining the duration of the *QT* interval associated with sinus bradycardia characteristic of athletes is even more difficult, especially when identifying the prolongation of the *QT* interval associated with an increase in HR (an example of a 10-year-old rhythmic gymnast diagnosed with LQT2). Moreover, when registering severe sinus arrhythmia on the ECG, the response of the *QT* interval to a change in the HR is not instantaneous and complete adaptation takes 1–3 min [13]. Problems can also arise when making a differential diagnosis between the U wave and the two-humped T wave, characteristic of LQT2, on the body-surface ECG.

Until now, there are conflicting opinions on the admission of athletes with a diagnosed congenital LQTS to training and competitions. The 2005 ESC guidelines for participation in sports competitions are the most restrictive [14]. They state that congenital LQTS is a contraindication for any sports, even in the absence of documented serious cardiac arrhythmias. The 2015 European Society of Cardiology guidelines for the treatment of ventricular arrhythmia and the prevention of SCD recommended avoiding intensive swimming, particularly in the case of LQT1; however, no other sports have been mentioned [15]. More recently, the 2015 American eligibility and disqualification guidelines for athletes with channelopathies (including LQTS), who take part in competitions, are less restrictive [16]. According to these guidelines, athletes with symptomatic LQTS (except for competitive swimming with LQT1) may be eligible to compete after the initiation of treatment and appropriate precautions, as long as they have been symptom-free during treatment for at least 3 months and these athletes and their family members received information about potential risks. In the Russian national recommendations on the admission of athletes with abnormalities in the cardiovascular system to training and competitions, for athletes with a history of an episode of cardiac arrest or syncope conditions, presumably associated with LQTS, regardless of the QTc interval or genotype, all sports are contraindicated, except for class IA [17].

Thus, these cases clearly demonstrate the existing difficulties in diagnosing LQTS in young athletes and the need for a comprehensive analysis of possible causes of LQTS, which, in some cases, will help preserve the possibility of continued involvement in the chosen sport and reduce the risk of SCD in other cases. To detect *QT* prolongation in young athletes, further studies including stress tests and molecular genetic testing are needed to search for gene mutations responsible for the development of this syndrome.

REFERENCES

1. Makarov LM. Sport and sudden death in children. *Russian Bulletin of Perinatology and Pediatrics*. 2017;62(1):40–46. (In Russ.) DOI: 10.21508/1027-4065-2017-62-1-40-46

2. Longo UG, Ambrogioni LR, Ciuffreda M, et al. Sudden cardiac death in young athletes with long QT syndrome: the role of genetic testing and cardiovascular screening. *Br Med Bull.* 2018;1(127): 43–53. DOI: 10.1093/bmb/ldy017

3. Krahn AD, Laksman Z, Sy RW, et al. Congenital Long QT syndrome. *JACC Clin Electrophysiol.* 2022;8(5):687–706. DOI: 10.1016/j.jacep.2022.02.017

4. Schwartz PJ. Idiopathic long QT syndrome: progress and questions. *Am Heart J.* 1985;109(2):399–411. DOI: 10.1016/0002-8703(85)90626-x

5. Sy RW, van der Werf C, Chattha IS, et al. Derivation and validation of a simple exercise-based algorithm for prediction of genetic testing in relatives of LQTS probands. *Circulation.* 2011;124(20):2187–2194. DOI: 10.1161/CIRCULATIONAHA.111.028258

6. Schwartz PJ, Crotti L. QTc behavior during exercise and genetic testing for the long-QT syndrome. *Circulation.* 2011;124(20): 2181–2184. DOI: 10.1161/CIRCULATIONAHA.111.062182

7. Adler A, Novelli V, Amin AS, et al. An International, Multicentered, Evidence-Based Reappraisal of Genes Reported to Cause Congenital Long QT Syndrome. *Circulation*. 2020;141(6):418–428. DOI: 10.1161/CIRCULATIONAHA.119.043132

8. Arthur AM, Ahmad S, Pieter G Diagnosis, management and therapeutic strategies for congenital long QT syndrome. *Heart.* 2021;108(5):332–338. DOI: 10.1136/heartjnl-2020-318259

9. Makarov LM, Komolyatova VN, Kupriyanova OO, et al. National russian guidelines on application of the methods of holter monitoring

in clinical practice. *Russian Journal of Cardiology.* 2014;2(106):6–71. (In Russ.). DOI: 10.15829/1560-4071-2014-2-6-71

10. Tardo DT, Peck M, Subbiah RN, et al. The diagnostic role of T wave morphology biomarkers in congenital and acquired long QT syndrome: A systematic review. *Ann Noninvasive Electrocardiol*. 2022. 28(1):e13015. DOI: DOI 10.1111/anec.13015

11. Singh M, Morin DP, Link MS. Sudden cardiac death in Long QT syndrome (LQTS), Brugada syndrome, and catecholaminergic polymorphic ventricular tachycardia (CPVT). *Prog Cardiovasc Dis.* 2019; 62(3):227–234. DOI: 10.1016/j.pcad.2019.05.006

12. Pelliccia A, Adami PE, Quattrini F, et al. Are Olympic athletes free from cardiovascular diseases? Systematic investigation in 2352 participants from Athens 2004 to Sochi 2014. *Br J Sports Med.* 2017;51(4):238–243. DOI: 10.1136/bjsports-2016-096961

13. Schnell F, Behar N, Carré F. Long-QT Syndrome and Competitive Sports. *Arrhythm Electrophysiol Rev*. 2018;7(3):187–192. DOI: 10.15420/aer.2018.39.3

14. Pelliccia A, Fagard R, Bjørnstad HH, et al. Recommendations for competitive sports participation in athletes with cardiovascular disease: a consensus document from the Study Group of Sports 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 // Eur Heart J. 2005;14(26):1422–1445. DOI: 10.1093/eurheartj/ehi325

15. Priori S.G., Blomström-Lundqvist C., Mazzanti A., et al. 2015 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: The Task Force for the Management of Patients with Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death of the European Society of Cardiology (ESC). Endorsed by: Association for European Paediatric and Congenital Cardiology (AEPC) // Eur Heart J. 2015;(36):2793–867. DOI: 10.1093/eurheartj/ehv316

16. Ackerman MJ, Zipes DP, Kovacs RJ, et al. Eligibility and disqualification recommendations for competitive athletes with cardiovascular abnormalities: Task Force 10: The Cardiac Channelopathies: A Scientific Statement From the American Heart Association and American College of Cardiology. *J Am Coll Cardiol*. 2015;66(21):2424–2428. DOI: 10.1016/j.jacc.2015.09.042

17. Boitsov SA, Kolos IP, Lidov PI, et al. Natsional'nye rekomendatsii po dopusku sportsmenov s otkloneniyami so storony serdechno-sosudistoi sistemy k trenirovochno-sorevnovatel'nomu protsessu. *Rational Pharmacotherapy in Cardiology.* 2011;7(6):4–60. (In Russ.).

СПИСОК ЛИТЕРАТУРЫ

1. Макаров Л.М. Спорт и внезапная смерть у детей // Российский вестник перинатологии и педиатрии. 2017. Т. 62, № 1. С. 40–46. DOI: 10.21508/1027-4065-2017-62-1-40-46

2. Longo U.G., Ambrogioni L.R., Ciuffreda M., et al. Sudden cardiac death in young athletes with long QT syndrome: the role of genetic testing and cardiovascular screening // Br Med Bull. 2018. Vol. 127, No. 1. P. 43–53. DOI: 10.1093/bmb/ldy017

3. Krahn A.D., Laksman Z., Sy R.W., et al. Congenital Long QT syndrome // JACC Clin Electrophysiol. 2022. Vol. 8, No. 5. P. 687–706. DOI: 10.1016/j.jacep.2022.02.017

4. Schwartz P.J. Idiopathic long QT syndrome: progress and questions // Am Heart J. 1985. Vol. 109, No. 2. P. 399–411. DOI: 10.1016/0002-8703(85)90626-x

5. Sy R.W., van der Werf C., Chattha I.S., et al. Derivation and validation of a simple exercise-based algorithm for prediction of genetic testing in relatives of LQTS probands // Circulation. 2011. Vol. 124, No. 20. P. 2187–2194. DOI: 10.1161/CIRCULATIONAHA.111.028258

6. Schwartz P.J., Crotti L. QTc behavior during exercise and genetic testing for the long-QT syndrome. Circulation. 2011. Vol. 124, No. 20. P. 2181–2184. DOI: 10.1161/CIRCULATIONAHA.111.062182

7. Adler A., Novelli V., Amin A.S., et al. An International, Multicentered, Evidence-Based Reappraisal of Genes Reported to Cause Congenital Long QT Syndrome. Circulation. 2020. Vol. 141, No. 6. P. 418–428. DOI: 10.1161/CIRCULATIONAHA.119.043132

8. Wilde AAM, Amin AS, Postema PG. Diagnosis, management and therapeutic strategies for congenital long QT syndrome // Heart. 2021. Vol. 108, No. 5. P. 332–338. DOI: 10.1136/heartjnl-2020-318259

9. Макаров Л.М., Комолятова В.Н., Куприянова О.О., и др. Национальные российские рекомендации по применению методики холтеровского мониторирования в клинической практике. Российский кардиологический журнал. 2014. № 2 (106). С. 6–71. DOI: 10.15829/1560-4071-2014-2-6-71

10. Tardo D.T., Peck M., Subbiah R.N., et al. The diagnostic role of T wave morphology biomarkers in congenital and acquired long QT syndrome: A systematic review // Ann Noninvasive Electrocardiol. 2022. Vol. 28, No. 1. P. e13015. DOI: DOI 10.1111/anec.13015

11. Singh M., Morin D.P., Link M.S. Sudden cardiac death in Long QT syndrome (LQTS), Brugada syndrome, and catecholaminergic polymorphic ventricular tachycardia (CPVT) // Prog Cardiovasc Dis. 2019. Vol. 62, No. 3. P. 227–234. DOI: 10.1016/j.pcad.2019.05.006

12. Pelliccia A., Adami P.E., Quattrini F., et al. Are Olympic athletes free from cardiovascular diseases? Systematic investigation in 2352 participants from Athens 2004 to Sochi 2014 // Br J Sports Med. 2017. Vol. 51, No. 4. P. 238–243. DOI: 10.1136/bjsports-2016-096961

13. Schnell F., Behar N., Carré F. Long-QT Syndrome and Competitive Sports // Arrhythm Electrophysiol Rev. 2018. Vol. 7, No. 3. P. 187–192. DOI: 10.15420/aer.2018.39.3

14. Pelliccia A., Fagard R., Bjørnstad H.H., et al. Recommendations for competitive sports participation in athletes with cardiovascular disease: a consensus document from the Study Group of Sports 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 // Eur Heart J. 2005. Vol. 26, No. 14. P. 1422–1445. DOI: 10.1093/eurheartj/ehi325

15. Priori S.G., Blomström-Lundqvist C., Mazzanti A., et al. 2015 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: The Task Force for the Management of Patients with Ventricular Arrhythmias

and the Prevention of Sudden Cardiac Death of the European Society of Cardiology (ESC). Endorsed by: Association for European Paediatric and Congenital Cardiology (AEPC) // Eur Heart J. 2015. No. 36. P. 2793–867. DOI: 10.1093/eurheartj/ehv316

16. Ackerman M.J., Zipes D.P., Kovacs R.J., et al. Eligibility and disqualification recommendations for competitive athletes with cardiovascular abnormalities: Task Force 10: The Cardiac Channelopathies: A Scientific Statement from the American Heart Association and American College of Cardiology // J Am Coll Cardiol. 2015. Vol. 66, No. 21. P. 2424–2428. DOI: 10.1016/j.jacc.2015.09.042

17. Бойцов С.А., Колос И.П., Лидов П.И., и др. Национальные рекомендации по допуску спортсменов с отклонениями со стороны сердечно-сосудистой системы к тренировочно-соревновательному процессу // Рациональная фармакотерапия в кардиологии. 2011. Т. 7, № 6. С. 4–60.

AUTHORS INFO

***Svetlana N. Chuprova,** associate professor;
eLibrary SPIN: 8696-7178;
ORCID: https://orcid.org/0000-0002-5661-3389;
e-mail: svetlana_ch_70@mail.ru

Irina Yu. Melnikova, Head of the Department;
eLibrary SPIN: 8053-1512;
ORCID: https://orcid.org 0000-0002-1284-5890;
e-mail: melnikovai@yandex.ru

ИНФОРМАЦИЯ ОБ АВТОРАХ

***Светлана Николаевна Чупрова,** доцент кафедры;
eLibrary SPIN: 8696-7178;
ORCID: https://orcid.org/0000-0002-5661-3389;
e-mail: svetlana_ch_70@mail.ru

Ирина Юрьевна Мельникова, заведующий кафедрой педиатрии; eLibrary SPIN: 8053-1512;
ORCID: https://orcid.org 0000-0002-1284-5890;
e-mail: melnikovai@yandex.ru

* Corresponding author / Автор, ответственный за переписку