



ТРАВМАТОЛОГИЯ TRAUMATOLOGY

DOI 10.22363/2313-0245-2023-27-2-254-264
EDN FTSUAC

REVIEW
ОБЗОРНАЯ СТАТЬЯ

Diagnosics and prevention of sports-related traumatic brain injury complication

Oleg A. Shevelev^{1,2} , Anderei V. Smolensky³ , Marina V. Petrova^{1,2} , Elias M. Mengistu^{1,2}  ,
Anastasia A. Mengistu¹ , Maria V. Vatsik-Gorodetskaya^{1,4} , Umayzat G. Khanakhmedova⁴ ,
Darina N. Menzhurenkova¹ , Sergey G. Vesnin⁵ , Igor I. Goryanin^{6,7} 

¹RUDN University Moscow, Russian Federation

²Federal Research and Clinical Centre for Intensive Care Medicine and Rehabilitology, Moscow, Russian Federation

³The Russian University of Sports “GTSOLIFK”, Moscow, Russian Federation

⁴City Clinical Hospital named after V.V. Vinogradov, Moscow, Russian Federation

⁵Medical Microwave Radiometry LTD, Edinburgh, United Kingdom

⁶School of Informatics, University of Edinburgh, Edinburgh, United Kingdom

⁷Okinawa Institute Science and Technology, Okinawa, Japan

 drmengistu@mail.ru

Abstract. Sports-related traumatic brain injuries (TBI) accounts for up to 20 % of all injuries that are obtained by athletes and its incidence rises annually due to rise in population involving in sports, growing popularity of extreme sports and high level of motivation to achieve record results among young sportsmen. *The aim of the review* is to present the potential benefits of using microwave radiothermometry and craniocerebral hypothermia technologies in sports-related TBI. The review considers most common form of traumatic brain injury in athletes — mild TBI, which in turn can provoke a wide range of complications and negative consequences in near and delayed periods after the injury. The main shortcomings of programs for complication prevention in treatment and rehabilitation of athletes after TBI are considered, which do not take into account the peculiarities

© Shevelev O.A., Smolensky A.V., Petrova M.V., Mengistu E.M., Mengistu A.A., Vatsik-Gorodetskaya M.V., Khanakhmedova U.G., Menzhurenkova D.N., Vesnin S.G., Goryanin I.I., 2023



This work is licensed under a Creative Commons Attribution 4.0 International License
<https://creativecommons.org/licenses/by-nc/4.0/legalcode>

of injury mechanisms, its significant differences from household, road or criminal injuries with brain damage. Lack of objective methods of instrumental diagnosis for injury severity is also described. In addition, pathophysiological component characteristics of sports TBI is accentuated: frequency of repetition, increase of brain and body temperature, peripheral redistribution of blood flow and hypocapnia, which significantly affects cerebral blood flow. Based on the analysis of the available scientific literature, it is elicited that TBI is an independent cause of cerebral hyperthermia development, which significantly aggravates the consequences of the injury. *Conclusions.* The authors propose an innovative way to use microwave radiothermometry method as a diagnostic tool for sports-related TBI. In addition, the review highlights the main recommendations for complications prevention by using craniocerebral hypothermia technology, which reduces overall physical and cerebral hyperthermia, and augments the resistance of cerebral cortex neurons to hypoxia and trauma. However, the authors believe that the described approaches in sports medicine are not used purposefully due to lack of awareness of sports team doctors and coaches.

Key words: hypothermia, sports, traumatic brain injury, rehabilitation, hyperthermia

Funding. The authors received no financial support for the research, authorship, and publication of the review.

Author contributions. Oleg A. Shevelev, Igor Goryanin, Andrei V. Smolensky — conceptualization, writing — original Draft; Marina V. Petrova — supervision; Sergey G. Vesnin — data curation; Elias M. Mengistu, Anastasia A. Mengistu — writing, review, editing; Maria V. Vatsik-Gorodetskaya, Umayzat G. Khanakhmedova, Darina N. Menzhurenkova — investigation and formal analysis. All authors have made significant contributions to the development concepts, research and manuscript preparation, read and approved final version before publication.

Conflicts of interest statement. Authors declare no conflict of interest.

Ethics approval — not applicable.

Acknowledgements — not applicable.

Consent for publication — not applicable.

Received 19.03.2023. Accepted 24.04.2023.

For citation: Shevelev OA, Smolensky AV, Petrova MV, Mengistu EM, Mengistu AA, Vatsik-Gorodetskaya MV, Khanakhmedova UG, Menzhurenkova DN, Vesnin SG, Goryanin II. Diagnostics and prevention of sports-related traumatic brain injury complication. *RUDN Journal of Medicine*. 2023;27(2):254–264. doi: 10.22363/2313-0245-2023-27-2-254-264

Introduction

In the structure of sports injuries, traumatic brain injuries (TBI) account for up to 20 % of all types of injuries [1]. About 97 % of sports TBIs are mild TBIs (MTBIs) and the neurological symptoms are often lenient so that most of the injured young, strong, highly motivated athletes tend to downplay the injury. This can also cause an underestimation of the severity and the extent of received injuries by a doctor or a trainer [2].

Minor brain injury is an acutely developed impairment of brain function, which is the result of a blunt blow with sudden acceleration, deceleration

or rotation of the head, in which the patient is in clear consciousness, or the level of wakefulness is reduced to moderate deafness, while there may be a short-term loss of consciousness (up to 30 minutes) and/or amnesia (up to 24 hours) [3,4]. In most patients, recovery after mild TBI occurs in a short time (within 1–2 weeks), and in 5–20 % of cases, symptoms of post-concussion syndrome (cognitive, emotional, and behavioral disorders) are noted for a long time. The severity of TBI is most often assessed using the Glasgow Coma Scale, and MTBI corresponds to a score of 13–15 points in the acute period after injury. Metabolic, ionic, and neurotransmitter disorders and neuroinflammation develop in

mild TBI, but changes on computed tomography (CT) scan and magnetic resonance imaging (MRI) may be absent.

Of great importance in worsening the prognosis of the course of injury is the syndrome of re-injury during the period of the special vulnerability of the brain when the brain is especially susceptible to changes in intracranial pressure, blood flow, and hypoxia.

Acute cerebrovascular disorders and neurotrauma are accompanied by a focal increase in brain temperature, which may not be reflected in changes in basal temperature. In these cases, it is diagnostically important to use non-invasive microwave radiometry (MWR), which makes it possible to identify the foci of cerebral hyperthermia. MWR is based on measuring the power of the intrinsic emissions of human tissues in the microwave range, which makes it possible to calculate the temperature of the cerebral cortex at a depth of 4–6 centimeters from the skin surface [5].

Up to date, in sports MTBI with neurological manifestations, symptomatic pharmacotherapy is usually carried out, and as recommendations, a reduction in physical activity during the rehabilitation period is suggested. The arsenal of rehabilitation technologies for mild TBI is limited. At the same time, it is known that a decrease in brain temperature provides the development of pronounced neuroprotective effects: an increase in the resistance of brain cells to ischemia, hypoxia, reperfusion, and trauma, limitation of glutamate-mediated excitotoxicity reactions, inhibition of the inflammatory response to damage and the development of oedema, as well as apoptotic and necrobiotic cascades [6–8]. It seems very tempting to use this colossal potential of brain protection in MTBI.

In the treatment of severe TBI, artificial hypothermia induction methods were previously widely used [5]. Low-temperature technologies of cerebral protection include various methods of general cooling of patients, achieving a decrease in body temperature to 32–33 °C [9], which is not applicable for mild TBI. The known technique of nasopharyngeal hypothermia is of little use in sports medicine due to the need to obturate the nasal passages with cooling systems [10, 11].

Also the craniocerebral hypothermia (CCH) method is known which is based on lowering the temperature of the scalp in the craniocerebral region in combination with neck cooling in the area of projections of the carotid arteries [12]. It is also possible to use selective craniocerebral hypothermia (SCCH) without cooling the neck, which has proven itself well in the treatment of acute ischemic stroke and many diseases accompanied by cerebral and general hyperthermia (paroxysmal sympathetic hyperactivity syndrome, delirious and withdrawal syndromes, pyretic fever) [13]. Selective CCH does not affect basal body temperature and other homeostasis parameters with a heat removal session of up to 4 hours and is the best candidate for use in sports with mild TBI.

Thus, there are convincing prerequisites that MWR and SCCH can be used to diagnose sports-related mild TBI and prevent the development of negative consequences of injury. In this regard, it seems important to consider the issues of the features of changes in the thermal balance of the brain in sports-related TBI and the use of selective CCH.

Temperature balance of the brain and CCH

The brain is characterized by the highest metabolic activity, accompanied by a powerful heat release (20 % of the body's total heat at rest), which requires at least 20 % of the total oxygen utilized by the body, 25 % of glucose and IOC, with a brain mass of not more than 2 % [fourteen].

Almost all processes occurring in the central nervous system are sensitive to temperature fluctuations — the resting potential and the action potential, the rate of excitation, the efficiency of synaptic interactions, the production and release of signal molecules, etc. [15,16]. Temperature internally affects the efficiency and rate of metabolism in the brain, and temperature fluctuations modulate behavioural and autonomic responses and affect cognitive functions [17–19].

Under conditions of rest and norm, the brain is moderately thermo-heterogeneous, and the level of functional and temperature heterogeneity increases with excitation (emotion, affect) and various patho-

logical processes (cerebrovascular accident, trauma), accompanied by the development of focal cerebral hyperthermia.

With direct invasive temperature measurement in the oesophagus, ear canal, arterial blood in the aorta and venous blood in the jugular vein bulb in athletes, it was shown that during physical exertion, causing an increase in temperature in the oesophagus to 37.8 °C, the blood temperature in the aorta increased to 38 °C, in the jugular vein up to 38.5 °C, while the tympanic temperature did not exceed 37.5 °C. An increase in the temperature of the blood flowing from the brain emphasizes the fact of the accumulation of cerebral heat during working hyperthermia [20].

The human brain has a spherical shape, which contributes to the retention of heat due to the effective ratio of surface area to its mass, and the removal of excess heat is limited since the brain is enclosed in a hard bone “case” of the skull, which makes it difficult to transfer heat to the outside.

The brain has certain passive ways of removing heat. The main pathway for removing excess heat from the brain is provided by a powerful influx of arterial blood [21], which is sufficient to maintain normal cerebral heat balance at rest [22].

However, with an increase in body temperature, the influx of warm blood worsens the conditions for removing excess heat from the brain, which begins to accumulate. A decrease in cerebral perfusion with oedema and an increase in ICP also impairs heat dissipation.

Another convection mechanism for regulating brain temperature is formed by cooling the cerebral cortex with venous blood flowing from the scalp through the emissary’s veins and reaching the venous sinuses of the dura mater through perforators [22]. This very short transit route of venous blood cooled in the external environment to the cerebral cortex seems to be very effective, but its contribution to the maintenance of brain thermo-homeostasis has not been sufficiently studied. At the same time, it is clear that the colder the scalp and the venous blood flowing from it, the more effective the cooling of the cerebral cortex will be.

It should be borne in mind that the brain is the only organ whose blood supply is carried out from the

surface. Therefore, the cerebral cortex in normal and at rest is somewhat colder than the basal structures.

Thus, the physiological mechanisms and anatomical security of maintaining the thermal balance of the brain are aimed primarily at cooling the cerebral cortex.

Insignificantly involved in the removal of excess heat from the brain direct heat transfer from the surface of the brain to the outside through the flat bones of the skull and soft tissues of the head due to their low thermal conductivity.

The described pathways for the removal of excess cerebral heat make it possible to understand the mechanisms of hypothermia induction during craniocerebral cooling, which requires factual evidence.

With CCH, the temperature of the scalp can be reduced to 5–8 °C. The outflowing venous blood under these conditions enhances the heat exchange between the jugular vessels and the internal carotid arteries. The blood flow in the scalp at low temperatures is not completely blocked due to the initial vasoconstriction and is partially restored after 15–20 minutes [23]. Cold blood penetrating the sinuses of the dura mater through the emissary’s veins enhances convection heat removal and contributes to a decrease in the temperature of the cerebral cortex. With CCH, a significant temperature difference is formed between the surface of the brain and the scalp, reaching 25–30 °C, providing an increase in the flow of heat to the outside by thermal conductivity.

There are calculated and experimental justifications for the effectiveness of induced brain hypothermia during craniocerebral cooling. In particular, an analytical solution of heat transfer during targeted hypothermia of the brain is presented, confirmed by experiments, where it is shown that the cooling of the scalp significantly affects the temperature in the superficial zone of the brain, ensuring its decrease without affecting the temperature of the basal structures [24].

The nature of the temperature distribution in the human brain was studied using magnetic resonance spectroscopy (MRS), where it was found that with a decrease in the temperature of the scalp, hypothermia of the cerebral cortex is formed, but the temperature of the subcortical structures remains at 37 °C [25].

When modeling the brain cooling process, it was shown that 4-hour cooling of the scalp at a temperature of about 10 °C can lower the temperature of the superficial areas of the brain to 33.2 °C at a depth of up to 25 mm [26].

These calculated data very closely match the model of the heat balance of biological tissues given in another study [27]. Experiments with thermal sensors implanted in the brain have shown that selective cerebral hypothermia in monkeys is reproduced when the scalp is cooled [28].

The use of MWR made it possible to show that 30–45 minutes of CCH induction in healthy individuals provides a decrease in temperature over the entire surface of the brain by 1.5–2 °C. The lengthening of the cooling period by up to 4 hours made it possible to reduce the average temperature of the cerebral cortex by 2.5–4 °C. The basal temperature did not change significantly during this duration of cooling, as did blood pressure and heart rate [29].

Features of sports MTBI and the use of SCCH

An increase in temperature during overheating due to physical exertion can lead to significant disorders of the cerebral circulation and contributes to the development of cerebral oedema, increased intracranial pressure, disorders of interneuron relations, a decrease in the level of consciousness and cognitive impairment [30].

Hyperventilation and a decrease in partial pressure of Carbon Dioxide (PCO₂) in the blood are accompanied by a decrease in cerebral perfusion due to regular reactions of autoregulation of cerebral blood flow. In addition, a peripheral redistribution of blood flow develops in favour of the working muscles and skin to increase heat transfer during sweating, dehydration, and hypovolemia increase. Taken together, these phenomena lead to a significant decrease in cerebral perfusion and oxygenation, forming a kind of “steal” syndrome of the brain, which becomes especially vulnerable during this period to traumatic injury [31].

An increase in brain temperature against the background of reduced perfusion and oxygenation underlies the central mechanisms of fatigue, impaired speed,

strength, and coordination functions, which also contributes to an increased risk of sports-related TBI [32].

The development of cerebral hyperthermia forms a cascade of reactions typical of neuronal damage during ischemia, hypoxia, reperfusion, and neurotrauma: glutamate release increases, proinflammatory cytokines (IL-1, IL-6) accumulate, and free radical processes increase [33]. Cerebral hyperthermia forms vicious circles of neuronal damage even in cases where there is no primary brain damage, and if it is present, it exacerbates the pathological process.

For sports-related TBI, especially in martial arts, it is typical to get repeated injuries in short periods.

Thus, the specific features of sports-related TBI are repeated frequent TBI, high body and brain temperature, and reduced cerebral perfusion. Post-traumatic changes are formed in conditions of high stress on the cardiovascular system. Timely objective assessment of MTBI is very often hampered by the effacement of symptoms and anti-gravity behavior of athletes seeking to continue participating in training and competitive cycles, which can cause underestimation of the severity of the injury. After sports MTBI, obtained in sparring in boxers and not accompanied by the formation of neurological symptoms, focal hyperthermia of the brain develops with foci of temperature increase up to 38–40 °C [34]. Localization of foci turns out to be individual, often manifesting itself in a certain projection of the cerebral cortex, which indicates the formation of “locus minoris resistentia” (lat.) — a weak spot that can eventually become the basis of structural brain disorders.

The use of MWR by recording temperature in 9 symmetrical regions of the left and right hemispheres makes it possible to build a map of the distribution of brain surface temperature and evaluate the differences recorded at rest, during exercise, and after mild TBI (Fig. 1).

Considering the neuroprotective potential of hypothermia and the pathogenetic role of cerebral hyperthermia, it seems appropriate to present the results of the practical application of hypothermia during physical exertion and MTBI obtained in several studies [35–38].

In athletes of cyclic sports, the axial temperature and the temperature of the cerebral cortex were re-

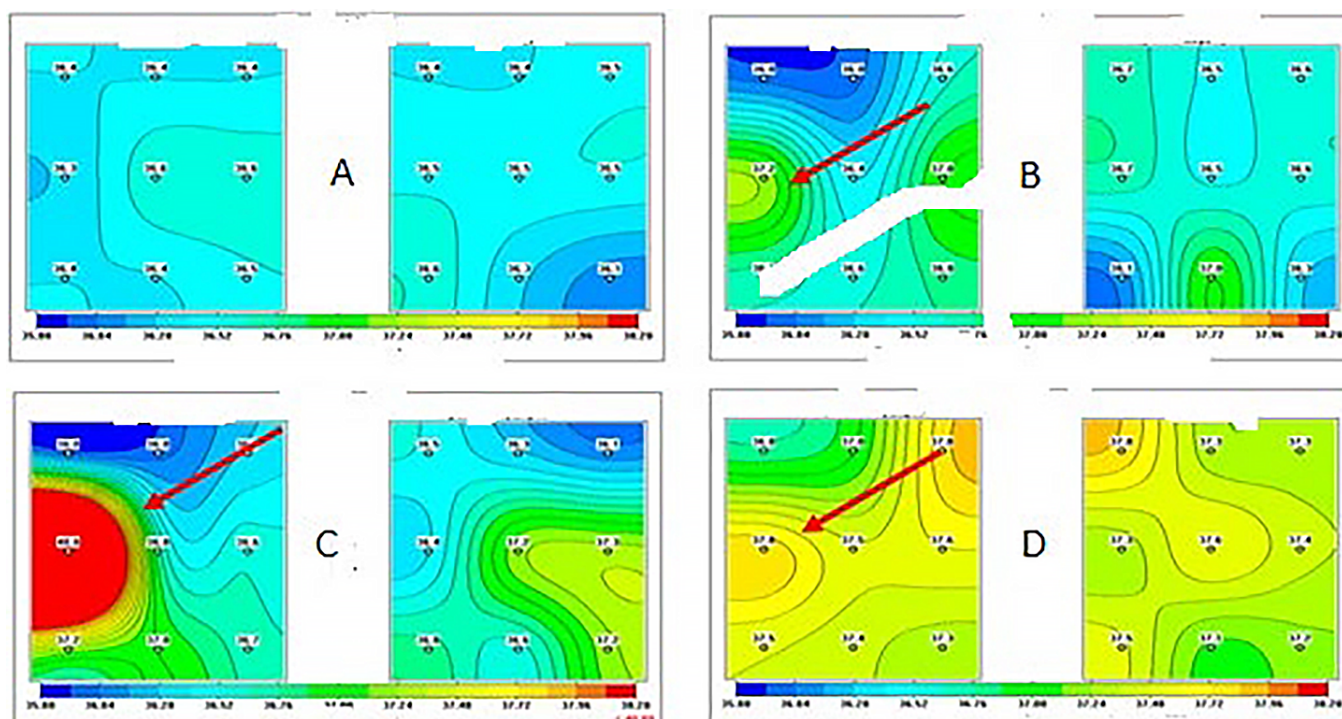


Fig. 1. Example of thermal maps obtained by microwave radiothermometry. A – before training, B – after a 20-minute warm-up workout, C – after sparring, and D – one hour after sparring. The arrow marks the area of hyperthermia typical for this athlete (according to Shevelev O.A. et al. [39])

corded using medical microwave radiometry (MWR). Athletes performed the PWC-170 test. Temperature measurements showed that the axial temperature after the test increased from 36.21 ± 0.07 °C to 37.67 ± 0.06 , and the brain temperature from 36.58 ± 0.07 °C to 38.17 ± 0.08 °C, which is higher than body temperature.

With an interval of a day, a second study was carried out on the same athletes, and the exercise test was preceded by a 60-minute CCH session. 20–30 minutes later (the period of spontaneous brain warming) after the hypothermia session, the athletes were asked to perform the PWC-170 test. At this stage of the study, after exercise, the axial temperature increased to 37.23 ± 0.03 °C, and the brain — up to 37.60 ± 0.07 °C.

These data demonstrate that the preventive CCH session allowed for a reduction in the severity of general and cerebral hypothermia caused by the test load. In addition, the CCH session preceding the PWC-170 test provided a significant increase in maximum oxygen

consumption by 9.5 %, the power of work performed at the aerobic threshold by 13.5 %, and at the anaerobic threshold by 5.6 %, compared with the results obtained during the test without a preventive hypothermia session.

The facts that preventive brain hypothermia can reduce the degree of development of physical general and cerebral hyperthermia, as well as increase aerobic and anaerobic performance, are extremely important in terms of optimizing the training of athletes and in the recovery period.

The introduction of single sessions and course use of CCH into the training programs for athletes can reduce the risks associated with working hyperthermia and overheating, improve sports performance, and protect the athlete's brain from development of negative consequences of accidental and “planned” (martial arts) sports-related TBI of varying severity.

In particular, after sparring, in which missed blows to the head were registered, the athlete's brain tempera-

ture in the focus of hyperthermia reached 38.1 ± 0.13 °C, and after the CCH session, it was 35.8 ± 0.25 °C. These facts are quite remarkable since they demonstrate the possibility of stopping focal hyperthermia, which is the

basis for preventing the development of sports-related TBI complications. An example of an athlete's brain temperature map is shown in Fig. 2.

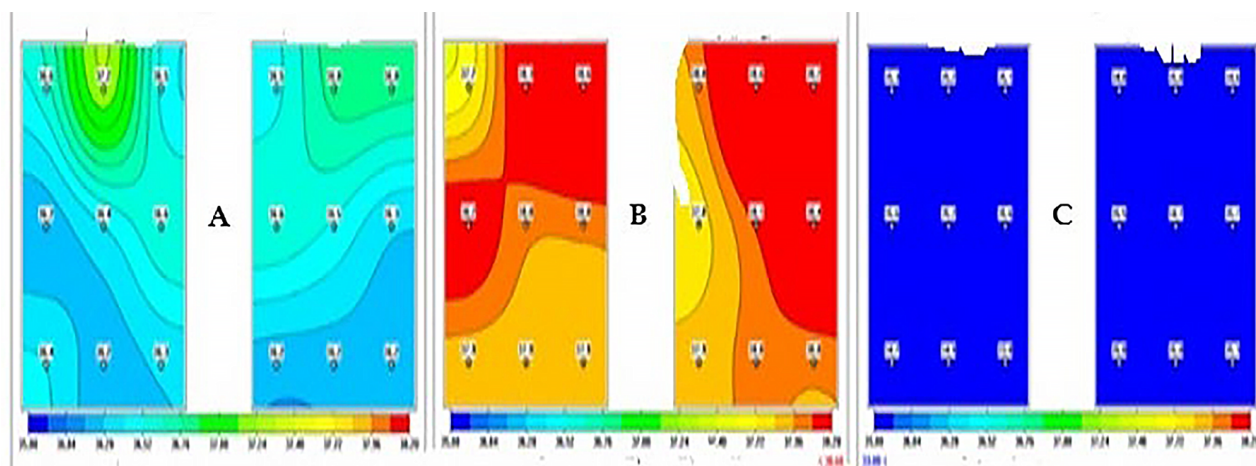


Fig. 2. Thermal maps obtained from athletes by using microwave radiothermometry.

A – before sparring, B – after sparring (3 rounds of 3 minutes each), and C – after 60 minutes of CCH, carried out immediately after sparring (according to Shevelev O.A. et al. [39])

It is notable that particularly in sports during trainings and competitive cycles is possible to bring the time of SCCH induction as close as possible to the moment of injury, what is fundamentally important, since the earlier the hypothermia procedure is started, the better the clinical effects will be [40].

Conclusion

MWR of the brain can serve as an objective tool for diagnosing sports-related mild TBI. Therapeutic hypothermia, used for cerebro-protection after total circulatory arrest, in cases of cerebral circulation disorders and brain injury, has long been known. The mechanisms of its action have been thoroughly studied, including urgent effects that develop during hypothermia, and delayed effects, i. e., molecular mechanisms based on the initiation of the expression of early response genes encoding stress-protective proteins by low temperatures. The accumulation of stress proteins prolongs the action of hypothermia, which is responsible for the effects of preventive cooling, and the increase in the resistance

of cells and tissues to the action of damaging factors is due to a wide range of cytoprotective reactions that develop with their participation. The evidence base for the effectiveness of hypothermia comes mainly from animal experiments and tissue culture and to fully extrapolate the results in relation to sports-related TBI, special extensive studies are required; however, given the potential risks of brain injuries due to sports-related TBI consequences and available clinical experience on hypothermia technology, it is advisable to recommend it for a wider application in sports medicine and rehabilitation.











References/Библиографический список

1. Theadom A, Mahon S, Hume P, Starkey N, Barker-Collo S, Jones K, Majdan M, Feigin VL. Incidence of Sports-Related Traumatic Brain Injury of All Severities: A Systematic Review. *Neuroepidemiology*. 2020;54(2):192–199. doi: 10.1159/000505424.
2. Brazinova A, Rehorcikova V, Taylor MS, Buckova V, Majdan M, Psota M, Peeters W, Feigin V, Theadom A, Holkovic L, Synnot A. Epidemiology of Traumatic Brain Injury in Europe: A Living Systematic Review. *J Neurotrauma*. 2021;38(10):1411–1440. doi: 10.1089/neu.2015.4126.

3. Potapov AA, Lichterman LB, Kravchuk AD, Okhlopkov VA, Aleksandrova EV, Filatova MM, Maryakhin AD, Latyshev YA. Mild traumatic brain injury: *clinical recommendations*. — Moscow: Association of Neurosurgeons of Russia, 2016. (In Russian). [Потапов А.А., Лихтерман Л.Б., Кравчук А.Д., Охлопков В.А., Александрова Е.В., Филатова М.М., Маряхин А.Д., Латышев Я.А. Лёгкая черепно-мозговая травма: клинические рекомендации // Ассоциации нейрохирургов России. 2016. С. 23].
4. Clinical Guidelines. Concussion of the brain. Association of Neurosurgeons of Russia, Approved by the Ministry of Health of the Russian Federation, revision 2021. (In Russian). [Клинические рекомендации. Сотрясение головного мозга // Ассоциация нейрохирургов России, утверждена Министерством здравоохранения Российской Федерации, 2021. Москва. 42 с.].
5. Ugryumov VM. Severe closed injury of the skull and brain. Leningrad: *Medicine*, 1974, 318 p. (In Russian). [Угрюмов В.М. Тяжелая закрытая травма черепа и головного мозга // Ленинград: Медицина. 1974. С.318].
6. Sun YJ, Zhang ZY, Fan B, Li GY. Neuroprotection by Therapeutic Hypothermia. *Front Neurosci*. 2019;13:586. doi:10.3389/fnins.2019.00586.
7. Dietrich WD, Bramlett HM. Therapeutic hypothermia and targeted temperature management for traumatic brain injury: Experimental and clinical experience. *Brain Circ*. 2017;3(4):186–198. doi:10.4103/bc.bc_28_17.
8. Lee JH, Zhang J, Yu SP. Neuroprotective mechanisms and translational potential of therapeutic hypothermia in the treatment of ischemic stroke. *Neural Regen Res*. 2017;12(3):341–350. doi:10.4103/1673–5374.202915.
9. Vaity C, Al-Subaie N, Cecconi M. Cooling techniques for targeted temperature management post-cardiac arrest. *Crit Care*. 2015;19(1):103. Published 2015 Mar 16. doi: 10.1186/s13054–015–0804–1.
10. Hine K, Hosono S, Kawabata K, Miyabayashi H, Kanno K, Shimizu M, Takahashi S. Nasopharynx is well-suited for core temperature measurement during hypothermia therapy. *Pediatr Int*. 2017;59(1):29–33. doi:10.1111/ped.13046.
11. Ibragimov N.K. Craniocerebral hypothermia + nasopharyngeal cooling: effects on cerebral blood flow, intracranial pressure, cerebral perfusion pressure in patients with craniocerebral trauma. *Central Asian Journal of Medicine*. 2018;4:47–56. <https://uzjournals.edu.uz/tma/vol2018/iss4/5>.
12. Gard A, Tegner Y, Bakhsheshi MF, Marklund N. Selective head-neck cooling after concussion shortens return-to-play in ice hockey players. *Concussion*. 2021;6(2): CNC90. doi: 10.2217/cnc-2021–0002.
13. Shevelev OA, Saidov SK, Petrova MV, Chubarova MA, Usmanov ES. Craniocerebral hypothermia as a method of therapy of disorders of the temperature balance of the brain in patients in the postcomatous period. *Physical and rehabilitation medicine, medical rehabilitation*. 2020;2(1):11–19. doi: <https://doi.org/10.17816/rehab20411>. (In Russian). [Шевелев О.А., Саидов Ш.Х., Петрова М.В., Чубарова М.А., Усманов Э.Ш. Краниocerebralная гипотермия как метод терапии нарушений температурного баланса головного мозга у пациентов в посткоматозном периоде // Физическая и реабилитационная медицина, медицинская реабилитация. 2020. Т. 2. № 1. С. 11–19. doi: <https://doi.org/10.17816/rehab20411>.].
14. Mrozek S, Vardon F, Geeraerts T. Brain temperature: physiology and pathophysiology after brain injury. *Anesthesiol Res Pract*. 2012;2012:989487. doi: 10.1155/2012/989487.
15. Guatteo E, Chung KK, Bowala TK, Bernardi G, Mercuri NB, Lipski J. Temperature sensitivity of dopaminergic neurons of the substantia nigra pars compacta: involvement of transient receptor potential channels. *J Neurophysiol*. 2005;94(5):3069–3080. doi: 10.1152/jn.00066.2005.
16. Fohlmeister JF, Cohen ED, Newman EA. Mechanisms and distribution of ion channels in retinal ganglion cells: using temperature as an independent variable. *J Neurophysiol*. 2010;103(3):1357–1374. doi:10.1152/jn.00123.2009.
17. Yu Y, Hill AP, McCormick DA. Warm body temperature facilitates energy efficient cortical action potentials. *PLoS Comput Biol*. 2012;8(4): e1002456. doi: 10.1371/journal.pcbi.1002456.
18. Craig AD, Chen K, Bandy D, Reiman EM. Thermosensory activation of insular cortex. *Nat Neurosci*. 2000;3(2):184–190. doi:10.1038/72131.
19. Kiyatkin EA. Brain temperature homeostasis: physiological fluctuations and pathological shifts. *Front Biosci (Landmark Ed)*. 2010;15(1):73–92. doi: 10.2741/3608.
20. Nybo L. Brain temperature and exercise performance. *Exp Physiol*. 2012;97(3):333–339. doi:10.1113/expphysiol.2011.062273.
21. Hayward JN, Baker MA. Role of cerebral arterial blood in the regulation of brain temperature in the monkey. *Am J Physiol*. 1968;215(2):389–403. doi: 10.1152/ajplegacy.1968.215.2.389.
22. Cabanac M, Brinell H. Blood flow in the emissary veins of the human head during hyperthermia. *Eur J Appl Physiol Occup Physiol*. 1985;54(2):172–176. doi: 10.1007/BF02335925.
23. Janssen FE, Van Leeuwen GM, Van Steenhoven AA. Modelling of temperature and perfusion during scalp cooling. *Phys Med Biol*. 2005;50(17):4065–4073. doi:10.1088/0031–9155/50/17/010.
24. Weiwu Ma, Wenxin Liu, Min Li. Analytical heat transfer model for targeted brain hypothermia. *International Journal of Thermal Sciences*. 2016;100:66–74. doi: 10.1016/j.ijthermalsci.2015.09.014.
25. Uyğun M, Küçüka MS, Çolpan CÖ. “3B modeling and temperature distribution of human brain”. 2016. *20th National Biomedical Engineering Meeting (BIYOMUT)*. Izmir, Turkey. 2016. pp. 1–4. doi: 10.1109/BIYOMUT.2016.7849378.

26. Vesnin SG, Sedankin MK. Development of a series of antenna applicators for non-invasive measurement of the temperature of human body tissues in various pathologies. *Bulletin of the Bauman Moscow State Technical University*. 2012; 43–61. (In Russian). [Веснин С.Г., Сединкин М.К. Разработка серии антенных аппликаторов для неинвазивного измерения температуры тканей организма человека при различных патологиях // Вестник Московского государственного технического университета им. Н.Э. Баумана. 2012. № 1. С. 43–61].
27. Polyakov MV, Khoperskov AV. Mathematical modeling of the spatial distribution of the radiation field in biological tissue; determination of the brightness temperature for diagnostics, *Vestn. Volgogr. state University*. Ser. 1, Mat. Phys.. 2016; 5(36): 73–84. doi.org/10.15688/jvolsu1.2016.5.7. (In Russian). [Поляков М.В., А.В. Хоперсков А.В. Математическое моделирование пространственного распределения поля излучения в биологической ткани: определение яркостной температуры для диагностики // Вестник Волгоградского государственного университета. Серия 1: Математика. Физика. 2016. Т. 5. № 36. С 73–84. doi.org/10.15688/jvolsu1.2016.5.7.].
28. Maloney SK, Mitchell D, Mitchell G, Fuller A. Absence of selective brain cooling in unrestrained baboons exposed to heat. *Am J Physiol Regul Integr Comp Physiol*. 2007;292(5): R2059–R2067. doi:10.1152/ajpregu.00809.2006.
29. Shevelev OA, Butrov AV, Cheboksary DV, Khodorovich NA, Lapaev NN, Pokatilova NS. Pathogenetic role of cerebral hyperthermia in brain lesions. *Clinical medicine*. 2017;(95)4:302–309. (In Russian). [Шевелев О.А., Бутров А.В., Чебоксаров Д.В., Ходорович Н.А., Лапаев Н.Н., Покатилова Н.С. Патогенетическая роль церебральной гипертермии при поражениях головного мозга // Клиническая медицина. 2017. № 4. С. 302–309].
30. Sharma HS. Hyperthermia induced brain oedema: current status and future perspectives. *Indian J Med Res*. 2006;123(5):629–652.
31. Bain AR, Morrison SA, Ainslie PN. Cerebral oxygenation and hyperthermia. *Front Physiol*. 2014;5:92. doi:10.3389/fphys.2014.00092.
32. Nybo L, Nielsen B. Middle cerebral artery blood velocity is reduced with hyperthermia during prolonged exercise in humans. *J Physiol*. 2001;534(Pt 1):279–286. doi:10.1111/j.1469–7793.2001.t01–1–00279.x.
33. Campos F, Pérez-Mato M, Agulla J, Blanco M, Barral D, Almeida A, Brea D, Waeber C, Castillo J, Ramos-Cabrer P. Glutamate excitotoxicity is the key molecular mechanism which is influenced by body temperature during the acute phase of brain stroke. *PLoS One*. 2012;7(8): e44191. doi:10.1371/journal.pone.0044191.
34. Konov AV, Shevelev OA, Smolensky AV, Belichenko OI, Tarasov AV, Khusyainov ZM, Garakyan AI. The use of local therapeutic craniocerebral hypothermia for the prevention of complications of mild traumatic brain injury in sports. *Therapist*. 2015;11:21–28. (In Russian). [Конов А.В., Шевелев О.А., Смоленский А.В., Беличенко О.И., Тарасов А.В., Хусьяинов З.М., Гаракян А.И. Использование локальной терапевтической краниocereбральной гипотермии для профилактики осложнений легкой черепно-мозговой травмы в спорте // Терапевт. 2015. № 11. С. 21–28].
35. Smolensky AV, Shevelev OA. Approaches to the prevention of mild traumatic brain injuries in basketball/ *Collection of articles based on the materials of the III International Scientific and Practical Conference “Integration processes of Science and practice”*. Moscow, November 25, 2020. (In Russian). [Смоленский А.В., Шевелев О.А. Подходы к профилактике легких черепно-мозговых травм в баскетболе // Сборник статей по материалам III Международной научно-практической конференции “Интеграционные процессы науки и практики”. 2020 г. Москва].
36. Shevelev OA, Smolensky AV, Tarasov AV, Miroshnikov AB, Khusyainov ZM, Garakyan AI. Temperature balance of the cerebral cortex in athletes boxers during training and competitions. *Sports and pedagogical education*. 2020;4:59–66. (In Russian). [Шевелев О.А., Смоленский А.В., Тарасов А.В., Мирошников А.Б., Хусьяинов З.М., Гаракян А.И. Температурный баланс коры головного мозга спортсменов-боксеров во время тренировок и соревнований // Спортивное и педагогическое образование. 2020. № 4. С. 59–66].
37. Smolensky AV, Shevelev OA, Tarasov AV, Miroshnikov AB, Kuzovleva EV, Khusyainov ZM. Optimization of post-exercise recovery and approaches to the prevention of complications of mild traumatic brain injury in boxing. *Almanac Sport*. 2020;32–34. (In Russian). [Смоленский А.В., Шевелев О.А., Тарасов А.В., Мирошников А.Б., Кузовлева Е.В., Хусьяинов З.М. Оптимизация восстановления после тренировки и подходы к профилактике осложнений легкой черепно-мозговой травмы в боксе // Спортивный альманах. 2020. С. 32–34].
38. Smolensky AV, Shevelev OA, Tarasov AV, Miroshnikov AB, Kuzovleva EV. Optimization of post-loading recovery in boxing. *Theory and methodology of impact sports of martial arts / Collection of articles based on the materials of the “All-Russian Scientific and Practical Conference with international participation”*; Moscow. 2021:100–105. (In Russian). [Смоленский А.В., Шевелев О.А., Тарасов А.В., Мирошников А.Б., Кузовлева Е.В. Оптимизация постнагрузочного восстановления в боксе. Теория и методика ударных видов спортивных единоборств // Сборник статей по материалам «Всероссийской научно-практической конференции с международным участием». Москва. 2021. С.100–105].
39. Shevelev OA, Grechko AV, Petrova MV. *Therapeutic hypothermia. Monograph*. Moscow: RUDN. 2020. 272 p. (In Russian). [Шевелев О.А., Гречко А.В., Петрова М.В. Терапевтическая гипотермия: монография. Москва: РУДН. 2020. С. 272].
40. Jackson TC, Kochanek PM. A New Vision for Therapeutic Hypothermia in the Era of Targeted Temperature Management: A Speculative Synthesis. *Ther Hypothermia Temp Manag*. 2019;9(1):13–47. doi:10.1089/ther.2019.0001.

Диагностика и профилактика осложнений спортивной черепно-мозговой травмы

О.А. Шевелев^{1,2} , А.В. Смоленский³ , М.В. Петрова^{1,2} , Э.М. Менгисту^{1,2}  ✉
А.А. Менгисту² , М.В. Вацик-Городецкая^{1,4} , У.Г. Ханахмедова⁴ , Д.Н. Менжуренкова¹ ,
С.Г. Веснин⁵ , И.И. Горянин^{6,7} 

¹Российский университет дружбы народов, г. Москва, Российская Федерация

²Федеральный научно-клинический центр реаниматологии и реабилитологии, г. Москва, Российская Федерация

³Университет физической культуры, спорта, молодежи и туризма, г. Москва, Российская Федерация

⁴Городская клиническая больница имени В.В. Виленко, г. Москва, Российская Федерация

⁵ООО Медицинская микроволновая радиометрия, г. Эдинбург, Великобритания

⁶Школа информатики Эдинбургского университета, г. Эдинбург, Великобритания

⁷Институт науки и технологий г. Окинавы, г. Окинава, Япония

✉ drmengistu@mail.ru

Аннотация. На долю спортивных черепно-мозговых травм (ЧМТ) приходится до 20 % всех спортивных повреждений, а встречаемость первых возрастает ежегодно за счет прироста числа людей, занимающихся спортом, растущей популярности экстремальных и контактных видов спорта, а также высокого уровня мотивации на достижение рекордных результатов среди молодых спортсменов. Основная цель данного обзора — представить возможности применения методики микроволновой радиотермометрии и технологии краниocereбральной гипотермии в контексте спортивной ЧМТ. В обзоре рассмотрена наиболее часто встречающаяся форма ЧМТ у спортсменов — легкая ЧМТ, которая в свою очередь может спровоцировать развитие очень широкого спектра осложнений и негативных последствий как в ближайшем, так и отсроченном периоде после полученной травмы. Рассмотрены основные недостатки программ по профилактике осложнений при лечении и реабилитации спортсменов после ЧМТ, которые недостаточно учитывают особенности механизмов развития самой травмы, ее значимые отличия от бытовых, дорожных или криминальных травм с повреждением головного мозга. Авторами описана актуальная проблематика отсутствия объективных методов инструментальной диагностики тяжести травмы. Детализована патофизиологическая составляющая, характерная для спортивной ЧМТ: периодичность повторения, повышение температуры тела и мозга пострадавшего, периферическое перераспределение кровотока и гипокания, которые значимо влияют на мозговой кровоток. На основании проведенного анализа имеющейся отечественной и зарубежной научной литературы можно сделать вывод, что черепно-мозговая травма является независимой причиной развития церебральной гипертермии, которая существенно усугубляет последствия полученной травмы. **Выводы.** Авторами предложен новаторский способ использования метода микроволновой радиотермометрии в качестве диагностического инструмента спортивных ЧМТ. Кроме того, в обзоре выделяются основные рекомендации для профилактики осложнений с использованием технологии краниocereбральной гипотермии, которая позволяет снизить общую физическую и церебральную гипертермию, а также способствует повышению устойчивости нейронов коры головного мозга к гипоксии и травме. Однако авторы считают, что описываемые подходы в спортивной медицине используются нецеленаправленно и связано это с недостаточной осведомленностью тренеров и врачей спортивных команд.

Ключевые слова: гипотермия, виды спорта, травматическое повреждение мозга, реабилитация, гипертермия

Информация о финансировании. Авторы не получали финансовой поддержки для исследования и публикации данной статьи.

Вклад авторов: Шевелев О.А., Горянин И.И. — концепция и написание исходного текста; Петрова М.В., Смоленский А.В. — кураторство; Веснин С.Г. — курирование данных; Менгисту Э.М., Менгисту А.А. — написание, рецензирование и редактирование; Вацик-Городецкая М.В., Ханахмедова У.Г., Менжуренкова Д.Н. — сбор данных и формальный анализ.

Все авторы внесли значительный вклад в разработку концепции, исследования и подготовку рукописи, прочитали и утвердили окончательную версию перед публикацией.

Информация о конфликте интересов. Авторы заявляют об отсутствии конфликта интересов.

Этическое утверждение — неприменимо.

Благодарности — неприменимо.

Информированное согласие на публикацию — неприменимо.

Поступила 19.03.2023. Принята 24.04.2023.

Для цитирования: *Shevelev O.A., Smolensky A.V., Petrova M.V., Mengistu E.M., Mengistu A.A., Vatsik-Gorodetskaya M.V., Khanakhmedova U.G., Menzhurenkova D.N., Vesnin S.G., Goryanin I.I.* Diagnostics and prevention of sports-related traumatic brain injury complication // Вестник Российского университета дружбы народов. Серия: Медицина. 2023. Т. 27. № 2. С. 254—264. doi: 10.22363/2313-0245-2023-27-2-254-264

Corresponding author: Mengistu Elias Mesfin — PhD student, Department of Anaesthesiology and Intensive Care, Institute of Medicine, RUDN University, 117198, Miklukho-maklaya st., 6, Moscow, Russian Federation. Email: drmengistu@mail.ru
Shevelev O.A. ORCID 0000-0002-6204-1110
Smolensky A.V. ORCID 0000-0001-5663-9936
Petrova M.V. ORCID 0000-0003-4272-0957
Mengistu E.M. ORCID 0000-0002-6928-2320
Mengistu A.A. ORCID 0000-0001-8999-6972
Vatsik-Gorodetskaya M.V. ORCID 0000-0002-6874-8213
Khanakhmedova U.G. ORCID 0009-0002-4893-2846
Menzhurenkova D.N. ORCID 0009-0002-7997-0079
Vesnin S.G. ORCID 0000-0003-4353-8962
Goryanin I.I. ORCID 0000-0002-8293-774X

Ответственный за переписку: Менгисту Эльяс Месфин — аспирант, кафедра анестезиологии и реаниматологии с курсом медицинской реабилитации, Медицинский институт РУДН, Российская Федерация, 117198, Москва, ул. Миклухо-Маклая, д-6. E-mail: drmengistu@mail.ru
Шевелев О.А. SPIN-код 9845-2960; ORCID 0000-0002-6204-1110
Смоленский А.В. SPIN-код 4514-3020; ORCID 0000-0001-5663-9936
Петрова М.В. SPIN-код 9132-4190; ORCID 0000-0003-4272-0957
Менгисту Э.М. SPIN-код 1387-7508; ORCID 0000-0002-6928-2320
Менгисту А.А. ORCID 0000-0001-8999-6972
Вацик-Городецкая М.В. SPIN-код 5531-0698; ORCID 0000-0002-6874-8213
Ханахмедова У.Г. ORCID 0009-0002-4893-2846
Менжуренкова Д.Н. ORCID 0009-0002-7997-0079
Веснин С.Г. ORCID 0000-0003-4353-8962
Горянин И.И. ORCID 0000-0002-8293-774X