

## SPORTS CRANIO-BRAIN INJURY. DIAGNOSTICS AND PREVENTION OF COMPLICATIONS

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### Abstracts

Sports traumatic brain injury (TBI) accounts for up to 20% of all sports injuries, and the number of cases is growing year by year due to an increase in the number of people involved in sports, the growing popularity of extreme and contact sports, as well as a high level of motivation to achieve record results. Mild TBI dominates, which can provoke the development of a very wide range of complications and negative consequences. In the programs for the prevention of complications and the rehabilitation of athletes after TBI, TBI features are not sufficiently considered, which significantly differ from household, road or criminal injuries. There are no instrumental methods for diagnosing the severity of the injury. Sports TBI is characterized by repeated frequent TBI, elevated body and brain temperature, peripheral redistribution of blood flow and hypocapnia, which significantly affect cerebral blood flow. Brain injury is an independent cause of the development of cerebral hyperthermia, which significantly worsens the consequences of TBI. To diagnose sports TBI, the method of microwave radiometry of the brain (MWR) can be used. For the prevention of complications, the technology of craniocerebral hypothermia (CCH), which allows for to reduce of physical general and cerebral hyperthermia, and increases the resistance of cerebral cortex neurons to hypoxia and trauma could be used. However, these approaches in sports medicine are used sporadically, which, is due to the lack of awareness of coaches and doctors of sports teams.

The purpose of the review is to present MWR and CCH in sports TBI.

### Keywords

hypothermia, sports, traumatic brain injury, rehabilitation, hyperthermia

### 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 classified as mild TBIs (MTBIs). The neurological symptoms of CVBI are often mild, and injured young, strong, highly motivated athletes tend to downplay the injury. This can cause an underestimation of the severity and extent of injuries received by a doctor or 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 light TBI occurs in a short time (within 1-2 weeks), and in 5-20% of victims, symptoms of post-concussion syndrome (cognitive, emotional, and behavioural disorders) are noted for a long time. The severity of TBI is most often assessed using the Glasgow Coma Scale, and LTBI corresponds to a score of 13-15 points in the acute period after injury. Metabolic, ionic, and neurotransmitter disorders and neuroinflammation develop in light TBI, but changes on CT and 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 centimetres from the skin surface [5].

At present, in sports light TBI 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 light 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. [6,7,8]. 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 light 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 known as the CCG technique 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 selectively use CCH without cooling the neck, which has positively proven itself in the treatment of the most acute period of 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 light TBI.

Thus, there are convincing prerequisites that MWR and CCH can be used to diagnose sports light 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 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 [17,18,19].

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 pathological 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 NMR spectroscopy, 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 modelling 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 RTM 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 LTBI and the use of CCH

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 blood PCO<sub>2</sub> 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 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 (IL1, IL6) 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 TBI, especially in martial arts, it is typical to receive repeated injuries in short periods.

Thus, the specific features of sports 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 behaviour of athletes seeking to continue participating in training and competitive cycles, which can cause underestimation of the severity of the injury.

After sports LBI, 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 RTM when the 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 light TBI (Fig. 1).

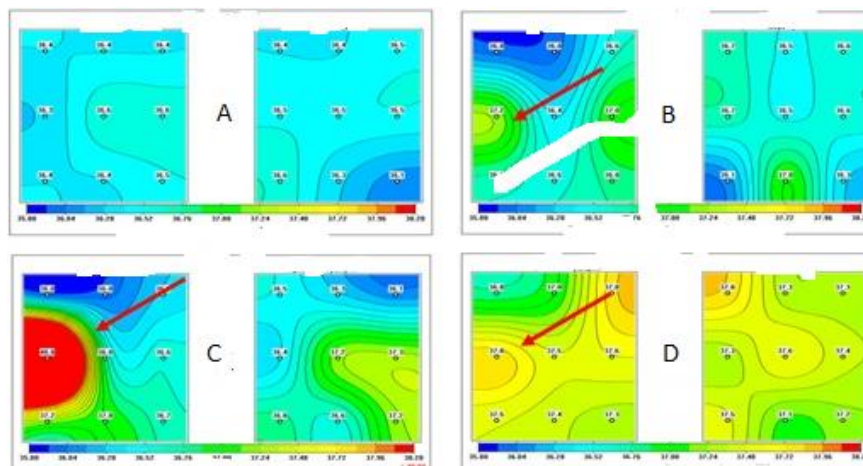


Figure 1

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])

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 recorded using medical microwave radiometry (MWR). Athletes performed the PWC-170 test. Temperature measurements showed that the axial temperature after the test increased from  $36.21 \pm 0.07^\circ\text{C}$  to  $37.67 \pm 0.06$ , and the brain temperature from  $36.58 \pm 0.07^\circ\text{C}$  to  $38.17 \pm 0.08^\circ\text{C}$ , 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 CCG 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  $37.23 \pm 0.03^\circ\text{C}$ , and the brain - up to  $37.60 \pm 0.07^\circ\text{C}$ .

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 CCG 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.

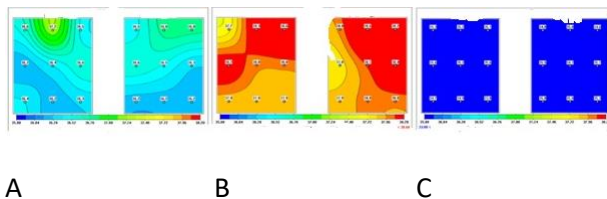
The facts that preventive brain hypothermia can reduce the degree of development of physical general and cerebral hyperthermia, as well as increase aerobic and anaerobic performance, are extremely important in terms of optimizing the training of athletes and in the recovery period.

The introduction of single sessions and course use of CCG into the training programs for athletes can reduce the risks associated with working hyperthermia and overheating, improve sports



performance, and also protect the brain of athletes from the development of the negative consequences of accidental and “planned” (martial arts) sports TBI of varying severity.

In particular, after sparring, in which missed blows to the head were registered, the temperature of the brain of athletes in the focus of hyperthermia reached  $38.1 \pm 0.13^\circ\text{C}$ , and after the CCG session, it was  $35.8 \pm 0.25^\circ\text{C}$ . These facts are quite remarkable since they demonstrate the possibility of stopping focal hyperthermia, which is the basis for preventing the development of sports TBI complications. An example of an athlete's brain temperature map is shown in Fig. 2.



**Figure 2.** 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 essential that in sports when planning training and competitive cycles, it is possible to bring the time of CCG to the moment of injury as close as possible, and this is fundamentally important, since the earlier the hypothermia procedure is started, the better the clinical effects of its use.

## Conclusion

MWR of the brain can serve as an objective tool for diagnosing sports light 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 [40]. 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. To fully extrapolate the results in relation to sports TBI, special extensive studies are required, however, given the potential risks of the consequences of sports brain injuries and the available experience in the clinical application of this hypothermia technology, it is advisable to recommend it for wider use in sports medicine and rehabilitation with control of thermal disorders. brain balance with MWR.

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