The Physiological Response of the Body to Low Temperatures

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Abstract

For residents of all northern countries, a great biological hazard is the cold, which negatively affects all the functions of the body. Fatal outcomes when exposed to the cold are very often associated with alcohol consumption. At the same time, the number of survivors, but those who become disabled due to supercooling, seriously exceeds the number of dead. In the case of progressive hypothermia in the human body, a number of negative reactions occur, leading to a drop in temperature, depletion of physical reserves of the heart, liver, muscles, breathing and heartbeat, reduce the tone of vessels and reduce the blood flow rate. All this leads to stasis of blood, oxygen starvation of tissues due to the excessive connection of oxygen with hemoglobin. Under the influence of cold, frostbite most often occurs, manifested by reactive inflammation and necrosis. The main cause is the cooling of the body surface and the thermoregulation disruption. In the course of the emerging necrosis, the role of tissue edema is great. In addition, with the influence of cold, the vessel spasms develop, the bloodstream slows down and the tissue ischemia is growing. Physical rehabilitation after the action of the cold should be carried out very actively. Its success is associated with the complexity and perseverance of the use of therapeutic physical culture, massage, and various physiotherapy.

Keywords: Low temperatures, Hypothermia, Frostbite, Body reaction

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Introduction

For the population of the northern countries, the effect of the cold is a great danger. More than one and a half thousand people die every year as a result of hypothermia in Russia. Fatalities are mainly associated with alcohol consumption. The number of people with disabilities due to supercooling is much greater than the number of dead (Medvedev & Gromnatskii, 2005; Mal *et al.*, 2018).

The cooling capacity of the external environment increases when the air temperature decreases in the case of its high humidity and wind speed (Mal *et al.*, 2020). At an air temperature of 0°C, fatal cooling occurs in 10-12 hours. In water, it develops earlier due to its higher thermal conductivity. In ice water, a person dies after 5-10 minutes (Amelina & Medvedev, 2009; Medvedev, 2016).

With progressive hypothermia in the human body, the body temperature is dropped, the depletion of functional reserves of the heart, liver, muscles, breathing and heartbeat, weakening the tone of the vessels. This leads to a weakening of the return of oxygen by erythrocytes tissues (Vorobyeva & Medvedev, 2018). All these changes are strongly oppressed by the thermal control ability of the brain. (Makhov & Medvedev, 2018a). As a result, there comes a moment when the ability to maintain body temperature at a constant level is lost and freezing develops with the extinction of vital functions (Mal, *et al.*, 2018). A "biological zero" of vital activity is achieved – this is the temperature of the tissue at which its activity reversibly ceases. The goal of the work is to consider aspects of the physiological reaction of the human body to low temperatures and the basics of physical rehabilitation after this condition.

Materials and Methods

The material for this work was published by published articles in open access. The search for literary sources was conducted in the database of the scientific electronic library eLIBRARY.RU, in the science of scientific information Scopus and in the Scientific Information Base of Web of Science. The study applied the following methods for obtaining information: induction and deduction, analysis, synthesis and generalizations.

Results and Discussion



In the conditions of the influence of cold, frostbite is often raised-damage to the tissues of the body, which is manifested by reactive inflammation and tissue necrosis (Makhov & Medvedev, 2018b). The main cause of frostbite is the cooling of the body surface and the violation of thermoregulation (Oshurkova & Medvedev, 2018b). In conditions of frostbite, there is no instant damage and fabric death (Fayazi *et al.*, 2019; Lenchenko *et al.*, 2019; Fayzullina *et al.*, 2020; Kalantarzadeh *et al.*, 2020; Karpov *et al.*, 2020).

In the pathogenesis of frostbite, the main role belongs to circulatory disorders. The cold leads to the vessels spasm, slowdown blood flow and complete inefficiency of blood circulation. At a temperature of +8°C, dissociation of oxyhemoglobin and oxygen transfer to tissues ceases. Warming up the damaged area does not stop the pathological mechanisms. The result is stasis, red blood cell aggregation, and thrombosis. The progression of thrombosis is due to activation of the hemostatic system and inhibition of fibrinolysis (Oshurkova & Medvedev, 2018a).

During the frostbite, three phases can be traced: the phase of inflammation, the phase of development of necrosis and its delimitation, and the phase of scarring and epithelization.

The phase of inflammation develops after the restoration of temperature. She is characterized by all signs of the inflammatory process - pain, swelling, hyperemia, local temperature increase, and impaired function. One of the pathogenetic factors in this phase is pain (Bespalov *et al.*, 2018a). Pain syndrome causes a violation of the functions of various body systems (Alanazi *et al.*, 2019; Tkacheva & Medvedev, 2020). With extensive and deep frostbite, shock can develop. Swelling of tissues in the phase of inflammation is localized not only in the skeleton of damaged tissues, but also spreads proximal and leads to secondary necrosis. It contributes to the development of toxemia, which disrupts the functions of internal organs (Makhov & Medvedev, 2018d).

The phase of development of necrosis and its delimitation can last several months. Locally formed areas of necrosis and their delimitation from healthy tissues. A demarcation line appears. From the necrosis zone, toxins enter the general bloodstream, and worsening the general condition. In addition, tissues that have undergone necrosis are a good breeding ground for all types of microorganisms. Therefore, complications of a purulent-inflammatory nature with the development of superficial suppuration, phlegmon, and abscesses are possible in this phase (Makhov & Medvedev, 2018c).

The scarring or epithelialization phase begins immediately after the rejection of necrotic tissues or their removal. Locally, the processes of regeneration and epithelization prevail. In the case of a small area of damage, the general condition practically does not suffer. With extensive frostbite, the general clinical manifestations are due to the development of complications (Medvedev, 2018c).

With the development of toxemia, typical signs of intoxication are observed: tachycardia, decreased blood pressure, fever, and severe leukocytosis. Signs of impaired function of the cardiovascular system, liver, and kidneys may be detected. Violations of the coagulation and anticoagulation systems of the blood are detected (Medvedev, 2018d).

Physical rehabilitation after a sharp cooling of the body should be actively carried out. Damaged limbs must be immobilized using improvised means (Medvedev, 2018e).

After establishing the depth of the lesion, the area and depth of frostbite are estimated. If they are relatively small and there is no threat of complications, a wait-and-see position is chosen (Medvedev, 2018b).

When frostbite is very often used therapeutic physical culture. It is shown regardless of the degree, location, and area of frostbite. It is prescribed for both surgical and conservative treatment (Medvedev, 2018a).

There are 3 periods of the use of physiotherapy exercises for frostbite.

Period I lasts 3-4 days. Here, medical physical culture has a restorative effect and provides prevention of complications. Actively apply breathing exercises for the prevention of pneumonia and exercises for the distal limb (Makhov & Medvedev, 2020).

Period II lasts from 3-4 days to the recovery period. Here, therapeutic physical culture normalizes brain activity, prevents complications, improves the functions of breathing and blood circulation. Simple exercises are used to perform, exclude breathholding, and straining (Agronina *et al.*, 2020).

Period III lasts up to 3 months. In this period, therapeutic physical culture improves trophism in tissues, maintains joint mobility, and prevents violations of their immobility. It can be started 24 hours after surgery (Glagoleva & Medvedev, 2020).

Massage therapy for frostbite begins at the stage of scarring. It helps to resolve scars, reduces pain, reduces swelling, and restores skin. Therapeutic massage for frostbite is carried out by stroking and rubbing around the damaged tissue (Vatnikov *et al.*, 2019).

Massage impacts need to be started with the body zone above, and after several sessions it is performed around damage. Receptions are selected taking into account the healing of the skin and performed by the fingertips (Bespalov *et al.*, 2018b).

In the second period of treatment, massage is performed daily or every other day. Massage helps restore skin trophism, muscle strength, and range of motion (Vorobyeva & Medvedev, 2019).

With frostbite, physiotherapy is often prescribed. They enhance blood circulation in damaged tissues, reduce inflammation, and enhance reparative tissue regeneration. Most often, during frostbite, diadynamic currents, ultrasound, electrophoresis, interference currents are used. Paraffin, ozocerite or mud applications, radon and hydrogen sulfide baths are used for resorption of scars.

It existed that the body temperature preservation system does not have its own effector organs and uses the mechanisms of other systems to preserve the temperature homeostasis. Not so long ago, messages appeared, showing that specific receptors were found in animals and humans that are responsible for the sensitivity of tissues to low temperature. It is known that the TRPM8 protein is simultaneously a cold receptor and menthol, and, it is found both in mice and in humans. The cooling of the tissues stimulates the current of calcium ions inside the cells, forming the potential difference in their membrane.

It was also possible to derive mutant lines of mice lacking the ability to produce this protein. All of them turned out to be almost insensitive to low temperatures. Since a certain sensitivity was retained, this, according to the researchers, indicates the existence of an additional mechanism of sensitivity to cold, although a much less powerful mechanism involving TRPM8. At the same time, pain sensitivity in mutant mice increased significantly. TRPM8 belongs to the same family of proteins on the basis of which a heat-detecting receptor is built; it is close in structure to capsaicin (an A-type fast K-channel blocker), which is involved in inflammatory reactions, which is part of pepper and causes it to be "hot" taste.

The participation of biologically active amines in the regulation of cytokine reactions is due to the increase in the content of proinflammatory cytokines (IL-6 and $TNF\alpha$) and the suppression of the level of anti-inflammatory cytokine IL-10, which indicates the stimulating effect of them on the development of inflammatory processes and enhancing reactivity by the immune system. The ability of catecholamines to affect antibody formation by suppressing the synthesis of IgG, IgA against the background of a compensatory increase in the concentration of serum reactors. There are also evidence of the impact on the immune system and other active amines - serotonin and histamine. In particular, the overwhelming effect of histamine and serotonin on the activity of phagocytic protection with the formation of the activity deficit (up to 62.5 and 54.0%) and the intensity of phagocytosis (75.1 and 55.1%) with the braking of the phagocyte function (from 50.68±2.41 to 46.70±2.24% and from 57.18±1.68 to 50.12±2.93%) in practically healthy men born and living in the north. Reported on a study of mRNA expression of serotonin 5-HT1A and 5-HT2A receptors in the frontal cortex, hypothalamus, hippocampus, and midbrain in cold-adapted rats (5 weeks at 4-6 °C) and control (5 weeks at 20-22°C). Long adaptation of the body to cold, without providing a significant impact on the expression of the MRNA 5-NT1A receptor, negatively affects the level of MRNA 5-NT2A receptor. These changes are different in different structures of the brain: in the hypothalamus, there is an increase in the expression of 5-HT2A receptor mRNA, and in the cortex its decrease, in the midbrain and hippocampus, no significant changes were detected.

The identified changes, according to the authors of the communication, are adaptive and, given their localization in the brain, regulatory, they testify in favor of the opinion about the involvement of the serotonergic brain system in the mechanisms of

the control of thermoregulation. A characteristic phenomenon for a stimulated immunodeficiency is the deterioration of quantitative and qualitative indicators of cellular immunity with less than 10-15% T-helpers and T-suppressors and the weakening of their functionality. The negative effect of cold is also shown on another important component of the local immunity system - the macrophage, which manifests itself in the form of inhibition of synthetic nuclear processes. In addition to the direct effect of lipid peroxidation products on the cells of the local immunity system, they also affect the vascular endothelium, which is actively involved in many metabolic processes, causing vasoconstriction and destruction of cell and subcellular membranes, which leads to disruption of capillary trophic and gas exchange processes. The activation of lipid peroxidation peroxidation leads to a cold leads to changes in the capillary channel, in particular, to the restructuring of the metabolic activity of erythrocytes caused by the growth of tissues in oxygen, as well as to the activation of a number of other cellular (granulocytes) and non-tissue systems. Lipid peroxidation increases the concentration of cholesterol and phospholipids in the blood.

Conclusion

In the case of progressive hypothermia in the human body, the body temperature is dropped, the depletion of functional reserves of the heart, liver, muscles, respiratory resistance and heartbeat. This leads to oxygen starvation of fabrics. The influence of cold gradually leads to frostbite. In its pathogenesis, the main role belongs to edema and circulatory impairment leading to the tissue ischemia. Physical rehabilitation after the supercooling of the body or frostbite should be very active. Its success is associated with the complexity and perseverance of the use of therapeutic physical culture, massage, and various physiotherapies.

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