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Review paper

The effect of hypothermia on the human body

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Abstract

Introduction: Hypothermia may appear to be a benign condition, but it is an insidious and life-threatening dysfunction of the thermoregulatory system that occurs in all regions of the world, regardless of season. Hypothermia is caused by cooling when core body temperature decreases below 35°C. The information obtained during a patient interview is also helpful in establishing the causes of hypothermia. The pathophysiology of hypothermia has not been fully elucidated to date. It is rarely diagnosed, in particular in the temperate climate. Hypothermia poses a serious challenge for medical personnel, in particular medical rescue teams who arrive first at the scene.

Aim: **The aim of this study was to present new information concerning hypothermia and the management of this condition.**

Material and methods: This article reviews the available literature and statistical data relating to hypothermia, and it discusses various approaches to managing hypothermia, in particular in pre-hospital care.

Results and discussion: **In homeotherms, significant changes in body temperature** affect the functioning of most organs and disrupt homeostasis. According to the European Resuscitation Council, hypothermia is one of the reversible causes of sudden cardiac arrest, which is why hypothermic patients should be adequately managed. Further research is also needed to expand our knowledge about hypothermia.

Conclusions: Hypothermia is an important cause of death particularly in the elderly, infants, and persons suffering from thermoregulatory disorders or diseases that influence thermogenesis. Therefore, education should be continued in this area and clinical studies should be conducted to deepen our understanding of this problem.

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1. INTRODUCTION

A total of 63 hypothermia-related deaths were reported in Poland between November 1, 2021 and March 31, 2021, marking a decrease of 10 deaths from the corresponding period in 2017–2018. Twelve people have already died from hypothermia in 2022, which can be attributed to low temperatures in the first months of the year in the Polish climate.[1]

The human body can lose heat in several ways. Radiation, namely the loss of heat in the form of infrared waves, can be observed with the use of a thermal imaging camera. Heat lost by radiation is difficult to minimize because the human body always radiates a certain amount of energy. The greater the difference between body temperature and the surrounding temperature, the more intense the thermal radiation process. Heat can also be lost through conduction, namely the transfer of heat through direct contact with the surrounding objects. Objects with a higher temperature transfer thermal energy to objects with a lower temperature through direct contact to equalize the temperature difference. Convection is yet another mechanism of heat loss. In this process, heat is transferred from the body to the surrounding air. Convective heat transfer is intensified by wind and rapid movement of air. Heat can also be lost by evaporation when water is changed from a liquid to a gas during respiration. The energy required to heat inhaled air accounts for several percent of the energy lost by the body. Heat loss by evaporation is determined mainly by the temperature of inhaled gases.

Some thermoregulation disorders also affect normothermia. Thermoregulation can be impaired by dysfunctions of the hypothalamus, the thermoregulatory center of the brain. The hypothalamus is composed of two parts: the 'heat loss' center in the preoptic anterior hypothalamus which is responsible for eliminating heat, and the 'heat maintenance' center in the posterior hypothalamic nucleus which minimizes heat loss and increases heat production in the body.[2]

Stimulation of the hypothalamus triggers mechanisms responsible for heat conservation and production (higher heart rate, cutaneous vasoconstriction, pilomotor reflex, higher metabolic rate). Damage to the lateral and posterior hypothalamus compromises cold-induced cutaneous vasoconstriction responses.

2. AIM

The aim of this study was to present new information concerning hypothermia and the management of this condition.

3. MATERIAL AND METHODS

This article reviews the available literature and statistical data relating to hypothermia, and it discusses various approaches to managing hypothermia, in particular in prehospital care.

Classification of hypothermia

The classification of hypothermia can be problematic. Hypothermia is generally defined as an environmentally-induced decrease in core body temperature below 35°C. Hypothermia is diagnosed by measuring core body temperature during a physical examination. The Swiss staging model for hypothermia is most widely used:

(1) HT1 – typical core temperature of 32°C–35°C, clear consciousness with shivering,
(2) HT2 – typical core temperature of 28°C–32°C, impaired consciousness without shivering,
(3) HT3 – typical core temperature of 24°C–28°C, unconsciousness, vital signs present,
(4) HT4 – typical core temperature of <24°C, no vital signs,
(5) HT5 – death.[3]

The first stage (35–32°C) corresponds to mild hypothermia which does not pose a threat to the patient's life. This stage is characterized by visible shivering, a thermogenic mechanism that is initiated to prevent a decrease in body temperature. The chemical energy stored by adenosine thriphosporate (ATP) is converted to kinetic energy, which activates involuntary muscle contraction. This process is highly energy-intensive, and large amounts of muscle glycogen are depleted, which can lead to hypoglycemia. Stage 1 hypothermia is characterized by a considerable decrease in cerebral metabolic rate and vasoconstriction. Urine production in kidneys increases (polyuria). This stage of hypothermia is easy to diagnose because shivering occurs only within the indicated temperature range.

The second stage (32°C–28°C) denotes moderate hypothermia which is not accompanied by shivering because usable energy has already been depleted. The patient's vital signs change considerably – the respiratory rate and the heart rate decrease to minimize the demand for oxygen. This stage is also characterized by impaired consciousness. The patient is confused, self-awareness is disrupted, and allopsychic disorientation may occur. The risk of supraventricular tachycardia and ventricular arrhythmia increases. Polyuria and pupil dilation are observed. Bradycardia, bradypnea and impaired consciousness are symptoms of moderate hypothermia (HT2) that support reliable diagnosis.

The third stage (28°C–24°C) corresponds to severe hypothermia when bodily functions are significantly altered. Vital signs are further impaired. Severe bradycardia accompanied by arrhythmia, usually ventricular arrythmia, are observed. The EEG pattern is flattened or even flat. All reflexes are absent. Urine production in the kidney decreases considerably. Third-stage hypothermia is generally characterized by loss of consciousness.

The four stage (<24°C) denotes life-threatening hypothermia. Vital signs are not detected during a physical examination. Apnea and cardiac arrest are observed.

According to the literature, prolonged resuscitation with non-invasive rewarming can convert pulseless electrical activity into ventricular fibrillation and lead to the return of spontaneous circulation.[4]

The fifth stage of hypothermia does not occur within a specified temperature range. Irreversible body cooling leads to death. Due to medical progress and the growing body of research into hypothermia, patients with the most severe drop in core body temperature can be effectively resuscitated. The fifth stage of hypothermia is not accurately defined, and it some cases even severely affected individuals can be saved.

Due to the growing availability of cheap electronic devices, temperature is presently measured in the auditory canal or on skin surface with the use of non-contact infrared thermometers. However, these measuring techniques are not reliable due to the influence of external factors, and should not be used by healthcare professionals. Contact thermistors are characterized by higher sensitivity, and they produce more reliable results. The difference between esophageal temperature measured with a contact thermistor and the temperature measured inside the auditory canal or on skin surface with a non-contact thermometer can exceed 10°C, in particular when ambient temperature is low. Rectal and urinary bladder temperature is also lower than core body temperature, and these measurements are not highly reliable.[4] During resuscitation and patient warming, core body temperature should be measured in the same location, and measurements performed in the lower third of the esophagus are most accurate and correlate with heart temperature.

4. RESULTS AND DISCUSSION

4.1. Significant influence of hypothermia on the human body

Numerous adaptive responses are initiated when body temperature decreases below 36.5°C. The stimulation phase lasts until core temperature drops to 34.0°C. This phase involves a stress response which stimulates the sympathetic nervous system and increases the secretion of catecholamines such as adrenaline, dopamine and noradrenaline. Respiratory and cardiac centers in the medulla oblongata are stimulated. The respiratory rate and the heart rate increase to produce more heat. The thermoregulatory center also plays an important role in the first stage of hypothermia. Skeletal muscle activity and tension increase to generate additional thermal energy. When normothermia is not achieved, shivering thermogenesis is initiated. The calorigenic effect observed during stimulation increases the secretion of hormones such as triiodothyronine (T3), thyroxine (T4) and thyroid stimulating hormone (TSH). Thyroid hormones increase the metabolic rate to produce and retain heat. Hypothermia leads to vasoconstriction and centralization of blood circulation. Sweat gland activity subsides. The Lewis reaction is often observed when momentary vasodilation takes place to supply blood to the peripheral vascular system. This mechanism prevents cold damage to the extremities. Vasodilation and peripheral circulation are completely inhibited when body temperature decreases below 24°C.

The reactivity of the medulla oblongata decreases when body temperature drops below 34°C. Cooler blood affects the sinoatrial node, atrioventricular node, bundle of His and Purkinje fibers, and it significantly slows down the heart rate. Arrhythmia, including heart block, is often observed. The activity of the potassium-sodium pump and the calcium pump decreases. Calcium, potassium and sodium ions do not easily cross cell membranes. Coronary blood flow is reduced by up to 40%. The respiratory system is inhibited. Pulmonary dead space increases, which causes blood to stagnate in the lungs. In hypothermia, the oxygen dissociation curve shifts to the left, and hemoglobin's affinity for oxygen increases.

Urine production can increase even three-fold during hypothermia. Tubular reabsorption in the nephrons decreases, and the reabsorption of sodium ions is compromised. Urine production increases despite reduced glomerular filtration. Dehydration increases blood viscosity and arterial blood pressure. When body temperature drops below 32°C, blood pressure decreases due to heart failure. Blood morphotic elements stored in the spleen are transferred to the circulatory system. Prothrombin time is significantly prolonged at temperatures lower than 33°C. Potassium ions are transferred from extracellular to intracellular space. High concentrations of potassium ions in extracellular fluid can be indicative of acute metabolic acidosis and tissue necrosis. Hypothermia induces changes in the acid-base equilibrium. Alkalosis caused by increased respiration in the initial stages of cooling is gradually replaced by metabolic acidosis. Acidosis progresses with a drop in temperature, microcirculatory failure, increased accumulation of carbon dioxide in the blood, and accumulation of lactic acid during shivering thermogenesis. Cerebral circulation decreases by 6%–10% for every 1°C drop in core body temperature. Consciousness and rational thinking are impaired when body temperature decreases to 35°C. A further decrease in temperature ultimately leads to loss of consciousness. Digestive system functions are disrupted, and paralytic ileus can occur at a temperature of 34°C. Increased gastric acid secretion and reduced bicarbonate secretion damage gastric and duodenal mucosa. Liver function is impaired. Hepatic blood flow, bile secretion and detoxification mechanisms are compromised, and liver enzyme levels (ALT and AST) increase. The pancreas ceases to secrete pancreatic juice which participates in digestion.

Patients become comatose when their body temperature drops below 27°C. General symptoms are similar to those observed in clinically dead patients, including pallor, decrease in skin temperature, undetectable peripheral pulse, failure to respond to stimuli, and muscle rigidity. Respiratory and circulatory functions are suspended, and the inotropic effects of adrenaline are reduced. When comatose patients are not resuscitated, all energy reserves become depleted and death occurs upon irreversible loss of cardiopulmonary function.[2]

A stable body temperature is required for physiological processes and hemostasis which controls blood flow in healthy and damaged vessels. Enzymatic clotting reactions are influenced mainly by pH and temperature. Even a small drop in body temperature disrupts platelet functions and clot formation. Bleeding time is prolonged at lower temperature.[2] Platelet hypersensitivity and coagulation in the extremities

are observed until the temperature of 33°C. Platelet activity is significantly reduced at lower temperatures. Thrombocytopenia, i.e. a decrease in platelet counts caused by platelet accumulation in the liver, spleen or migration to peripheral blood vessels, is observed at a temperature of 30°C. At this temperature, platelet aggregation and adhesion are only impaired. Complete failure of the coagulation system occurs below 16°C. The function of the coagulation system is reduced by 10% for every 1°C drop in body temperature.[2]

4.2. The effect of hypothermia on heart function

Cardiac arrhythmia (sinus bradycardia, atrioventricular block, atrial fibrillation, ventricular arrhythmia, prolonged PR interval, QRS complex and QT interval) is often observed during ECG examinations of hypothermic patients. These abnormalities may include the Osborn wave, also known as the camel-hump sign, J wave or late delta wave, which is a hypothermia-related elevation of the J point at the junction of the QRS complex (R wave) and the ST segment (R' wave).[5]

The effects of hypothermia on cardiovascular and respiratory systems were explored by Joseph Osborn in the 1950s.[6,7] His experimental research demonstrated that an abnormal J wave is associated with hypoventilation and respiratory acidosis in hypothermic patients. The J wave disappears when normal body temperature is restored. The mechanism underlying the Osborn wave has not been fully elucidated. Several explanations have been proposed, including differences in the action potential of cardiomyocytes, as well as differences in the transmural endocardial-epicardial gradient, which induce significant changes in early repolarization (phase 1 of cardiac action potential) that are clearly visible during an ECG exam.[8–12] Abnormal ECG patterns associated with acidosis and myocardial ischemia are also observed in the ST-T segment.

Atrial beats and atrial arrhythmia are observed when core body temperature drops below 29°C. Atrial fibrillation with a slow ventricular response is easily diagnosed in around 50% of hypothermic patients.[12–19] A further decrease in body temperature leads to ventricular fibrillation, nodal rhythm and asystole.[20–22]

The trauma triad of death is a medical concept which combines acidosis, coagulopathy and hypothermia (Figure). The higher the number of components in this combination, the poorer the prognosis. It is a vicious circle where one process leads to or intensifies another process. Hypothermia affects the entire body and disrupts all bodily functions. It impairs respiratory, circulatory, nervous, urinary, digestive functions and induces changes in metabolism and coagulation. These functional disorders are exacerbated by the continued decrease in core body temperature. This condition is dangerous to health and potentially life-threatening. Hypothermia is difficult to diagnose due to the absence of specific symptoms and the scarcity of specialist measuring equipment, in particular in medical rescue units. The adverse effects of low temperature on the body's vital functions have to be rapidly minimized in patients diagnosed with hypothermia.[23–26]

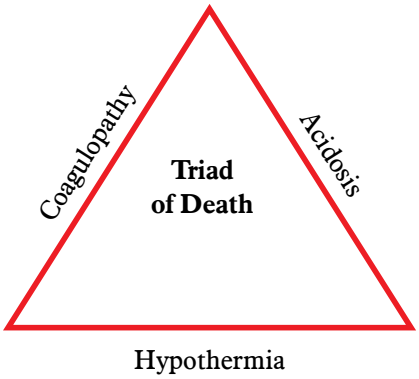

Figure. Trauma triad of death.

5. CONCLUSIONS

(1) Mortality statistics indicate that hypothermia-related deaths continue to pose a considerable problem in Poland.
(2) Medical personnel providing pre-hospital and hospital care should be familiar with the classification of hypothermia and its symptoms.
(3) Hypothermia exerts a particular effect on cardiac activity. Cold-related cardiac symptoms can disappear with a rise in body temperature. Measurements performed in the lower third of the esophagus are most accurate.
(4) Body temperature can vary considerably in different parts of the body, and the results of temperature measurements can be misleading for medical personnel. Measurements performed in the lower third of the esophagus are most accurate.
(5) Hypothermia affects nearly all systems of the human body, and it significantly influences organ function.

Conflict of interest

Authors declare no conflict of interest.

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