

COLD PHYSIOLOGY AND COLD INJURY

Per-Ola Granberg, MD. Ph.D.
Department of Surgery, Karolinska Hospital, Stockholm, Sweden

Introduction

Thermosensitive end organs exist in the **skin** and possibly elsewhere. A cascade of events follows perception of an acute cold stress. In order to minimize heat loss cold stress induces peripheral vasoconstriction via the sympathetic nervous system, which increases the insulating capacity of the shell up to sixfold. In order to compensate for heat loss shivering is an early event, where involuntary muscle contractions increase metabolic rate 2 - 6 fold. Decreasing core temperature induces tachycardia and elevated blood pressure but at lower temperatures bradycardia and hypotonia occur. Death due to ventricular fibrillation or asystole is taking place between 28°-25° C. Cold stress further causes an osmolar diuresis with sodium and chloride as the main constituents. The augmented urine flow decreases blood volume, lowers physical working capacity and increases blood viscosity - all negative events in a hazardous situation. Hypothermia induces depression of mental functions starting with apathy and bizarre behaviour and ending in lethargy and coma around 30°-28° C. The paradoxical feeling of heat with undressing in agony seems to depend on cerebral receptor disturbance.

Cold injuries can affect the whole body and cause hypothermia or be local. The local injuries are subdivided into cold/wet injuries and cold/dry injuries.

Acute hypothermia

Acute hypothermia is basically of two types - Acute immersion hypothermia and more prolonged Exhaustion hypothermia. Field treatment of hypothermia deteriorates into a real challenge, when the victim becomes unconscious. The first aim is to prevent further heat loss. Try to insulate the victim in every possible way. Passive, gentle rewarming is often the only choice and is mandatory in exhaustion hypothermia due to dehydration, hypovolemia and electrolyte disturbances. Cardio-pulmonary resuscitation is many times more situational than medical and, when starts, has to be continuously established even during evacuation if needed.

Non-freezing cold injuries (NFCI)

People are often not aware of the fact, that cold injuries may occur without freezing. In NFCI the exposure time is prolonged, in excess of 12 hours. Wetness and immobilization with venous stasis are contributory factors, as well as dehydration, inadequate food intake and stress. These injuries affect almost exclusively legs and feet. Numbness, paraesthesia and pain disturbing night sleep are often the first signs. The waxy-white discoloured feet are felt heavy and woody and are often anaesthetic to pain, touch and temperature. In a field situation the treatment should be limited to prevent further cooling and to induce gently rewarming - dry socks, hot drinks and additional insulating clothing. All forms of rapid rewarming are contraindicated!

The patient should be transported on a stretcher to the nearest hospital, where conservative treatment is recommended.

Freezing cold injuries (FCI)

In FCI, actual freezing of the tissue takes place. The process is located to the extracellular space. When water is transformed into ice, the osmolality of this space increases creating a passive diffusion of water from the intracellular compartment. The degree of this cell-dehydration will determine the severity of the injury. FCI are best divided into superficial and deep frostbites. The former is limited to the skin and nearest subcutaneous tissue. A stinging, pricking pain in the waxy-white and numb area is often the first symptom. If, however, FCI extends into a deep injury the skin turns white, is felt hard and adherent to the surroundings. Treatment of FCI amounts to prevent a superficial injury to turn into a deep one. Thaw

the frozen area by passive rewarming of heat from a warmer part of the body. The most effective treatment is thawing in warm water at 40°-42° C. It is, however, important not only to treat the local injury but also the whole individual. Pull on more clothes, give warm beverages and force the victim to own muscle activity. Deep FCI are thawed in warm water and the patient is put to bed with the injured areas elevated. After 6-12 hours blisters appear. Don't rupture them. Arrange for adequate transportation to nearest hospital, where further treatment should be conservative. Early amputations must be avoided unless septicemia occurs. Treatment with heparin, low molecular dextran and vasodilators have so far not proven useful in controlled studies. Early sympathectomies are contraindicated.

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