

## Survival from Severe Accidental Hypothermia in an Elderly Woman: A Case Report

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### Abstract

**Introduction:** Accidental hypothermia is an environmental condition with basic principles of classification and resuscitation that is associated with significant morbidity and mortality despite aggressive treatment. Hypothermia is defined as a decrease in core body temperature (CBT) below 35°C and may be staged in mild (35°C-32°C), moderate (32°C-28°C) and deep (<24°C). The latter group is associated with significant risk of circulatory arrest. The considerations regarding acute illness and complicating hypothermia presents some challenges concerning stabilization and treatment.

**Case presentation:** An 88 year-old woman with a previous medical history of dementia and first -degree atrioventricular block was found outside her home. As a result of exposure, the patient developed severe hypothermia with a core temperature of 19°C, hypoventilation, Glasgow Coma Scale 4, and sinus bradycardia. Critical care was initiated right away and included intubation, internal and external re-warming, and various drugs during orotracheal intubation. The next day she was extubated and discharged from the intensive care unit without any complications, and within four days she was discharged from the hospital.

**Conclusions:** We describe some considerations regarding treatment and complications of severe hypothermia in an elderly patient, and a review of the physiological changes with exposure to hypothermia.

**Keywords:** Hypothermia; Pharmacokinetics; Core temperature; Resuscitation; ICU care

### Case Presentation

On a cold winter morning with an outside temperature of 1°C, an 88 year-old woman was found lying in her garden in front of her house. The patient was of normal stature, dependent on daily care for medication, but lived alone in a house with a medical history of severe dementia, first-degree atrioventricular block, and stroke.

### Pre-hospital care

The ambulance call center dispatched paramedics and an emergency doctor to the site within 10-15 minutes. The patient was unresponsive, with a GCS (Glasgow Coma Score) of 4, only providing unintelligible sounds. She was breathing slowly and had a heart rate of 25-30 beats/minute with a sinus rhythm. The patient was moved carefully transferred to the ambulance, and an initial temperature reading showed 19°C. A large caliber peripheral intravenous line was provided for rewarming with warmed fluid at 37°C, warm blanket, and full body insulation with aluminum foil. An uncomplicated, rapid sequence induction and subsequent intubation was performed using alfentanil 1 mg, propofol 50 mg and suxamethonium 50 mg iv at the site. She was admitted to the emergency room in-hospital one hour after being found, without any complications during transport to the hospital.

The patient was subsequently admitted to the intensive care unit (ICU). Core temperature in the bladder was not immediately measurable in emergency room, but two hours later in the ICU care it was registered to 21.1°C, despite ongoing treatment with external warming using a Bairhugger® blanket set at 42°C, and 2 liter of 37°C crystalloid. In the ICU the patient was normoventilated using the PRVC mode, FiO<sub>2</sub> 0.5 and PEEP 5 cm H<sub>2</sub>O. At this point, she was severely hypotensive (non-invasive blood pressure, 60/20 mmHg) and bradycardic (30 beats/minute). An ECG showed no sign of ischemia. A feeding tube was placed to avoid aspiration.

The first arterial blood gas analysis showed pH 7.29, base excess -10.3 mmol/L, and plasma glucose 9.7 mmol/L. Further laboratory data revealed hemoglobin 7.7 mmol/L and lactate 3.5 mmol/L, INR 1.1 and thrombocyte count 235 mmol/l, and potassium 4.3 mmol/L. Internal and external rewarming was continued according to Danish Guidelines by warm crystalloid infusion (37°C) and warmed forced air and a Bairhugger®. No epinephrine was used on the premise that drug responses could be unpredictable due to hypothermia until core temperature had reached 30°C.

Physical manipulations were kept at a minimum due to the risk of cardiac arrhythmias. During rewarming, the patient was circulatory stable and gradually reached 35°C the next day. In all, approximately 3.5 liter of warmed intravenous fluid was administered during her stay at the ICU. Urine production began as the core temperature reached 30°C, and although the patient exhibited atrial fibrillation at 34°C, it self-converted to normal sinus rhythm without any medication. A

small amount of propofol for sedation and tube acceptance was administered during the first night. Treatment with antibiotics was started in the case of sepsis.

### In hospital care

The following day, the patient was extubated; she was fully awake, mentally inconspicuous and cardiopulmonary stable. She was discharged the next day, to a medical ward, and two days later she was discharged to her home. She now required more daily care because of her severe dementia while waiting for a nursing home [1-4].

### Discussion

It was decided to not contact Danish Specialist Center for Resuscitation in Hypothermia for concerning treatment of severe hypothermia with ECMO, on the basis of advanced age and comorbidity and a DNR (do-not-resuscitate) decision. According to previous records, the patient did not want life prolonging treatment or resuscitation in the case of cardiac arrest. This explains why our in-hospital treatment differs from recommended guidelines. A decreased physiologic reserve, social isolation, chronic diseases and medications that impair compensatory responses are factors that increase the risk of developing hypothermia and accompanying complications in elderly people [5].

Hypothermia is related to a variety of physiological changes affecting most organ systems [5-7]. Age involves changes that increase the risk at critical point in development of hypothermia impaired thermoregulation, a lower rate of metabolism, a less effective vascular response, a reduction in body mass composition and shivering in response to cold, inadequate diet, a decreased resting peripheral blood flow, and nonconstrictor vasomotor response to cold [5,7-8] In the bird's eye view, some general temperature dependent effects may be observed as: consciousness, cerebral and total body metabolic rate, oxygen consumption, CO<sub>2</sub> production, cardiac output, renal blood flow and respiratory function are all progressively reduced by depth of hypothermia [7].

Core body temperature (CBT) is normally tightly regulated by an effective thermoregulatory system, and even during different stages of illness the regulatory system maintains a stable core body temperature [6].

Normal body temperature is controlled by effective thermoregulatory defenses [6]. Thermal information from skin, peripheral tissues and core organs are integrated and processed prior to arriving at the hypothalamus, which is the dominating thermal control center [6]. A balance between heat production (thermogenesis) and heat elimination (thermolysis) is maintained. This active equilibrium keeps the normal CBT on an average 37°C [6]. The initial response of the body to cold exposure is to maintain a normal CBT by means of active movements, peripheral vasoconstriction and involuntary shivering. The risk of clinically significant arrhythmias including cardiac arrest increases as the CBT drops below 32°C, and increases substantially if the temperature becomes less than 28°C [1,3,4]. Cardiac arrest often occurs at the core temperature < 24°C. [4]

### Cardiorespiratory system

In deep hypothermia, a relatively high blood flow is distributed to the brain. Hypothermia registered in the hypothalamus and the fourth ventricle is partly responsible for shivering and cognitive and

respiratory dysfunction [6]. Hypothermia affects cerebral functioning, with initial impairment of cerebral cortex, followed by subcortical structures. Finally, respiratory arrest occurs when medullary cellular activity is suppressed; at 20°C, a flat EEG and no respiratory response by CO<sub>2</sub> increase or hypoxia may be observed [4]. The coldest core temperature a person has been revived from is 13.7°C [9]. The oxyhaemoglobin dissociation curve initially shifts to the left, causing impaired oxygen delivery and tissue hypoxia, which in turn causes anaerobic metabolism, lactate acidosis and an eventually right shift of the dissociation curve [7,8]. Fluid shifts predispose to noncardiogenic pulmonary oedema, and the functions of respiratory muscles are gradually impaired [6]. Heart rate becomes bradycardic and unresponsive to atropine, a variety of ECG changes occurs due to temperature sensitive purkinje fibers, and catecholamine release is gradually blunted [8]. Finally, an increased risk of arrhythmias become resistant to cardio version attempts, reduced chest wall elasticity makes chest compressions difficult and most antiarrhythmic agents are largely rendered ineffective. As the pacemaker cells are extremely sensitive at this point, non-life threatening arrhythmias related to hypothermia seen below 34°C should not be treated with defibrillation or drugs. Changes in ADH secretion, tubular reabsorption and renal blood flow cause cold-induced diuresis with the risk of hypovolemia. Hypokalemia may occur due to the influx of potassium into cells. However, it is generally accepted that natriuresis and pH changes should not be actively corrected, since they may lead to ventricular fibrillation as well [8].

### Haematological system

Several complex events occur in the coagulation system during hypothermia, most of which lead to increased bleeding tendency. The activity of coagulation factors for the clotting cascade is progressively reduced with decreasing temperature, bone marrow function is suppressed, vascular permeability is increased, and thrombocytopenia occurs due to sequestration in the liver and the spleen [8]. Fibrinogen levels and fibrinolysis increase, with impaired endothelial synthesis of prostacyclin promoting platelet aggregation. In spite of this hypocoagulable state, microinfarcts and even DIC syndrome may appear [8]. Immune function is inhibited by leukocyte depletion and impaired neutrophil migration and phagocytosis ability. Local vasoconstriction decreases oxygen partial pressure in tissues, macrophages become less responsive to interferone, and increased corticosteroid levels enhance immunosuppression [7]. Insulin levels may vary and glucagon levels increase, but blood glucose depends primarily on the metabolic state rather than the degree of hypothermia. Mitochondrial dysfunction may lead to apoptosis [7,8].

### Clinical pharmacology

The effects of hypothermia on drug disposition are not fully understood due to the complex interaction between pharmacokinetics including metabolism and drug response [8]. Drug metabolism during hypothermia may be more complex than simply increasing the concentrations of active metabolites. The speed of enzyme-mediated reactions is temperature-dependent, and the speed of various reactions is significantly influenced by hypothermia [10]. Decreased splanchnic blood flow and failure to utilize glucose suppresses hepatic detoxification and conjugation processes, prolonging the elimination half-lives of many drugs [10]. Having said this little is actually known about pharmacokinetics and dynamics during hypothermia; blood/gas solubility of gas anesthetics is increased, causing longer emergence

time. When CBT is below 30°C, adrenalin and other vasoactive drugs should be administered with great caution [10,11]. Reduction in vasoactive drug metabolism may cause potentially toxic concentrations [10]. Epinephrine should be actively withheld. The body responds poorly to vasoactive drugs at low temperatures, and the reduction in metabolism may give rise to potentially toxic concentrations when administered on a repeated basis.

A rebound effect during rewarming is an obvious complication, when adding up doses due to insufficient effects [8]. The risk of unanticipated toxicity or subtherapeutic dosing is highly increased. Hypothermia decreases the potency and efficacy of certain drugs [10]. Hypothermia may decrease insulin sensitivity and the amount of insulin secreted by the pancreas, leading to hyperglycaemia. Doses of insulin required to maintain normoglycaemia are likely to decrease when the patient is rewarmed, meaning that hypoglycaemia may easily develop in the rewarming phase [7,10].

## Conclusion

Our knowledge about the treatment of severe accidental hypothermia in elderly people is limited, since it is based on studies of rewarming of younger patients. Advances in safety and availability of rewarming techniques have improved the prognosis for patients suffering from hypothermia [12], but the prognosis for the elderly is unknown.

In retrospect, we reflect on the various procedures done to support vital organ functions, and some of the concurred conditions. In this particular case report, the following benefits and disadvantages may be addressed: The advantages of sufficient oxygenation and prevention of aspiration outweigh the risk of triggering ventricular fibrillation by performing a tracheal intubation [12]. Warm fluids consisting of crystalloids as part of an internal rewarming regimen was rapidly infused combined with external rewarming.

In conclusion, the actions during treatment of a severe hypothermic patient should always cause the practitioner to reflect on the physiological and pharmacological changes that depart from normal resuscitative practice, and to institute a less aggressive treatment approach. This is especially important for the insertion of central lines due to the risk of triggering cardiac arrhythmias, and the administration of virtually all kinds of drugs due to the changes in drug metabolism and effect. We did not encounter a plethora of physiological complications in this case story. In this case we want to highlight that a simple approach may be a successful strategy even in fragile, elderly patient with severe accidental hypothermia. Generally a careful, active external rewarming and a low stress strategy for treatment of hypothermia in elderly patients is recommended.

## Authors' Contributions

MV conceived of the case, did the clinical research and drafted the manuscript. KJ did the pharmacological research and assisted in writing the manuscript. VLL did research and assisted in drafting the manuscript.

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## Conflicts of Interest

The authors have no conflicts of interest to disclose.

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