Journal of Prehospital Medicine

Volume 2, Number 1
July - September, 1988

Hypothermia: Implications for Prehospital and Emergency Department Management

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Hypothermia is best defined as a body core temperature below 95 F (35 C)(1-9). It is a very common medical emergency that may occur in any part of the country, at any time of the year. Hypothermia is a leading cause of death in rescuers and adventurers, therefore, survival is dependent upon knowledge, attitude, and training (2). Research has shown that approximately 75% of all incidents occur outdoors, at temperatures between 30 - 50 F (3), and that mortality rates range from 11%-48% (3,10). It is discouraging to note that approximately two thirds of the victims of hypothermia suffer from some degree of alcohol intoxication (3).

PHYSIOLOGY OF TEMPERATURE REGULATION

Temperature homeostasis is the responsibility of the hypothalamus. The hypothalamus pays little attention to the heat-generating process, but instead regulates the rate at which heat is lost to the environment through the skin. When the hypothalamus senses that the blood is cooler than normal, it stimulates massive vasoconstriction to shunt blood from the periphery to major organs such as the brain, heart, and lungs (2,11). Cardiac output also decreases, facilitating heat conservation. Normal heat regulating mechanisms serve to conserve heat rather than generate it, and for every 10 F decrease in temperature, metabolism is lowered by 50% (5,8,11).

There are five methods of heat transfer: conduction, convection, radiation, evaporation, and respiration (2,4,12).

Conduction is reliant upon direct contact between surfaces. Heat is transferred from one object to another and always moves from a warmer to a colder object.

Convection is the transfer of heat by movement or motion. Heat is transferred directly into the air surrounding the object.

Radiation is dependent upon a temperature gradient between the body and the surroundings. The warmer the object, the more heat is radiated away.

Evaporation is the cooling effect resulting from conversion of surface moisture to a vapor.

Respiration is a form of heat lost through evaporation. As inhalation occurs, air is warmed and evaporates upon exhalation.

The rate of heat production is governed by the difference between the body's core temperature and the ambient air temperature. This rate may be increased by external heat sources, exercise and shivering, and ingestion of food and the digestive process (4). Shivering is the body's major mechanism of heat production, but is dependent upon an intact central nervous system and neuromuscular function. This explains why patients with spinal cord injuries don't shiver below the level of injury (12).

Other factors related to alterations in temperature regulation include the patient's age. Small children lose heat, often three times as fast as adults (2,5,8,11,13,14). Neonates have an even greater problem because the surface area of their head is greatly disproportionate to body mass (2).

The elderly often have a decreased awareness to cold, and may even develop failure of the temperature regulating system (2). In addition, they are often dependent upon medications that alter the body's temperature response (2,5,8,9,12,13,14). Medications such as barbiturates, phenothiazines, alcohol, morphine, diazepam, vasodilators, and even general anesthetics alter the body's response to temperature (8,12).

Diabetics and individuals who suffer from hypoglycemia are prone to hypothermia as insulin production and release can be manipulated by subtle temperature changes (5,13).

Many feel that the amount and distribution of adipose tissue is a major factor in temperature regulation (15). The thicker the fat layer, the greater the resistance will be to cold. Females generally have more adipose tissue but may also have a greater surface area to weight ratio. Evidence indicates that those individuals with a greater surface area to weight ratio will cool quicker (11,15). Individuals of this type typically are tall, thin, and have long arms and legs (11,15). Males usually have greater muscle mass which acts as an insulator, and will produce heat when shivering occurs.

PHYSIOLOGIC RESPONSE TO HYPOTHERMIA

The determining factor for development of hypothermia seems to be the ability of the body to compensate rather than the actual environmental temperature (2). Some consider the hypothermic patient to be similar to a victim of multiple trauma, with all organ systems involved (17,18). The following describes the effects of hypothermia on the various body systems at the different levels of hypothermia.

As the body core temperature falls from normal to 95 F (35 C), cardiovascular responses include peripheral vasoconstriction induced by stimulation of the hypothalamus. As blood is shunted from the periphery to the core, cardiac output is

improved initially (2,16,17). Vasoconstriction is quickly followed by shivering as the central nervous system (CNS) responds (2,3,4,16,17,19,20). Other CNS impairments include poor judgement and a decreased ability to perform tasks that require fine motor coordination (3,4,19,20).

When the core temperature is in the range of 90 - 95 F (32 - 35 C), cardiopulmonary effects at this level of hypothermia include increased peripheral vasoconstriction causing tachycardia and hyperpnea with an increase in oxygen consumption (17). CNS reaction to this temperature range involves increased attempts at thermogenesis by violent, uncontrolled shivering. Thinking becomes impaired and individuals may become amnesic (11, 19).

At 86 - 90 F (30 - 32 C), hypothermia becomes a true medical emergency. Shivering ceases and the muscles become rigid (3,13,16,19). Movements become erratic and jerky. Pupils become dilated and the victim assumes a glassy stare (2,13). Skin is cold to touch due to profound vasoconstriction. Cardiopulmonary effects become more pronounced. Bradycardia develops (3,17) and a J-wave or Osborn wave may be recognized on the electrocardiograph (2,3,8)(Fig. 1). Cardiac output is greatly reduced and hypotension is common (3,16,17). Respiratory alkalosis may present secondary to hyperpnea and resulting decrease in carbon dioxide level. Oxygen consumption may be increased a much as four hundred times normal (16). The victim may become unconscious due to decreased cerebral perfusion.

In the range of 82 - 86 F (28 - 30 C), the effects of hypothermia are profound. With increasing myocardial irritability, ventricular fibrillation may result (2,6,13,16,18). The victim will appear cyanotic (4,13,19) and heart and respiratory rates will be decreased. CNS effects include decreased level of consciousness, and pupils become nonreactive (3,19).

Severe hypothermia occurs with core temperatures of 78 - 82 F (26 - 28 C). Victims will be comatose by this stage, (3,16,19,20) and are flexive (19). Heart sounds will be inaudible and rescuers will be unable to palpate a pulse or blood pressure (3). Erratic arrhythmias such as atrial fibrillation, atrial flutter, extrasystoles, ventricular fibrillation and asystole present (2,19).

Below 78 F (26 C), failure of the cardiac and respiratory center of the brain is common. Spontaneous ventricular fibrillation and asystole may occur, and individuals have the signs of clinical death (19). Pulmonary edema is another common finding as the cardiopulmonary systems fail (12). At temperatures below 62 F (17 C), the electroencephalogram will display an isoelectric line (21).

INTERVENTIONS

The main goals of prehospital intervention include prevention of further heat loss, transport to a facility familiar with hypothermia resuscitation, and prevention of cardiopulmonary arrest (18). It is imperative to determine the core temperature prior to treatment measures, as some interventions may not be provided in certain situations.

Standard thermometers register only to 94 F (34.4 C). Hypothermia thermometers which register much lower readings should be used in any patient with a rectal temperature below 95 F (35 C). These are available from many medical supply sources (18).

Prevent further heat loss by removing wet clothing and placing the patient in blankets, a sleeping bag, or anything that will insulate against additional heat loss. In some situations, it may be necessary to have a warm rescuer huddle with a hypothermic patient to promote the transfer of heat (6,13). Complete rewarming in the field should not be attempted, and should be initiated only when the patient is under complete physiologic control (16).

When performing an initial assessment of a hypothermic patient, it is necessary to take at least one full minute to assess for the presence of vital functioning (6,9,17,18,23). There is much debate over whether or cardiopulmonary resuscitation (CPR) should be initiated in the field for the profoundly hypothermic patient, especially if cardiac monitoring is unavailable (22). The concern expressed is that chest compressions may precipitate ventricular fibrillation in this type of patient (18,22).

While Mills (24) argues that some patients have a rhythm that can't be detected, therefore CPR is contraindicated, Zell (7) and Steinman (6,18) assert that CPR should not be withheld because the brain will be better perfused with chest compressions, and withholding CPR may cause cerebral anoxia and result in brain ischemia. The important idea is that at least one minute be used to adequately assess the patient for the absence

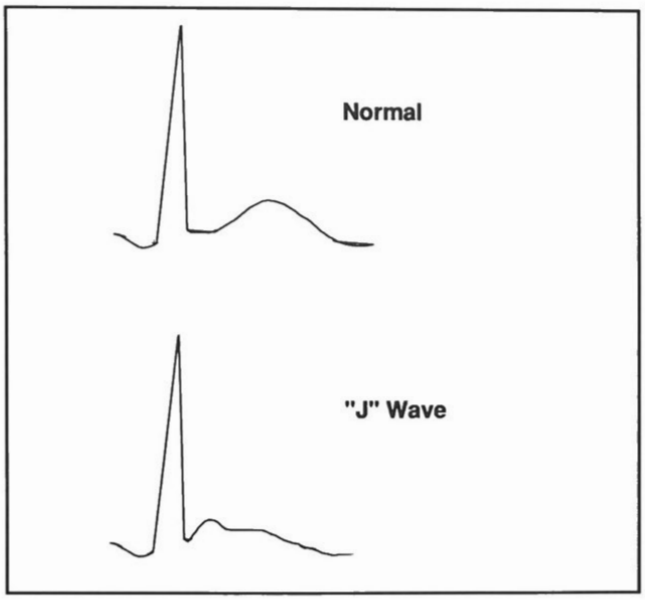

Figure 1 - J-Wave (Osborn Wave) - The J-wave may appear as an upward deflection after the QRS complex.

of respirations and pulse prior to initiating resuscitative measures in the field.

Advanced prehospital care differs relative to the stability of the patient. In the unstable patient, basic life support measures may be all that is necessary. If the patient has an adequate airway, intubation should be avoided, as the procedure may increase the risk of inducing ventricular fibrillation in the unstable patient (22). Instead, provide mouth-to-mouth or mouth-to-mask ventilation to present warmed, humidified oxygen (18).

Defibrillation is dangerous with core temperatures below 86 F (30 C). The hypothermic myocardium doesn't respond well to countershocks and may be damaged in the process (6,18,22).

Pharmacologic therapy is also contraindicated in the profoundly hypothermic patient. Body systems at decreased temperatures function differently than at normal temperatures, and so it may be difficult to predict the distribution of drugs throughout the patient (3). Medications will not be properly metabolized and cleared by the liver and kidneys, and toxic levels may accumulate and become activated during the rewarming process (6,9,18,23). Research indicates that dysrhythmias may be unresponsive to atropine sulfate, procainamide, countershocks, and even pacing (3).

Medical antishock trousers (MAST) serve to complicate rather than improve the situation, and should be avoided unless the patient is known to be hypotensive secondary to fluid depletion or blood loss (18). The MAST, when deflated, may create a sudden bolus of cold, acidotic venous blood into the core circulation from the lower extremities. Sudden temperature changes have been shown to produce ventricular fibrillation in the hypothermic myocardium, and should be avoided (6,18).

The hypothermic patient should be handled extremely gently, especially if the core temperature is below 90 F (32 C), as ventricular fibrillation and other life threatening dysrhythmias may develop secondary to myocardial irritability (2,4,6,13,16,18). Maintain the patient in a horizontal position to decrease the risk of orthostatic hypotension and to promote better cerebral perfusion (18).

Warm intravenous (IV) fluids may be administered to hypothermic patients. Preferable fluids include 5% dextrose and normal saline. Lactated ringer's is less preferable because the hypothermic liver may be unable to metabolize the lactate (16,18). Cautious administration is necessary since rewarming presents with many complications. The rescuer may administer a fluid challenge of 300 - 500 ml and follow with a continuous infusion at 75 - 100 ml per hour (6,18).

Intravenous fluids may be warmed by placing the container inside the rescuer's clothing in contact with the body. The IV tubing may also be wrapped around the rescuer's arm or a hot pack. If a microwave oven is available, fluids may be rapidly warmed by this method (22). The average time required to warm one liter of solution is two minutes at high settings, though time may vary from oven to oven (22). It is estimated that by administering IV fluids through a central intravenous line, core temperatures may be increased by approximately 4 - 8 C per hour (22).

Warmed, humidified oxygen can be provided to the patient as described earlier by the rescuer providing mouth-to-mouth or mouth-to-mask ventilation. This is the fastest method, since no special equipment is required. In addition, using warm water in a humidifier, wrapping oxygen tubing around hot packs or the rescuer's arm can provide the same result (3,4,6,7,13,18).

Hot packs to the head, neck, groin, and trunk can provide additional warmth to the patient, but should be well insulated to prevent direct contact with the skin. Third degree burns have been reported secondary to direct application of heating devices because the skin is so sensitive to the heat and is easily thermally burned (18).

Emergency department (ED) treatment should utilize the same principles as in the prehospital setting. In the ED setting, prolonged, successful resuscitations have been reported, with some efforts exceeding two hours duration(5). It is thought that patients should be warmed to at least 90 F (32 C) before resuscitative efforts are discontinued (25).

Additional non-invasive heating methods that may be employed in the emergency department include the use of heating pads or electric blankets (2,6,13,16,25,26), and immersion in warm water (5,25,27). Aggressive invasive measures may be employed in the ED setting, and include gastric lavage (3,5,13) and peritoneal lavage(2,3,5,7,13,25,26) using warmed fluids. Cardiopulmonary bypass, mediastinal lavage, and hemodialysis are effective invasive measures, but are not commonly performed in the emergency department (22,28).

In the emergency department setting it is appropriate to rewarm the patient approximately 1 - 2 C per hour, but temperature must be monitored carefully and closely, preferably by rectal probe (7,8). Continuous electrocardiograph monitoring should be employed as well (8).

Frequent laboratory studies are indicated and should include complete blood counts, electrolytes, glucose, coagulation studies, arterial blood gases, BUN, and creatinine (5,8,18).

A foley catheter should be inserted to monitor urine output. Initially, there is a diuresis secondary to peripheral vasoconstriction. As renal blood flow is slowed due to increased viscosity, the glomerular filtration rate decreases and one should expect the urine output to be reduced as well.

Complications associated with rewarming can effect the patient in the prehospital or emergency department environment. Rewarming shock is caused by the application of external rewarming techniques and results in vasodilation with venous peripheral pooling (2,5). Afterdrop also occurs during the rewarming process. As vasoconstricted vessels are dilated, the cool, stagnant blood returns to the core circulation. This decreases the core temperature to produce afterdrop. Toxins built up in the periphery may produce cardiac standstill as the stagnant blood returns to the core circulation (4).

SPECIAL SITUATIONS

Cold Water Drowning

Cold water drownings may present with the clinical signs of death and dilated pupils with a body cold and rigid (29). Research indicates that victims of prolonged hypothermic submersions may be saved despite the absence of life signs (30). The prolonged survival may be facilitated by the mammalian diving reflex which is activated when the face is immersed in cold water. This sudden shock produces bradycardia and vasoconstriction, shunting blood from the extremities to increase perfusion to the brain, heart, and kidneys. The reflex also serves to conserve oxygen (29,30).

In cold water drownings, resuscitative efforts should be continued even if the victim was submerged longer than the usual four to six minutes, because of the protective nature of the mechanism. In drownings of this type, cardiac arrest is usually not a primary occurrence. Arrest usually occurs after oxygen is decreased to a critical level. The American Heart Association's guidelines for CPR state, "In cold water less than 70 F - always start CPR, even after 30 minutes."

Motor Vehicle Trauma

Hypovolemia initiates and promotes general cooling of the body (31). Pozos and Born (32) claim that, "Any accident in which there is a blow to the head, spinal cord or neck will likely involve some impairment of thermoregulatory capability." Thus, patients suffering from any of the above injuries should be treated prophylactically for hypothermia. When utilizing a spine board for these patients, place a blanket between the patient and the board as well as over the patient to conserve body heat.

Alcohol is a factor in approximately one third of all motor vehicle accidents. Alcohol is a vasodilator, so, mixed with blood loss and potential injuries, the problems may be compounded (3).

CONCLUSION

It is important to remember that human beings are not dead until they're warm and dead (9). Many individuals have been mistaken for dead because they have presented with clinical signs of death. Patient's should be given the benefit of the doubt. Assess the patient for signs of life for at least one minute, and longer in special situations.

Hypothermia can occur in any area, at all times of the year. And anyone can suffer from hypothermia - including the ill and injured. Accidental hypothermia is a life threatening medical emergency, and perhaps should be considered in many patients exhibiting clinical signs of death, or whose mechanism of injury or illness may be consistent with that of hypothermia.

REFEREE COMMENTARY

Daniel F. Danzl, MD (Department of Emergency Medicine, University of Louisville) - Field treatment of accidental hypothermia has been appropriately termed the "art of the possible". This review nicely synopsizes the pathophysiology which must be understood to direct pre-hospital resuscitation.

There are several pre-hospital interventions mentioned in this article deserving comment.

Continued controversy regarding the potential hazards of endotracheal intubation reflects (in my opinion) coincidental episodes and the miscitation by Fell[1] of a series of hypothermic overdoses reported by Lee & Ames.[2] Fell stated that "endotracheal intubation was followed by cardiac arrest in a large proportion of cases", while Lee & Ames merely cautioned of that possibility. In a recent multicenter survey of 428 cases, endotracheal intubation was performed on 117 patients by multiple operators in various settings.[3] Patients were pre-oxygenated and no induced arrhythmias were recognized. We nasotracheally intubated 40 pre-oxygenated hypothermic patients without incident in an earlier series.[4]

Ledingham noted no intubation arrhythmias in his prospective series of 44 cases.[5] One of the Mt. Hood patients did develop ventricular fibrillation (VF) in the OR while being intubated and placed on cardiopulmonary bypass. More common factors causing VF include mechanical jostling, and sudden acid-base or electrolyte fluctuations. Therefore, the pre-hospital indications for endotracheal intubation in hypothermia should be similar to those in normothermia. Blind nasotracheal intubation after pre-oxygenation is preferable to cricothyroidotomy when cold-induced trismus or potential cervical spine trauma is present.

Regarding pharmacologic therapy, the roles of prevention and the ideal treatment of ventricular arrhythmias in the field in hypothermia are not resolved. Bretylium tosylate has been extremely effective in several animal studies. Although human data are sparse, bretylium is the only agent possessing anti-arrhythmic activity during hypothermic conditions. Two cases of chemical defibrillation after infusion of 10 mg/Kg bretylium in accidental hypothermia have been reported.[6,7] Bretylium appears to be the agent of choice for VF in hypothermia and I would not withhold it. However, bretylium prophylaxis is still investigational, since toxicity, optimal dosage and particularly the ideal rate of infusion are unclear.

Regarding selection of field rewarming modalities, emphasis on the history obtained at the scene is critical. A chronic subclinical course of hypothermia in an elderly indoor victim presents different rewarming challenges than an acute exposure. As the author notes, active external rewarming must be limited to the trunk. In the chronically hypothermic victim, external heat application to the extremities extinguishes peripheral vasoconstriction. This results in the sudden central return of cold, hyperkalemic, acidotic blood.

While mouth-to-mask ventilation provides some heated humidified oxygen, there are more efficient lightweight portable first aid devices that deliver heated humidified air at all operating temperatures in the field (eg. Hayward & Douwens' Uvic® Heat Treat® Systems (Thermo-Genesis Int.; and the Hypothermia Oxygen Warmer from Bow/Parm Inc). When equipment is available, active core rewarming via heated inhalation is the treatment of choice in the field. I would avoid insertion of central intravenous catheters via the subclavian route. If inserted too far, the catheter tip can irritate the cold myocardium. Also, be sure to thoroughly shake microwaved solutions to avoid

'hot spots'. Lastly, rewarm patients to 95 F (35 C) rather than 90 F unless there are contraindications to CPR in the first place. Obviously lethal injuries, "do not resuscitate" status, or rescuer endangerment by evacuation delays are examples. These have been some dramatic recoveries late during rewarming.[8]

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David S. Smith, Ph.D (Smith Aquatic Safety Services, St. Charles, MO) - One of hypothermia's more tragic aspects is that everyone knows where to look for it. Hypothermia undoubtedly occurs in its purest, primary form in out-of-doors locations to backpackers, cross-country skiers (water skiers aren't immune either), immersed boaters and hunters, etc.

If the truth be known, however, the number of primary hypothermia cases are relatively few, especially compared to hypothermia's continual presence and potential as a telling, secondary malady following trauma, in comparatively warm environments.

Hypothermia appears when the ability to generate or conserve heat through internal or external sources is reduced or lost. The cause for such compromise may be intrinsic in that the individual cannot produce heat metabolically, through loss or lack of shivering, as when intoxicated, or through an inability to chemically convert body fats. Other intrinsic causes may reside in hypothyroidism in the elderly where the patient cannot thermoregulate, or again in intoxication where an impaired person's blood vessels, primarily in the dermis, do not dilate or constrict in response to heat or to cold stress.

If the cause is exogenic, the victim cannot conserve or generate heat by moving closer to a heat source such as a fire; adding layers of clothing or other insulation; eating; or exercising.

The typical out-of-doors hypothermic, if not intoxicated, succumbs in stages as depicted in the preceding article. However, in the traumatized, hypovolemic, and intoxicated accident victim, where all the above listed bars to heat generation or retention are present, the drop in body temperature is precipitous, frequently by-passing stages such as shivering. Moreover, where lack of brachial blood pressure may appear at a body temperature of 88 - 89 F in a primary hypothermic, the hypovolemic - intoxicated accident victim may present no pupillary response, rigidity of appendages, and drastically slowed heart and respiratory rates at body temperatures just a few degrees below normal. This can be expected when severe blows to the head are indicated.

One of the all-to-frequent results of such massive reductions of vital signs, especially in triage, or multi-casualty situations, is that viable, but severly metabolically depressed victims are prematurely given up for dead.

If such situations exists, with a high frequency, why hasn't more attention been given them in the literature? The answer is simple. No one has been looking for them. Lack of clinical suspicion Is the handmaiden and co-hangman of hypothermia.

If field and clinical assessments were to routinely follow an ABCD protocol, where, after essential ABC needs were addressed, providers then attempted to accurately define and to deal with bodily temperature levels, a total revision and revelation of thinking, re: the presence and effect of hypothermia might ensue. For instance, giving every trauma victim warm IV's and warm oxygen would grow. ER's might reconsider their now prevalent ice box temperature levels. Practitioners would be more aware of the thermal needs of children, as well as burn victims who have damaged or lost their main thermoregulatory organ - their skin.

Similarly, more attention should be paid to near drowning mechanisms. Even though extensive pockets of non-believers still exist both among medical and emergency responders, more providers are becoming aware of the underwater potential of dive reflex and hypothermia. Even among the initiated, however, a number of problems remain. For instance, ice water is not necessarily a prerequisite to long term underwater survival. The U.S. record is 66 minutes completely submerged without resulting brain damage. This record was set by a then three year old girl, on June 10th, 1986 in Salt Lake City. Please note the date. The more we understand the myriad factors influencing changes in body temperature levels, the more pronounced the "not dead until (re-warmed and dead" dictum.

For instance, a 70 F water temperature may be obtained on the surface. But what is the temperature and depth at which the victim is found? The relative motion of water past the body can effect heat loss more than simple temperature levels. If the body is small in comparison with a relatively larger head, as in a child's case, flow rate is extremely important. Increased pressure with depth also plays a role in triggering dive reflex.

Similarly, the foregoing article indicates that death occurs at bodily temperature levels around 78 F. This was once thought true. However, a large number of recent cases involving younger children, where the patient has been rewarmed and revived, without sequelae, whose temperatures were below 60 F, have cast suspicion on thinking that respiratory and cardiac arrest occur at higher temperatures. Due to cold skin's inability to transmit sound and electrical impulses, plus faint heart and shallow respiration rates of only one or two a minute, the patient may again appear to be dead, when they are, in fact, alive.

Author's Reply - One point in particular of Dr. Danzl's deserves re-emphasis. I agree with his remarks about about airway priority. The indications are the same for hypothermic and normothermic patients. When it is clearly needed, the benefits of airway control outweigh the risks of potentially inducing ventricular fibrillation. His comment about rewarming to 95 rather than 90 F before deciding the victim should not undergo addtional resuscitation is not inconsistent with my views. My mention of 90 F was based on what I found in the literature and not in personal experience. In the interest of giving patients the benefit of the doubt, rewarming to 95 F before terminating CPR is reasonable. I would like to thank Dr. Danzl and Dr. Smith for their reviews and comments.

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