

“DOCTORS AMPUTATE FROSTBITTEN FEET OF ALL-AMERICAN RUNNER”

This tragic recent headline involving Marco Cheseto serves as a grim reminder of the potential devastation inflicted by frostbite and hypothermia from prolonged cold exposure. As we approach the winter season in Northeast Ohio, it would be prudent to review some of the more common serious environmental injuries which threaten our patients. Although there are several types of cold injuries (trench foot, chilblains) I will focus on two of the most severe—frostbite and hypothermia.



FROSTBITE

As with any other disease or traumatic entity, there are **risk factors** associated with the likelihood of frostbite occurrence. Obviously, the weather conditions are the primary determining factor. The majority of occupational outdoor cold injuries occur during the few coldest days of winter. Rates of cold injury dramatically increase when temperatures fall below 10 degrees F (-12°C). Wind, wetness and duration of cold exposure are all determinants for frostbite. Other risk factors include pre-existing disease (Diabetes, Raynaud's, chronic alcoholism) and age. The very young and very old are susceptible to frostbite and other cold related syndromes. Occupation is a risk factor. Military personnel, construction workers,

athletes who compete in winter sports are at risk. Smokers and those with peripheral vascular diseases have higher rates of frostbite complications.

The head, hands and feet are the usual areas affected by frostbite. Fortunately, most cases of frostbite are mild, but severe cases can lead to amputation of the affected areas. The damage from frostbite occurs because of cellular damage and tissue necrosis *inflicted by the freezing*. The degree of tissue injury depends on the temperature, the duration of exposure and the velocity of freezing. Tissue damage also occurs as a result of the *thawing* process. Immediately after thawing, a complex cascade of **chemical, cellular and vascular events** occur, which involve arachidonic acid, platelets, leukocytes, red blood cells and vasoconstriction. The end result is vein and **arterial thrombosis**, ischemia, necrosis and gangrene. It is interesting to note, that in our sepsis lecture, we pointed out that the final common pathway to end organ damage was also *thrombosis of the vasculature*.

Frostbite injuries, similar to burn injuries, are classified according to depth of injury and amount of tissue damage based on appearance (see diagram above). The exact degree of tissue injury is very often difficult to determine until days or weeks after the injury. First-degree injury is called **frostnip** and is usually identified by skin erythema, mild swelling, and lack of blisters. The patient may experience some stinging and burning sensations, but the overall prognosis is excellent. Second degree frostbite involves full-thickness skin freezing, edema and formation of clear blisters filled with prostaglandin-rich fluid. Blisters can form within 6 to 24 hours and after a few days they slough to form hard black eschars. Patients complain of numbness, followed by throbbing and aching pain. The prognosis is generally good.



Note the **clear blisters** in this case of second degree frostbite.

Third degree frostbite involves damage that extends deep below the dermis layer. Hemorrhagic blisters are noted and skin necrosis and eventual sloughing occurs. The patient may lose sensation initially, and later complain of burning, throbbing and shooting pains. The prognosis is poor.



Frostbite - Day 2



Note the hemorrhagic blisters in the above two cases of third degree frostbite.

Fourth degree frostbite involves the subcutaneous tissue, nerves, muscle, bone and tendon. Amputation is inevitable.

TREATMENT OF FROSTBITE

In the field, the goal is to prevent further cold injury, hypothermia and dehydration. The EMT should protect the patient from the cold and wind. Wet clothing should be removed and replaced with dry garments. Immediate dry heating of the frozen area may do more harm than good. Thawing is best done when there is no risk of refreezing and with warm circulating water at 104 to 107 degrees. Blisters may be left intact and treated with aloe vera cream, which alleviates some of the effect of the arachidonic acid cascade. The injured digits should be separated with cotton and wrapped with sterile, dry gauze. Elevation of the extremity may help prevent further edema and reduce pain. Antibiotics are controversial. Some authorities advocate the use of topical antibiotic ointments.

In some of the more severe cases, Penicillin IV is utilized. Tetanus immunization should be current (frostbite is a tetanus-prone wound).

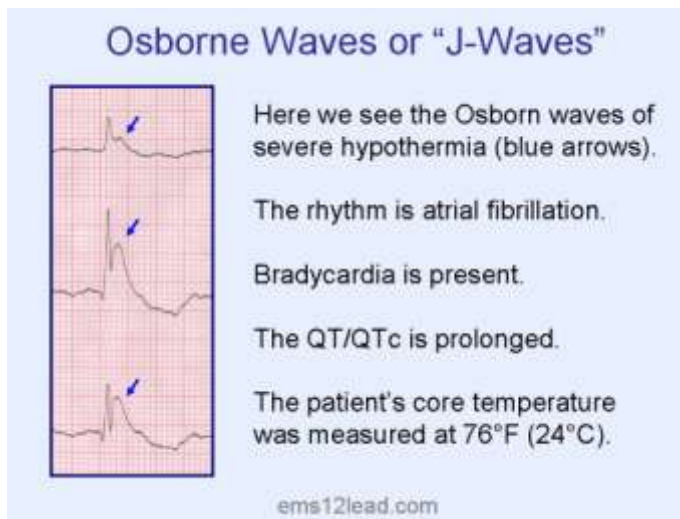
HYPOTHERMIA

Hypothermia has played a significant role in the history of the world, which is essentially a chronology of wars. Hannibal's crossing of the Alps, Valley Forge, Napoleon's invasion of Russia were major events which were severely impeded by hypothermia, which is defined as a core body temperature of $\leq 35^{\circ}\text{C}$ (95°F). Heat is conserved by peripheral vasoconstriction and behavioral responses. If behavioral responses are impaired (trauma, drug or alcohol intoxication, dementia), hypothermia risk increases. Heat can be generated by shivering and increased metabolic rates.

Accidental (environmental) hypothermia can occur in healthy individuals. Body temperatures of 32 to 35 degrees Centigrade fall into the mild hypothermia range. Generally, the body is able to make physiological adjustments to retain and generate heat. Heart rate, cardiac output and blood pressure rise to compensate for the lower body temperature. If the temperature drops below 32 degrees C, there is a progressive slowing of body responses and metabolism is reduced. Shivering (which is a major source of compensatory heat production) no longer occurs when the body temperature falls below 30 to 32 degrees C. As opposed to the physiological changes which occur in mild hypothermia (increase in heart rate, blood pressure and cardiac output), with **severe hypothermia there is decrease in cardiac output and blood pressure**. Hypothermia can be exacerbated by prior medical problems and alcohol. Some conditions can actually cause hypothermia, such as hypoglycemia, hypothyroidism, stroke, and Sepsis. Please recall from our sepsis lecture that fever is usually present, *but hypothermia can also occur*.

Hypothermia is associated with various EKG changes and arrhythmias. The Osborne, or J wave, is a slow positive deflection in the latter portion of the QRS complex. This J wave is characteristic of hypothermia. (see below). A typical sequence of rhythm disturbances for progressively severe hypothermia could

include: sinus bradycardia, atrial fibrillation with slow ventricular response, ventricular fibrillation and asystole.



The J wave is characteristic of severe hypothermia

Clinical features of hypothermia include loss of coordination, decreased blood pressure, confusion, lethargy and eventually coma. Metabolically, a “cold dieresis” ensues, which causes dehydration. Oxygen release to tissues is impaired by hypothermia. Acidosis may occur due to severe respiratory depression (and buildup of carbon dioxide) as well as to accumulation of lactic acid from shivering and poor tissue perfusion.

TREATMENT of HYPOTHERMIA

The patient should be removed from the cold environment and wet clothing removed. The patient should be moved gently because vigorous manipulation of the patient could precipitate ventricular fibrillation in the hypothermic, irritable heart. As a result, the EMT should spend extra time (up to 45 seconds) checking for pulses and signs of life in the severely hypothermic patient prior to initiating CPR.

Indications for airway control and intubation are as per usual. Warmed oxygen and intravenous fluids should be used. Most rhythm disturbances (sinus

bradycardia and atrial fibrillation) do not require specific therapy and will convert spontaneously with rewarming. The hypothermic heart is resistant to atropine, pacing and countershock— good reasons to rewarm the patient. Ventricular fibrillation will also be refractory to treatment until the heart is rewarmed. The 2005 AHA guidelines recommend a single defibrillation attempt, and if this is unsuccessful, CPR should be initiated until rapid rewarming is performed. Defibrillation should be reattempted when the core temperature is 30 degrees C.

Glucose levels should be checked and D50 administered if indicated. In alcoholics, thiamine should be given.

Rewarming Techniques

Passive rewarming is physiologically sound –it allows for slow, steady and safe body temperature increases with minimal stress to the cardiovascular system. **Active rewarming** is indicated for those patients with severe hypothermia, hypothermia secondary to underlying disease, and patients with cardiovascular compromise. Passive rewarming allows the patient to warm up on their own, using their own heat produced by metabolism. Active rewarming is divided into Active External and Active Core rewarming methods.

Active External Rewarming (AER) is the application of exogenous heat to the body surfaces. In the ED, warm water immersion—though beneficial-- is not practical. Rewarming with heated air forced through slits in plastic or paper blankets is the mechanism utilized by Bair Hugger, and is quite successful. There are some disadvantages to AER. It is ineffective in patients with poor perfusion or cardiac arrest. Application of external heat may cause peripheral vasodilation and venous pooling. Washout of lactic acid from peripheral tissues may lead to rewarming acidosis. The core temperature may continue to decline after rewarming has been initiated. Core temperature after drop has been attributed to the return of cold blood into the core induced by external warming.

Active Core Rewarming has advantages because the internal organs, including the heart, are warmed resulting in improvement of organ function. Peripheral

vasodilation is avoided. Warm humidified air or oxygen can be given by mask or ET tube. IV fluids can be warmed to 40 degrees C. NG or colonic lavage can be accomplished with warm saline. Peritoneal lavage is effective. More extreme measures include pump-assisted cardiopulmonary bypass whereby blood is shunted through a warming device before being returned to the patient. In general, if the patient has stable cardiovascular status, many experts believe that rapid rewarming is not necessary. Others would argue that profoundly hypothermic patients, although stable, should be rapidly rewarmed. Since there have been no prospective, controlled studies comparing rewarming modalities in humans, specific guidelines are not available.