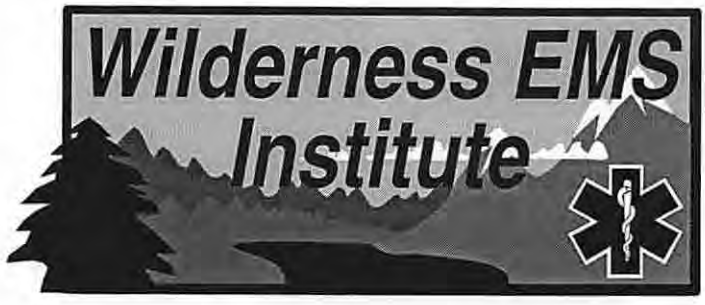
Chapter IX:

Cold-Related Disorders



Appalachian Search and Rescue Conference
Center for Emergency Medicine of Western Pennsylvania

Wilderness EMT Textbook

Chapter IX: Cold-Related Disorders

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Background Information

The ASRC-CEM Wilderness Emergency Medical Services Institute

The ASRC-CEM *Wilderness Emergency Medical Services Institute,* previously named the *Wilderness Emergency Medicine Curriculum Development Project,* is devoted to developing curricula for wilderness EMS providers and medical control physicians, and fosters wilderness EMS research. It is a cooperative venture of the Appalachian Search and Rescue Conference and the Center for Emergency Medicine of Western Pennsylvania. The ASRC is a large, tightly-knit wilderness search and rescue organization with eight teams throughout the mid-Appalachian states. The Center for Emergency Medicine is an emergency medicine and prehospital care research and teaching organization. It provides a medical helicopter service, an emergency medicine residency, Emergency Medical Services for the city of Pittsburgh, and conducts a variety of related projects.

The WEMSI Wilderness EMT Curriculum

This chapter is part of the WEMSI Wilderness Emergency Medical Technician Textbook. In concert with the WEMT Curriculum, the Textbook has been in development since 1986, and took as its starting point a program Dr. Conover developed for the National Association for Search and Rescue in 1980. The Project also draws on many other sources. These include the Wilderness EMT program of SOLO (Stonehearth Open Learning Opportunities), the WEMT program developed by Wilderness Medical Associates, and the Winter Emergency Care Course of the National Ski Patrol. The Wilderness Medical Society's educational and research publications provide needed background for the Textbook. The National Association of EMS Physicians has developed and has published clinical guidelines for delayed/prolonged transport; WEMSI protocols are also available as a model.

With textbooks used by its EMT and SAR prerequisites, the WEMT text provides the material needed to complete the Wilderness Prehospital Emergency Care curriculum established by the Wilderness Medical Society. (Indeed, early drafts of this textbook were a major resource for the WMS curriculum.) We assume that students have the knowledge and skills of an EMT-Basic or EMT-Paramedic. (The curriculum can accommodate both EMTs and paramedics in the same class.) We also assume that students have the knowledge and skills of the Virginia Ground Search and Rescue Field Team Member standards or better. (EMT standards are available from state EMS offices or the U.S. Department of Transportation. The Virginia GSAR standards and GSAR Manual are available from the Virginia Department of Emergency Services, 310 Turner Road, Richmond, VA 23225-6491.) The curriculum is competency-based rather than hours-based, but can be competed in 5-6 intensive days. The curriculum also recommends clinical training, for which guidelines are available in the Curriculum.

WEMT Textbook Chapter Development

An outline for each of the twenty sections was created by a Task Group of five to twenty selected members, but draws on many published sources and consultants. A Task Group Leader guides the Task Group in reviewing and revising the section, and the Curriculum Coordinator supervises all aspects of curriculum development. When the outline satisfies the Task Group, it goes to the **Editorial Board,** including officers of the ASRC and CEM. It also includes experts in emergency medicine, search and rescue, and education, and a State EMS director. Once acceptable to the Board, it is released to the public.

The Task Group Leader and Editor-in-Chief then produce a Textbook chapter based on the outline. Having a single editor provides a coherent, unified style. Basing chapters on the Task Group's Lesson Plans, as approved by the Editorial Board, ensures accuracy. Each chapter provides a glossary of terms new to a reader with basic EMT and SAR training. In the complete textbook, these glossaries are merged and alphabetized. Each chapter also provides references to support its statements and for further reading. Background that need not be presented in a class based on the Curriculum appear *in a small, italic font.*

The textbook will be commercially published when completed. All profits will be used to support curriculum development. The textbook will be submitted for publication in 1997. Until then, preliminary versions of the chapters will be printed in this format. These preliminary versions are for use at classes only when authorized by WEMSI. A Course Guide with information about Wilderness Emergency Medical Technician training and course scheduling, and a checklist for recommended in-hospital training are available. For a price list of available publications, write to: Center for Emergency Medicine, 320 McKee Place, Suite 500, Pittsburgh, PA 15213-4904, (412) 578-3203, or email wemsi+@pitt.edu.

We solicit suggestions from those reading any of our Lesson Plans or Textbook chapters. Please send your comments to the Editor-in-Chief, (see title page).

Educational Objectives

1. Define chilblain (pernio), describe the suspected cause, describe prevention, and outline the usual treatment.
2. Describe the differences between immersion foot (trench foot) and frostbite; outline the features of the three phases of immersion foot.
3. Describe the diagnosis, pathophysiology, causes and predisposing factors, prevention, immediate treatment, and extended management of frostnip and deep frostbite. Specifically, describe:
   a) the diagnostic and treatment differences between frostnip and deep frostbite;
   b) the difference between the initial and secondary phases;
   c) the effects of trauma on frostbitten tissue;
   d) predisposing factors, including the effects of common drugs including tobacco;
   e) recommended rewarming methods and post-rewarming wilderness treatment; and
   f) the basis for the adage "it's OK to walk on frostbitten feet," and its dangers.
4. List criteria for diagnosing hypothermia without a thermometer.
5. Define "incipient hypothermia" and its management.
6. List predisposing factors for accidental hypothermia.
7. Explain why it is important for hypothermic patients to avoid exertion, and why it is important not to transport hypothermic patients in the head-up position.
8. Define:
   a) mild and deep hypothermia;
   b) primary and secondary hypothermia; and
   c) acute, subacute, and chronic hypothermia.
9. Describe the role of Basic Cardiac Life Support (CPR) in the severely hypothermic patient, including:
   a) artificial respiration and oxygen;
   b) the appropriate ways to check for cardiac function in a very cold patient;
   c) when external cardiac compression is appropriate in a very cold patient and when it is not; and
   d) the appropriate rate for CPR (external cardiac compression and artificial respiration) in a severely hypothermic patient.
10. Describe the appropriate use of Advanced Cardiac Life Support techniques in the severely hypothermic patient, including:
    a) orotracheal intubation;
    b) cautions for avoiding ventricular fibrillation, and management of ventricular fibrillation in a hypothermic patient;
    c) the role of cardiac drugs in the severely hypothermic patient; and
    d) the role of bretylium in the hypothermic patient.
11. Discuss the role of cardiopulmonary bypass rewarming in decisions about where to transport nearly-dead hypothermic patients.
12. Explain the importance of the concepts "adding heat" and "active insulation."
13. Explain how the dictum "don't rewarm hypothermic patients in the field" may lead to poor patient care; explain why rapid rewarming is impossible in the wilderness, and why rescuers should add as much heat as possible to wilderness hypothermia patients.
14. Assuming access to a bathtub and hot water, but no way to transport hypothermic patients to a hospital for rewarming (e.g., a disaster or being stranded in winter), discuss the reasons for selecting rapid or slow rewarming for different types of patient.
15. Discuss the pros and cons of delaying the evacuation of a hypothermic patient for rewarming, or of delaying the evacuation for fluid replacement.
16. Explain the importance of administering fluids, intravenous or oral, and the importance of food calories, for hypothermic patients.
17. Define:
    a) passive rewarming;
    b) active rewarming;
    c) surface rewarming;
    d) core rewarming;
    e) afterdrop; and

f) rewarming shock.

18. Outline the advantages, disadvantages, proper technique, and appropriate uses of the following methods for adding heat to a hypothermic patient:
    a) a warm sleeping bag;
    b) warm inspired air or oxygen;
    c) warm IV solutions;
    d) heat packs;
    e) hydraulic sarong; and
    f) charcoal vest.
19. Discuss the advantages, disadvantages, and technique of warm bath rewarming of hypothermic patients when unable to transport to a medical facility.

Disclaimer

Recommendations for medical treatment in this curriculum are presented for training purposes only. We have attempted to ensure that all recommendations are consistent with current medical practices, but all care provided by WEMTs must be by the order of a physician. Your physician medical director must set protocols and standing orders, and you must follow them, even if they conflict with the recommendations in this curriculum.

Local Cold Injury

Chilblain (Pernio)

Background

Chilblain* is the mildest form of local cold injury. Strictly speaking, the problem is one for dermatologists, not one for you as a WEMT. However, chilblain appears in many lists of cold injuries, including the EMT textbooks and the EMT-Paramedic curriculum, so we include it here. Chilblain is a chronic problem for some people when exposed to the cold for long periods, but is in no way a medical emergency. Chilblain is caused by repeated exposure to cold but not necessarily freezing conditions, especially when wet or windy. Chilblain is similar to immersion foot in that it is caused by spasm of small arteries in the skin. Sun exposure may also play a part. [1] Some EMT textbooks confuse chilblain and frostnip.[2] They are not the same.

Diagnosis

Chilblain is a small area of red or cyanotic, slightly swollen, and sometimes scaling skin, usually on the face, ears, back of the hands, toes, and fingers. Changes generally appear some 12-24 hours after cold exposure in susceptible people. The rash and burning sensation generally disappear in a few weeks. Some people may go on to have a chronic form. Those who smoke, and those with Raynaud's Syndrome (spasm of the finger and toe arteries in the cold) seem particularly susceptible to chilblain.

Prevention

When out in the cold, wearing gloves and a facemask offers some protection. Staying in a warm climate such as the Caribbean offers even more protection. Keeping the skin from drying seems to help, too; a traditional American frontier preventative is rendered bear grease, though any good greasy skin cream should help. Avoiding tobacco is very important. Nifedipine (e.g., Procardia®, Adalat®), a vasodilator, prevents chilblain.[3]

Treatment

Nifedipine is excellent for treating chilblain. No other effective remedy is known.[3†]

* Note the spelling; it's not "chill-blain" or "chilblains."

† Dr. Murray Hamlet has treated about half a dozen patients with a presentation of cold exposure, cyanosis, erythema, pain, and skin thickening and scaling, with dimethyl sulfoxide (DMSO) 50-70% applied to the affected skin twice a day. This resulted in immediate relief of the pain, and possibly more rapid healing. No controlled studies of DMSO for this use are available.

Immersion Foot and Trench Foot

Background

Immersion foot is a condition that looks much like frostbite of the feet. However, immersion foot occurs at temperatures above freezing, but when the feet are continuously immersed in cold water. It is probably caused by reflex vasoconstriction. Trench foot is similar, and associated with wet, cold feet, but is associated with trauma from marching (or long search and rescue tasks). Another contributor to trench foot may be swollen feet leading to tight boots and poor circulation. Since wilderness search and rescue situations are in some ways similar to battle, we might take heed from the experiences of World Wars I and II. In 1944, General Patton remarked that he had more casualties from trench foot than from the German army.[4]

Phases and Diagnosis

Immersion foot develops in three phases.

The first phase is of vasospasm, with a cold, pale foot. The foot is numb and feels "wooden." If this lasts for a long time because of cold, wet conditions, edema may develop.

The second phase occurs after rewarming. The foot becomes red, hot, and painful, sometimes with increased foot pulses. The foot may form blisters, and will look like a mildly frostbitten (and rewarmed) foot, but the heat and warmth is more notable.

The final phase is healing, which may last for weeks. Repeated cold exposure may result in chronic injury with redness, sensitivity, and scaling of the skin. Cold sensitivity and pain may linger for years.

Treatment

Treatment for immersion foot is much the same as for frostbite, but there is little or no need for active rewarming, because the patient usually doesn't ask for medical attention until after it has rewarmed.*

Prevention

Waterproof boots and frequently-changed dry wool socks are the keys to prevention of immersion foot. Vapor-barrier socks (waterproof bags or socks) worn under thick wool socks may help keep socks from getting wet from perspiration, and are appropriate in extreme SAR situations. You can help educate SAR team members and leaders about the need for careful foot care, emphasizing that immersion foot or trench foot turns a rescuer into a rescuee. Dehydration and exhaustion make trench foot more likely, as well as decreasing the team member's efficiency. SAR team members often ignore their feet during the excitement and confusion of a search or rescue. When operations go on for several days, foot care becomes even more important. During long searches in the cold, inspecting members' feet during rest breaks or returns to Base Camp might be a duty for team leaders or base medical personnel.

Frostnip

Background

Frostnip, also known as **superficial frostbite** or first-degree frostbite, is freezing of the superficial tissues, but sparing the deeper tissues. This is seldom a problem in your rescue patients, but may be a problem in team members and WEMTs involved in winter operations.

Diagnosis

Frostnip commonly affects fingers, toes, ear lobes, and noses, and you can recognize it by a sudden blanching of the nose, ear, or fingertip. Although

* Clinical experience from the U.S. Army also suggests that there is no role for vasodilators in the treatment of immersion foot or trench foot. This is unlike late frostbite, which has a zone of vasospasm and responds remarkably well to intra-arterial reserpine.

the part is pale or yellowish, it is still soft to the touch, not hard or "woody" as in deep frostbite. Numbness is not a useful symptom for diagnosing frostnip. A frostnipped area may be numb, but cold skin is numb well before it becomes frostnipped.

Prevention

The wind-chill temperature does not represent a good index of hypothermia danger. However, it is a good index of the danger of frostnip and frostbite. Face masks and goggles are a necessity for windy winter search and rescue operations. If you don't have face masks in bitterly cold weather, or when the wind is very strong, it is a good idea for team members to check each other's faces regularly for the blanching that is the first sign of frostnip, and to stop and rewarm immediately.

Treatment

Treatment of frostnip is simple: rewarming by a warm hand over the nose or ear, or by placing a frostnipped finger in the mouth, in an armpit, or in a warm pocket. On rewarming, the affected part tends to turn red, painful, and possibly slightly swollen, but no permanent damage results. Providing a warm armpit for a friend's frostnipped toes is supposedly a mark of true friendship. Oxygen is of no proven benefit, but you may administer it if readily available.

Deep Frostbite

Deep frostbite is different from frostnip: the deep tissues are also frozen.

Diagnosis

In deep frostbite, the subcutaneous tissues are frozen solid, and the affected part feels hard, like a piece of wood or frozen meat.

Pathophysiology

The primary problem in frostbite is formation of ice crystals in the interstitial fluid (between the cells; also known as intercellular fluid). These crystals grow, pulling water out of cells and causing cell damage by dehydration.

Freezing causes great tissue damage, but the effects are postponed until the part is rewarmed. Once the frozen parts are rewarmed, the damage becomes evident, with excruciating pain and blistering.

Trauma to frozen tissue may push sharp ice crystals into cells, rupturing them. Napoleon's battle surgeon, Baron Larrey, suggested rubbing the affected parts in snow to increase circulation. We now know this will cause much more damage than it cures.

Unless circulation is impaired, for instance by too-tight boots, or from dehydration or hypothermia or exhaustion, the human body is highly resistant to freezing. Note that wearing two pair of socks with a pair of boots fitted for one pair of socks is a common cause of frostbite ("two-sock frostbite"), even appearing in the military medical literature.[5] Even a vigorous, healthy person may get frostbite if it is very cold and the wind is very strong across an unprotected nose or ear. It is also possible to get frostbite by touching a bare hand to a piece of supercooled metal, or by spilling subfreezing stove fuel on a hand. (Gasoline and alcohol are still liquid at temperatures below the freezing point of water.)

A secondary phase of damage occurs after rewarming. The frozen and thawed tissues become inflamed, with damage to the inner lining of blood vessels, which leads to small clots of platelets.[6] The damaged capillaries also leak fluid, causing tissue swelling, so the blood becomes very concentrated in the capillaries; even where there are no platelet plugs, red blood cells become so closely packed that they clog the small vessels. (These findings have been documented by angiograms, nuclear scans, and plastic casts made of blood vessels in frostbitten animals.[7] Between the normal tissue and the inflamed (frozen and thawed) tissue, the blood vessels go into spasm, decreasing blood circulation to the damaged tissue.[8] A major goal of management is to prevent this additional damage after rewarming.

Resistance and Predisposing Factors

People vary in their resistance to frostbite. Prior frostbite or immersion foot is known to predis-

pose to additional local cold injury. There are probably racial variations in frostbite susceptibility. In Korea, blacks suffered 52% of the frostbite cases, even though they formed only about 10% of the exposed population.[9] Smoking, shock, hypothermia, malnutrition, fatigue, high altitude, and chronic medical problems all probably predispose to local cold injury, because they interfere with peripheral circulation.[10]

Alcohol is thought by some to increase peripheral blood flow and thus might be thought to help prevent frostbite; however, it predisposes to hypothermia, which may make frostbite more likely, and clouds the judgment, which is probably the #1 cause of frostbite.

People can become acclimatized to cold, specifically to have better blood flow to cold extremities.[11] This probably protects against frostbite.

Frostbite can be graded into first, second, third, and fourth degree as with burns. However, the extent of injury cannot be assessed until many hours or days after the injury, so grading is of no particular use to WEMTs.

Immediate Treatment

The first thing to remember is to check for life-threatening hypothermia before treating frostbite. Generally, you can safely treat both frostbite and hypothermia at the same time. However, if faced with an unstable patient with both frostbite and hypothermia, you might want to delay rewarming of the frostbitten extremities until the patient was stable.

The best "street" treatment is to transport rapidly to a hospital where definitive rewarming can occur. However, when the rescue team cannot begin transport for a long time, or there is a long transport time to the hospital (more than an hour), you should rewarm en route. If the patient is hypothermic, rewarm the core and protect the patient from further cold exposure before worrying about frostbite. However, there is no justification for keeping the frostbitten part cold during transportation. Rapid rewarming is better than slow rewarming, so some recommend applying cold packs to a frostbitten limb, or leaving it out of the patient packaging during evacuation. This is ridiculous. The chance of causing more extensive frostbite far outweighs any potential advantage of "preventing slow rewarming" during transport. Indeed, studies show that slow rewarming (e.g., room temperature air or a warm sleeping bag) is better than very slow rewarming (e.g., ice water bath, or leaving the frostbitten limbs cold).[12]

Rapid Rewarming — The proper definitive treatment, in the hospital or in the field, is **rapid rewarming in 105-110°F (41-43°C) water.** There are decades of experimental studies[12, 13] and clinical experience that shows this treatment is best.[14, 15] However, don't rewarm until there is no danger of re-freezing. (Refreezing causes severe additional damage.[12]

Folk and medical traditions worldwide used to recommend slow rewarming. There were several reasons for this. First, the writings of Hippocrates can be interpreted as warning against rapid rewarming. Such classical sources were highly regarded in Europe in the Middle Ages, from which we obtain much of our folklore and medical superstitions. Second, rapid rewarming often meant rewarming in front of a fire, which can cause uneven heating and cause burns. This is probably the reason Napoleon's surgeon general, Baron Larrey, recommended against rapid rewarming. Third, slow rewarming is much less painful than rapid rewarming, and with rapid rewarming, the parts become more red and swollen. Nonetheless, the final tissue loss is less with rapid rewarming. So, regardless of what you hear about slow rewarming by rubbing with snow or immersion in ice water, use rapid rewarming.[12]

Frostbitten limbs are numb, so don't cook them in too-hot water, or burn them by rewarming in front of a fire. If you don't have a thermometer, your elbow makes an adequate improvised replacement. Hold your elbow in the water for a few minutes. It should feel very warm but not painful. (Fingers and hands are not as accurate for checking absolute temperature. Think of how hot even cool water feels on your hands after you've been out in the cold for a while.)

Frostbite in Litter Patients — The best treatment for frostbite in a litter patient would probably be as follows. Wrap the frostbitten extremities in towels or thick pieces of clothing soaked with warm (40-42°C = 104-110°F) water. Next, wrap waterproof plastic and pieces of closed-cell foam around the towel-wrapped extremity, then place the patient in the litter. A liter of water can be quickly heated on a stove while the patient is prepared for evacuation. The water will cool during evacuation; this is acceptable. There is no benefit to providing continued warmth once the frostbitten part has rapidly thawed.[12] (If the evacuation will be very long, you may want to take the wet towels off several hours later to allow the skin to

dry.) Heat packs and heating pads are well-known for the burns they cause during rewarming,[16] sometimes even to extremities that were not truly frostbitten. If it is so cold that you must use heat packs to prevent the wet towels from cooling too fast, even when insulated, make sure the hot packs aren't in direct contact with the skin. Some might argue against thus treating frostbite in a patient who is also hypothermic. True, rewarming of the periphery should be avoided in hypothermia, but the hands and feet (not the arms and legs) have direct venous connections to the core, so rewarming of the hands and feet is quite acceptable, even if the patient is hypothermic.[17]

Dextran — There is good evidence from animal studies that giving intravenous low-molecular weight dextran will decrease tissue loss. *[13, 18-21]

To preserve as much tissue as possible, dextran should be started as soon as possible, even before rewarming. Even if the frostbite has been slowly rewarmed, giving IV dextran later may help minimize damage.[22]

Dextran should be continued for up to five days.[23]

Dextran is discussed further for the treatment of hypothermia, below. (See also the chapter on *Wilderness Trauma for more about dextran.)*

Extended Management

The guidelines below are for the extended management of frostbite, usually after your responsibility as a WEMT ends. However, you may evacuate patients with rewarmed frostbite, so you should be familiar with the basics of how to care for rewarmed frostbite. We also include some additional material (in small italics) that may be of use if you are caught in an extended bivouac or disaster situation with no access to a hospital.

Appearance — After rewarming, the skin will break down. It will look like a second degree burn, with redness, blistering, and tissue necrosis. As long as there is no significant infection, the devitalized areas will turn black ("dry gangrene") and fall off in their own time.

Pain — Tissue that has been frostbitten and rewarmed is very painful. Plan to give large doses of narcotics, if you have them available.

Protect from Damage — Tissue that has been frostbitten and rewarmed is very susceptible to further damage from pressure. Protect the area from pressure. (E.g., surround a damaged foot in a large, soft dressing, and keep Stokes litter straps away from the frostbitten area; perhaps, place a cutout cardboard box over the extremity to prevent pressure from sleeping bag or blankets.)

Protect from Refreezing — Refreezing is known to cause very severe damage.

Avoid Tobacco — Patients must **not** smoke, because of the vasoconstrictor effect of tobacco. Caffeine may also have a vasoconstrictor effect, so don't give the patient caffeine (coffee, tea, or cola drinks).

***Avoid Surgery** — If the devitalized areas get infected ("wet gangrene"), a surgeon should debride (trim) the infected areas, and the patient should receive antibiotics. (In olden times, wet gangrene was prevented by placing maggots on the area; they only ate devitalized tissue, thus removing sources of wet gangrene.) Otherwise, there is no reason for amputation or debridement. Such surgery may cut off tissue that could recover and heal. Damaged tissue gradually turns black and separates, leaving healthy pink tissue underneath.*

Drug Therapy

Aspirin — Low doses of aspirin (a single aspirin a day, or less) will decrease platelet stickiness, and will probably decrease platelet "clots" in the small vessels. The anti-platelet effects of aspirin may be decreased at doses more than one or two a day. One study of the effect of aspirin to prevent stroke showed that a dosage of 30 mg. a day (half of a "baby" aspirin) was as effective as larger doses and less likely to cause gastrointestinal problems.[24] but for a variety of reasons, the standard anti-platelet dosage in the U.S. seems to have standardized on 160-162.5 mg (two baby aspirins, or half an adult tablet) a day.

Ibuprofen — A non-steroidal anti-inflammatory drug (such as ibuprofen) may limit tissue damage that usually happens after rewarming. (One study showed that anti-inflammatory medicine methi-

* The only known human study, by Doolittle and Sumner at Bassett Army Hospital in Alaska, was terminated prior to completion. The animal studies and the excellent clinical results when dextran was used in humans convinced experimenters it was unethical to continue the study. (The study required that "control" patients not receive dextran.) This information is unpublished, and provided by Dr. Murray Hamlet of the Army's Research Institute of Environmental Medicine.

mazole* worked better than aspirin, but no studies comparing low doses of aspirin vs. ibuprofen are available.)[25] The usual dose for average size adult is 800 mg. three times a day. If you only have aspirin, 4 aspirin tablets four times a day (16 a day) will provide an anti-inflammatory effect, but at the cost of losing some of the anti-platelet effect. (Too-high aspirin levels are usually signaled by ringing of the ears: tinnitus. Those developing tinnitus on large doses of aspirin should cut down on the dosage.) People should take aspirin or ibuprofen on a full stomach or with antacids to avoid stomach irritation. Don't give pregnant patients, or those with peptic ulcer or kidney problems, aspirin or ibuprofen.

Vasodilators *— The most impressive improvement in the vasospasm of human frostbite comes from intra-arterial reserpine,[8] even though some researchers did not see vasospasm in animal models.[26] Unfortunately, reserpine is no longer sold for intra-arterial use. Some suggest that the inflammatory second phase of frostbite (and immersion foot) has much the same pathology as chilblain (see above).[27] If so, the excellent record of nifedipine in treating chilblain suggests that nifedipine should also be used for frostbite or immersion foot.[3] However, unpublished preliminary animal studies by the Army Research Institute of Environmental Medicine showed that vasodilators other than reserpine (such as nifedipine, lidocaine, and ketanserine) were far inferior to reserpine, resulting only in a slight improvement in outcome. One published Army study of a vasodilator other than reserpine showed no improvement.[28]*

Sympathectomy *— Sympathectomy (cutting the sympathetic nerves) to an area has been intermittently tried, over the years, for the treatment of frostbite. Several studies show it to be useful, but the best studies show it to be inferior to administering dextran.[13] Too, dextran can be readily given in the field, whereas sympathectomy must be delayed until reaching the hospital and arranging for an operating room. Sympathectomy is also known to cause impotence in men.[8] And, research reports continually cite the importance of immediate treatment, regardless of whether it is dextran or sympathectomy.*

Aloe Vera *— Some recent research has focused on the use of medicines such as ibuprofen and extracts of the aloe vera plant on frostbite. One animal study shows that aloe is effective.[25] However, aloe does not penetrate the deeper tissues. The clinical human study most often discussed as showing support for aloe in frostbite suffers from problems with selection bias and lack of adequate controls.[29, 30] We need more studies to tell whether aloe really works. If you wish find out more about these facets of scientific research, we recommend you read the book Studying a Study and Testing a Test.[31] Using aloe on frostbite is reasonable and unlikely to do harm, if unproven, and is recommended in some protocols,[32] but is not a standard part of most wilderness medical kits, as are aspirin and ibuprofen. A recent review article in the Journal of the American Academy of Dermatology says: "Well-controlled trials in humans must be carried out with the use of a standardized preparation of aloe vera before aloe vera's use can be recommended in the treatment of any skin condition; however, the evidence for aloe vera's beneficial effects is ample enough to warrant further clinical trials."[33]*

Army researchers have also tried many other drugs for frostbite, including antihistamines, with no positive results.

Walking on Frostbitten Feet

You cannot walk on a foot with frostbitten toes once the toes have been rewarmed, because of the massive tissue damage and severe pain when you walk. You **can** walk on frozen feet with less damage and with much less pain. So, if your survival depends on continued hiking, you may walk on frostbitten feet. However, as this will cause additional damage to your feet, we recommend this only when your survival is at stake (e.g., getting down off a mountain when suffering from high-altitude pulmonary edema, or caught in a severe storm).

Hypothermia

General

Hypothermia is a complex topic, and there is much that we don't know about it. Worse, what we **do** know about hypothermia is not well-publicized. As a result, care of hypothermic patients is sometimes poor. This chapter presents what we know about field treatment of hypothermia.

Case Reports

Ethical constraints limit hypothermia laboratory research on humans to mild hypothermia, or at the most, the warmer end of deep hypothermia. Animal studies are difficult to apply to humans. And, large hospital studies of deeply hypothermic patients are hampered by the small number of cases seen at any one hospital. Not only that, most of the cases seen in hospitals are the result of alcohol or drug intoxication, massive trauma,

* Methimazole is most commonly used to decrease the function of the thyroid gland, so it would not be a good choice to actually treat human frostbite.

chronic illness, or to a lesser extent, immersion hypothermia. Therefore, it is important for you, as a WEMT, to observe carefully and report any cases of deep hypothermia you care for, especially cases related to outdoor exposure. Please feel free to contact the Task Group Chairman with any observations you make— you may be able to help others by publishing your observations in a medical journal.

Definition

Hypothermia is an abnormally low body core temperature. Normal is 99° F (37° C), and hypothermia is any temperature less than 95° F (35° C). Core temperatures down to 90° F (32° C) are generally considered mild hypothermia. Temperatures below this are termed severe hypothermia. Differences in **type** of hypothermia are as important as core temperature, as discussed below.

Diagnosis

You may definitively diagnose hypothermia only by a low rectal or tympanic membrane (eardrum) or esophageal temperature. Oral (mouth) and axillary (armpit) temperatures may be much lower than the "core" temperature, especially in mouth breathers or those who are chilled. (**Important note:** patients with a very cold oral or axillary temperature, even if they aren't truly hypothermic by rectal or tympanic membrane temperature, are likely severely chilled, as discussed below under *Incipient Hypothermia.*[34] Such patients need shelter, food, water, and rewarming.) Unless a person has been smoking or drinking warm drinks, though, a normal oral temperature rules out hypothermia, and an oral temperature above 90° F (32° C) rules out deep hypothermia. Similarly, you can use a warm axillary temperature to rule out hypothermia. We note there are several reports of wilderness rescuers being unwilling to take rectal temperatures, even though equipment is available. It is **not** difficult to obtain a rectal temperature on a wilderness patient, even in extreme environments. However, rescue teams can avoid this unpleasant task by obtaining an infrared tympanic membrane (ear) thermometer suitable for wilderness use. See the chapter on *Patient Assessment for more on the measurement of temperature in the wilderness.*

Some texts and many articles in outdoor magazines make a point of providing a chart of the signs and symptoms that develop at a particular core temperature, during the onset of hypothermia. But, the individual variation in response to cold is so wide as to make such charts meaningless.[35, 36] You generally will see ataxia (a staggering gait) early in hypothermia, because cold may directly affect the nerves to the legs, even though the core is still at a near-normal temperature; later, you will note the characteristic blank stare and then slurred speech as the brain and other core organs are affected by the cold. You may see shivering, or shivering may stop, at various temperatures, depending on the particular person. Those with exhaustion (subacute hypothermia) may not shiver at all.

As noted in the chapter *on Heat-Related Disorders*, thirst is not an accurate indicator of dehydration; similarly, people's perception of core temperature is poor. Feeling cold is not a good indicator of hypothermia.[37]

You should suspect mild hypothermia in a cold search and rescue team member when you see evidence of mental or physical dysfunction. Examples of mental dysfunction include impaired short-term memory, difficulty concentrating, slurred speech, withdrawal from activities, and apathy. Examples of physical dysfunction include difficulty walking, numb fingers, and impairment of fine coordination, as in tying boots. You may also see protective efforts, such as uncontrollable shivering, or holding arms tightly against the sides. The most important treatment for such a case of hypothermia is to **bivouac immediately** and rewarm the team member before going on.[38] Impairment of thinking is a significant factor with even mild hypothermia, and what is worse, the hypothermic person may not be able to detect it. Thus, even mild hypothermia may contribute to many accidents and deaths from other causes.[39]

One particularly interesting study looked at the effects of mild hypothermia (34-35° C=93-95° F) on mental functioning. There were two major findings. First, memory: subjects were able to recall previously learned information without difficulty. However, their ability to remember new

information was only about 70% of normal. (Perhaps this explains the common observation of hypothermic people forgetting where they put objects such as their mittens.) Second, thinking: subjects could perform calculations as accurately as when normothermic. However, it took them about half again as long (1.5 times normal).[40]

Some people who are found dead of hypothermia have, paradoxically, undressed themselves. Some suggest that, as compensatory mechanisms fail, a last flush of warmth reaching the body surface creates a feeling of warmth.[41, 42] One reference to the Gallipoli campaign of 1915 describes how those who were hypothermic acted: "Filled with a spurious warmth, they took off their coats, boots, and some their tunics."[43]

There is a characteristic "J" wave that is seen on the electrocardiogram (EKG) of some patients with hypothermia. It is sometimes called the "Osborn" wave after one author who described it.[44, 45] However, Tomaszewski first described the J wave, so it should by rights be called a Tomaszewski wave if someone's name must be attached to it at all.[46] This is only of academic interest, because (1) the J wave makes no difference in prognosis or treatment of hypothermia, and (2) for diagnosing hypothermia, an EKG makes a poor substitute for a thermometer.

Incipient Hypothermia:

All the classic signs of hypothermia listed above may be present in a person with a normal core temperature. However, this person, if not intoxicated or ill with some other disease, has probably been subject to severe cold stress, has cooled his or her periphery as much as possible, and has just about exhausted all compensatory mechanisms. Fatigue, exhaustion (glycogen depletion) and excessive cooling of the bulk of the extremities, as occur in such people, are a prelude to hypothermia. Treat them as if they have mild subacute hypothermia, for if you don't, they will soon become hypothermic![34]

Predisposing Factors

A great many medical problems can predispose to hypothermia in urban hypothermia patients.[47] However, the predisposing factors encountered in wilderness travelers show a different spectrum from those found in urban patients.

In animals, alcohol causes a drop of about 2° in rectal temperature, which can be prevented by keeping the animals in a warm room.[48] Alcohol does cause at least some vasodilation, leading to increased heat loss, and suppresses shivering to at least some degree. There is also a direct effect on the hypothalamus to predispose to hypothermia, particularly in the elderly.[49]

More recently we have come to realize that, in healthy cold-stressed humans, physiological effects from ingesting small amounts of alcohol are not likely to cause significant hypothermia.[50-52] However, ethanol **does** predispose to hypothermia in the same way it predisposes to snakebite: people do really stupid things when they're drunk. Like sleeping out under a bridge when it's very cold. Or like day-hiking in cotton clothes with no raingear.[53]

The active ingredient in marijuana (THC) has been found to cause a core temperature drop of up to 2° C in monkeys, and may predispose humans to hypothermia.[54, 55]

Any factor that limits the body's ability to produce heat, such as exhaustion, fatigue, starvation, dehydration, or illness, may interfere with the shivering responses and predispose to hypothermia. Any factor that limits the heat-conserving vasoconstrictor response, or limits the ability of the hypothalamus or the body to recognize or respond to cold, will also predispose to hypothermia. Examples include old age, infancy, those with diabetic neuropathy (which affects the nerves that control vasodilation), those with hypothyroidism (a low level of the hormone that regulates the metabolic rate), iron deficiency,[56] and those on certain medications.

Depressed people, those with psychiatric problems, and those with seizure disorders account for a large number of lost people. The effects of psychoactive drugs on metabolism and body temperature are complex. However, we think that tricyclic antidepressants such as amitriptyline (e.g., Elavil®), benzodiazepines such as diazepam (e.g., Valium®), chlordiazepoxide (e.g., Librium®), and alprazolam (Xanax®), antipsychotic medications such as haloperidol (e.g., Haldol®), thioridazine (e.g., Mellaril®), and trifluoperazine (e.g., Stelazine®),[42, 57-60] and

anti-seizure medications phenobarbital and valproic acid (e.g., Tegretol®)[61] all likely predispose to hypothermia. You should check carefully for hypothermia in such patients, even in a warm environment.

Other medications, commonly used by wilderness travelers or carried in their wilderness medical kits, may perhaps predispose to hypothermia. The evidence is not conclusive, but enough for you to be especially vigilant in watching for hypothermia in those taking these medicines.

Some wilderness travelers take phenothiazines such as hydroxyzine (Atarax®, Vistaril®) and promethazine (e.g., Phenergan®) to prevent motion sickness; these medications are thought to predispose to hypothermia.

Narcotics such as codeine and hydrocodone increase heat loss, and may directly influence the brain to cause hypothermia.[62-68]

Steroids such as prednisone, and non-steroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen (e.g., Motrin®) prevent shivering, at least in some situations, and thus probably predispose to hypothermia.[69, 70]

Acetaminophen (e.g., Tylenol) and aspirin also seem to depress the shivering response in cold exposure.[71, 72]

Benzodiazepines, including drugs such as diazepam (e.g., Valium®), alprazolam (Xanax®), and flurazepam (e.g., Dalmane®) are used for anxiety and as "sleeping pills"; they also seem to predispose to hypothermia,[73, 74] though some studies don't support this effect.[75]

Alcohol and barbiturate intoxication are well-known for leading to severe hypothermia, but surprisingly, barbiturates and alcohol have a protective effect, allowing survival from very low temperatures.[51, 76]

Hypoglycemia is well-known as a cause of secondary hypothermia. Giving IV glucose will usually correct this type of hypothermia rapidly. The only exception would be a Type I (insulin-dependent) diabetic, who might also need insulin.[77, 78]

People can acclimatize to cold by increasing the blood flow to cold extremities, which probably helps prevent frostbite.[11] Increasing the amount of fat in the diet will increase endogenous heat production by a straightforward biochemical effect, and may offer some slight protection against cold. Unfortunately, however, people do not acclimatize to cold in any way that helps prevent hypothermia. People can "acclimatize" to sleeping in cold environments, but this seems to merely be the ability to tolerate development of mild hypothermia during sleep without shivering or discomfort, not any significant protection against hypothermia.[57, 79, 80]

Handling Hypothermic Patients:

Besides the well-publicized need for **gentle** handling to avoid ventricular fibrillation,[81] there are two more major considerations for handling profoundly hypothermic patients:

No Exertion

There are several well-documented cases of deeply-hypothermic people who died during rescue. Most of these were people who were inactive, and most were immersed in cold water. When they exerted themselves during rescue, they collapsed and died. A reasonable supposition is that sudden activity of cold arms and legs caused a reflex vasodilation, and then a sudden rush of cold, acidotic blood in toward the heart, causing ventricular fibrillation. While similar cases regarding hypothermia victims on land are not as well documented, it seems prudent to tell patients **not** to help in any rescue efforts.[71]

No Tilting

Some of the earliest English-language hypothermia articles mention unconsciousness, sudden seizures, and deaths of hypothermia victims who were being carried with the head of the litter elevated.[82, 83] We can reasonably surmise that these patients suffered inadequate cerebral circulation from a combination of low blood volume and the head-up position. While the use of warm IV solutions may help prevent this problem, it still seems prudent to transport hypothermic patients in a level position.[84] When rescuers **must** transport a patient in the head-up position, (e.g., raising the patient out of a narrow pit in a cave) and anti-shock trousers are available, consider inflating the

trousers before the vertical section and deflating them afterward; discuss this with your Wilderness Command Physician.

Hypothermia Etiology

Hypothermia is a heterogeneous spectrum of illnesses grouped by a single, abnormal, vital sign: a low temperature. An unvoiced assumption in most of the hypothermia literature is that statistics from one type of hypothermia (most commonly urban hypothermia) can be generalized to other types of hypothermia. There are sound theoretical grounds for thinking that different types of hypothermia should be treated differently.

Several well-known papers suggest that the prognosis for hypothermia is grim, and that aggressive rewarming is particularly hazardous, especially in the field. Yet, these studies included mostly older individuals, many with intoxication or severe illness.[85-88] *Strong anecdotal evidence supports the view that patients* **without** *underlying disease or intoxication (i.e., young, healthy people with "mountain" hypothermia) are much more likely to survive a given degree of hypothermia. And, anecdotal evidence very strongly suggests that young, healthy victims of immersion hypothermia have a very good prognosis. This compares with ill or intoxicated "urban" hypothermia patients with the same core temperature, who often die. Further, although much of the literature condemns rapid external rewarming in the management of severe hypothermia, anecdotal evidence for the utility of rapid rewarming in immersion hypothermia is abundant and pervasive.*

Several published papers point to the heterogeneous nature of hypothermia. An editorial in **The Lancet** *states "Immersion hypothermia is very different from mountain hypothermia ... Disturbances of fluid and electrolyte balance, and increases or reductions in blood-glucose and cortisol levels, have been recorded in this group {mountain hypothermia}, whereas in immersion hypothermia fluid shifts are not prominent and blood glucose and cortisol are high."*[89]

Lloyd states: "There are at least three common types {of hypothermia}: firstly, `immersion' hypothermia, where the cold stress is greater than the maximum heat production of the body; secondly, exhaustion hypothermia, where the critical factor in depletion of the body's usable food (fuel) stores; and, thirdly, subclinical chronic hypothermia, which is the usual type found in the elderly, in whom, though the core temperature may be normal, the chronic cold stress has resulted in considerable intercompartmental fluid shifts—which complicate the management of any superimposed acute episode. The types can be distinguished only by the case history; for example, a climber disabled by a broken leg will probably cool as if immersed, whereas a strong swimmer lost overboard in relatively warm water is a candidate for exhaustion hypothermia."

" . . for thin or emaciated prisoners with little hope the best treatment for immersion hypothermia is immersion in a hot bath. Clinical experience with more normal victims has widened the scope and current opinion is that for immersion hypothermia the best treatment is in a hot bath, with the caveat that it may only be of real value if done within 20 minutes."[90]

Charles Bova, Medical Director of New Mexico Emergency Medical Services, writes: "I suggest that for `immersion' accidental hypothermia rapid, external rewarming may be the treatment of choice, whereas slow, passive rewarming has fewer complications, such as rewarming shock, than do other methods of treating accidental hypothermia."[91] *Coniam, writing in* **Anaesthesia**, *says: "There is general agreement that the victim of sudden immersion with rapidly produced hypothermia is best warmed rapidly by immersion of the trunk in a warm bath (40-42° C)".*[92]

One letter discussing hypothermia mortality figures states "Primary and secondary hypothermia are potentially as different from one another as a gunshot wound to the head is from carcinoma of the lung."[93]

Johnson, writing in the Journal of the **American College of Emergency Physicians** *in 1977, agreed with Lloyd, identifying three clearly-identifiable subsets of hypothermia: immersion, exposure, and urban.*[94]

Lloyd's review summarizes the division of hypothermia into acute, subacute, and chronic.[36]

Hypothermia Classification

Hypothermia is not a disease. It is an abnormal vital sign. Hypothermia may be caused by many different things. A low core temperature may indicate underlying medical disease such as hypothyroidism. Or, it may come from simple exposure to overwhelming cold.*

For example, suppose you were with a group cross-country skiing across a frozen pond. The ice breaks under your friend's skis, precipitating him into the pond. You pull him out, but he is already hypothermic.

Hypothermia may also result from a combination of minor medical disease plus a mildly cold environment. For instance: an elderly woman on phenothiazines has a home heating furnace that breaks. Friends find her 2 days later in her bed, cold and incoherent.

In both cases, the core temperature is the same: 89° F (31° C). Should they be treated exactly the same? No. Is the treatment the same for all patients with a pulse of 130 the same? No. The definitive treatments are very different for sinus tachycardia, atrial flutter, and ventricular tachycar-

* This section on hypothermia classification has, at present, no effect on the field treatment of hypothermia. However, understanding hypothermia classification will lead to better understanding of the general management of hypothermic patients, so we include it here. Physicians discussing hypothermia may refer to these classifications, so WEMTs will want to prepare themselves by understanding this material.

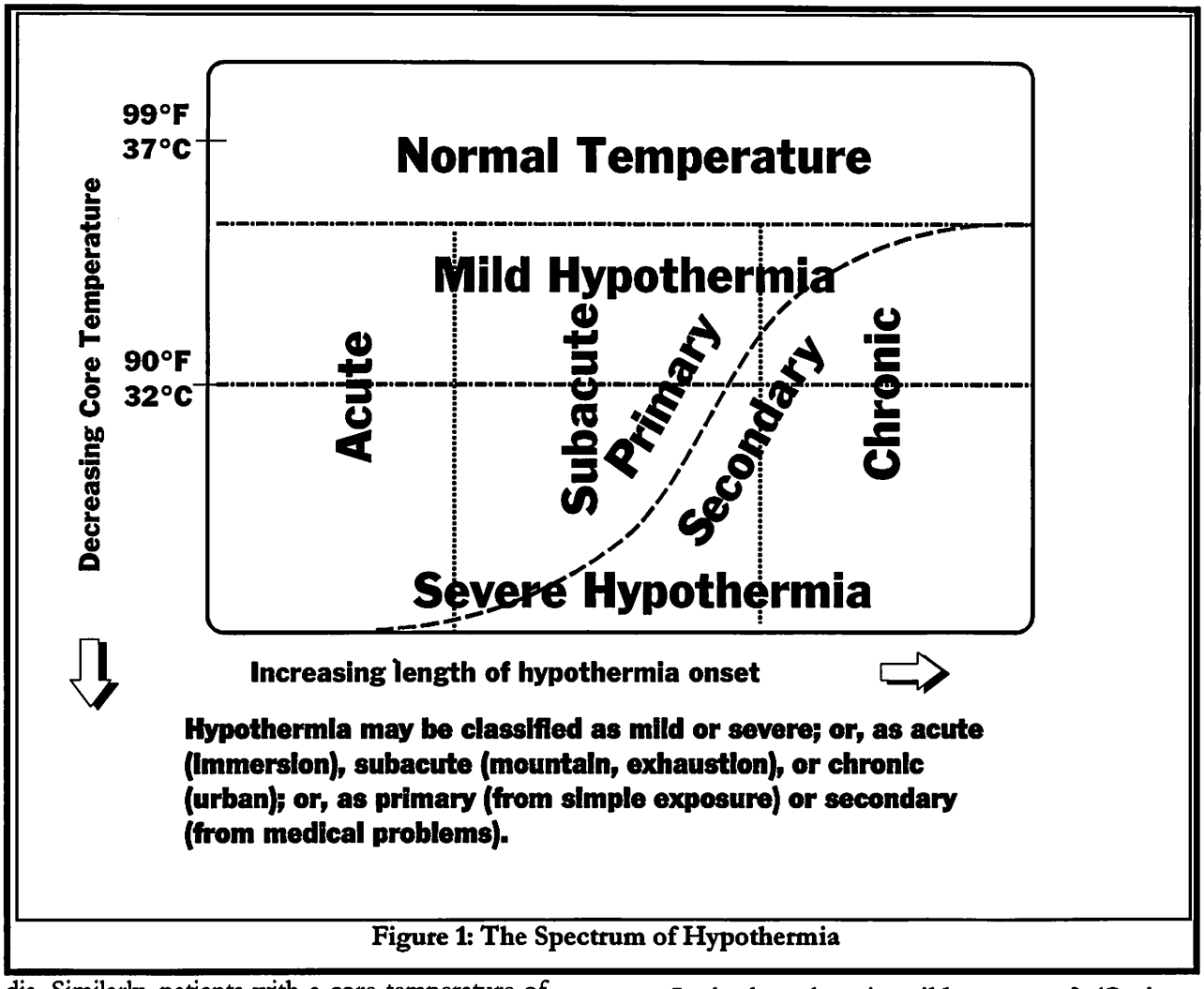

Figure 1: The Spectrum of Hypothermia

dia. Similarly, patients with a core temperature of 89 ° F (31° C) may need to be treated differently, due to their underlying medical problems, and depending on how fast they got down to 89° F (31° C). At present, the treatment differences are all at the hospital, rather than in the field. However, as we acquire new knowledge and new technology, field treatment of hypothermia may change.

For any particular hypothermia patient, you should be able to tell whether he or she has mild or severe hypothermia, whether it is primary or secondary, and whether it is acute, subacute, or chronic. This may not make any difference in how you provide heat to the patient (see below). But, it may help you understand how the patient is likely to do, to better assess the need for fluid replacement, and to know what other problems you might expect.

Is the hypothermia **mild** or **severe**? (Or **incipient**? See the section above on incipient hypothermia, under **diagnosis**.)

Is it **primary** or **secondary** hypothermia?

Is it **acute (immersion)** hypothermia, **subacute (mountain, exhaustion, exposure)** hypothermia, or is it **chronic (urban)** hypothermia?

Severity?

Hypothermia is **severe** when the core temperature is less than 90° F (32° C), otherwise it is **mild**. The figure of 90° (32° C) is somewhat arbitrary. However, it correlates well with the prognosis for primary hypothermia. Anyone without medical problems and with a core temperature of

90° F (32° C) or above will do well with just about any treatment.*

Primary vs. Secondary?

Hypothermia is **primary** when cold stress simply overwhelms normal compensatory mechanisms. Hypothermia may also be **secondary**, that is, caused by exposure to mild cold stress in the presence of intoxication or illness. Most urban hypothermia patients have at least a component of secondary hypothermia.

Acute, Subacute, or Chronic?

Acute Hypothermia — Hypothermia may come from sudden and overwhelming severe cold stress, as when a jet pilot ejects into the North Sea, or when you ski through the crust of a frozen pond. This is **immersion** or **acute** hypothermia. (Note, however, that a person who is lightly clothed but very active in the cold may maintain a comfortable core temperature, yet cool as rapidly as if immersed if he or she stops. For example, if an entrant in a cross-country ski marathon were hurt or stranded, he or she would cool as if immersed.[95] The pertinent physiologic features are:

- the fluid and electrolyte abnormalities are less severe than with subacute and chronic hypothermia;
- reserves of glycogen that are exhausted in subacute hypothermia (and probably in chronic hypothermia) are often still there (see the chapter on *Thermal Regulation* for a description of glycogen); and
- the prognosis is good for complete recovery, no matter which rewarming method you use. (But see the note about exertion above.)

Subacute Hypothermia — Another cause of primary hypothermia is **exhaustion**, often in the setting of a **mountain** trip. It is the result of several hours of exposure to cold, usually associated with strenuous hiking or other activity and the resultant exhaustion and fatigue. This is **subacute** hypothermia, also known as mountain or exhaustion hypothermia. Subacute hypothermia develops when an active outdoorsperson is exposed to a cold stress less severe than ice-water immersion, and occurs only after compensatory mechanisms fail, often over many hours.

Subacute hypothermia is characterized by glycogen depletion (exhaustion of energy stores) and possibly by adrenaline and cortisone depletion. (Adrenaline and cortisone are important hormones for the response to stress.) This depletion may make the victim unable to shiver.

One study purported to show that glycogen depletion didn't affect spontaneous rewarming rates. However, it seems unlikely that the study brought volunteers to the same state of total exhaustion, including total liver glycogen exhaustion, that is present in most cases of subacute ("exhaustion") hypothermia.[96]

Cold diuresis, intravascular volume depletion, and orthostatic hypotension are features of this kind of hypothermia.[97, 98] (Cold diuresis is discussed in the chapter on *Thermal Regulation*; orthostatic hypotension is discussed in the chapter on *Patient Assessment*.)

Subacute hypothermia has a better prognosis than chronic hypothermia, but worse than acute (immersion) hypothermia.

Chronic hypothermia, also known as "urban" hypothermia, occurs after mild or moderate cold exposure over days, and causes many abnormalities in body metabolism. It is often caused by mild cold stress along with a major predisposing problem such as diabetes or certain medications. Chronic hypothermia is a result of mild chronic cold stress in a person with an underlying deficiency of compensatory mechanisms. Many studies that purport to show that hypothermia has a very poor prognosis limited their study to chronic hypothermia.[85-88] Although chronic hypothermia patients are generally volume-depleted, they often have peripheral edema. The pertinent physiologic features are: severe fluid depletion; variable glycogen depletion, but with impaired glucose metabolism, resulting in ketosis (similar to diabetic ketoacidosis);[99] and poor prognosis that varies depending on the underlying medical cause for the chronic hypothermia.

"Mixed Hypothermia" — Patients lost in the wilderness may have hypothermia that combines more than one "type" of hypothermia. For instance, a patient with chronic hypothermia may get confused, wander away into the mountains, then develop superimposed acute hypothermia.

* A Jacuzzi followed by a cup of hot cocoa in front of a roaring fireplace is a particular favorite.

Immersion vs. Submersion — There is a very important distinction between victims of **immersion hypothermia** and victims of **cold-water submersion** (near-drowning). Don't confuse the two. In immersion hypothermia, the patient is cold but has sustained no brain damage: the treatment is to rewarm as soon as possible. In cold-water submersion, however, the primary problem is brain damage from lack of oxygen, and the patient's hypothermia is actually protecting the brain from additional damage; you **don't** want to rewarm the near-drowning victim in the field. Instead, maximize oxygenation and circulation and let the patient stay cold until you reach a medical facility.

Hypothermia, Fluid Balance, and the Heart

Fluid Loss

Hypothermic patients almost always have a below-normal volume in their blood vascular system. There seem to be two different processes that cause this. First, there is a shift of fluid from the blood vessels into other parts of the body. On rewarming, this fluid moves back into the blood vessels. Secondly, the kidneys remove fluid through the process of "cold diuresis," even if the blood volume is already low. This fluid is lost and does **not** reappear during rewarming. Wilderness patients may also have been dehydrated even before they become hypothermic. Dehydration is common in wilderness mishaps, whether from poor fluid intake or from respiratory losses due to cold winter air. One study noted the need for an average of 4,500 cc of fluid over 72 hours when rewarming severely hypothermic patients.[100]

One situation in which you might not want to start vigorous hydration is in a hypothermic patient who also has a severe head injury with a decreased level of consciousness. Another would be a cold water submersion (near-drowning) patient. In either case, giving lots of fluid might increase brain swelling and brain damage. However, in both cases, giving some dextran (see below) might help protect the brain.

Hypothermic patients are already very vasoconstricted, so MAST garments won't help the patient's circulation at all.[101]

Animal hypothermia studies explain this in more detail. As the body becomes hypothermic, fluid moves from peripheral blood vessels into those of the internal organs. The capillaries in these organs are more "leaky," and allow water to pass from the blood into the interstitial fluid.[102] This fluid returns quickly to the vascular system on rewarming. Depending on the degree of hypothermia, from 10% to 30% of blood volume will redistribute this way.[103] Cells of internal organs also take up water,[104] which is probably returned to the circulation on rewarming, but more slowly. Lloyd suggests that older people with chronic hypothermia may, with rewarming, have enough of this "edema fluid" return to cause fluid overload.[71] If you are rewarming an older patient with chronic hypothermia (unlikely in the wilderness but more likely in disasters), you may want to monitor for signs of fluid overload during rewarming.

As described in the chapter on ***Thermal Regulation****, cold causes the kidneys to excrete fluid, even when the patient is dehydrated ("cold diuresis"). The longer the patient is hypothermic, the more fluid is lost through the kidneys. One animal study showed that acutely hypothermic animals that were rewarmed slowly developed shock, while those that were rapidly rewarmed did not.[105] This may have been due to the longer time at cold temperature and thus increased fluid shift into cells or increased loss from cold diuresis.*

Fluid Replacement for Cold-Injured Patients*

Dextran — An ideal intravenous fluid for reversing the fluid shifts of hypothermia is low-molecular weight dextran (Dextran 40, e.g., Rheomacrodex®). Dextran is known to expand blood volume by pulling interstitial fluid back into the blood. In a study of hypothermic dogs, dextran did increase blood volume,[106] so it is reasonable to think it will work in hypothermic humans. Dextran will also likely help frostbite (see above). A given amount of dextran will expand volume more than the same amount of saline or Ringer's solution. Dextran is also thought to improve cerebral blood flow after brain injury, e.g., after head trauma or near-drowning. With the weight constraints of wilderness rescue, dextran thus seems an ideal first IV fluid for the cold-injured patient.†[103, 107]

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* for a discussion of IV fluid replacement in general, see the chapter on *Wilderness Trauma.*

† Elderly patients with chronic illness and chronic hypothermia (which is unlikely in wilderness search and rescue) should probably not receive dextran, for complex reasons discussed under the section on bypass rewarming, below.

Normal Saline — Wilderness patients are likely to have fluid losses as well as fluid shifts, both from pre-existing dehydration and from cold diuresis. Thus it is appropriate to follow dextran with normal saline ("NS").

Lactated Ringer's — Ringer's Lactate solution ("LR") contains lactate, which is metabolized in the liver. This provides patient with some energy. However, below about 30° C (86° F), the liver cannot metabolize lactate. Thus, there is no point in giving lactate to patients with deep hypothermia, at least until they warm to above 30° C (86° F).[103]

The Heart

In most cases, hypothermia does not affect the muscular pumping action of the heart.[108] However, there are isolated cases of decreased heart function due solely to cold, particularly in those who have been hypothermic for a long time (approximately 10-48 hours).[100, 109] When administering fluid to hypothermic patients, particularly those with chronic hypothermia or long-standing hypothermia, monitor carefully for signs of fluid overload (discussed in the chapter on *Wilderness Trauma).*

Basic CPR (BCLS) and Hypothermia

Severe hypothermia may be almost indistinguishable from death. We know that the patient who is severely hypothermic can have a pulse and respiration that are barely detectable, yet sufficient to maintain life without brain damage. As with any patient, you must follow the ABCs, but with some modifications.

"Some People Are Dead When They're Cold and Dead"

The standard dictum is that "nobody is dead until he's warm and dead." Clearly this is not true; there are many people who have a core temperature of about 70° F (21° C) or lower and are dead. (In the morgue.) But, how do you tell if someone is just cold, or cold and dead? At present, there is no good answer. You must assume that all cold bodies you find can potentially be resuscitated, unless there are reliable signs of death. And, you must remember that dilated pupils, rigor mortis, dependent lividity, and necrosis (frostbite) are **not** reliable signs of death in a very cold person. Even in a cold environment, however, you can reliably diagnose death by a mouth and nose that are plugged with ice, removal of the head from the body, massive head injuries with loss of large portions of brain matter, or major disfiguring injuries such as transection of the torso.

An article in JAMA suggests that hyperkalemia (high blood potassium) may be a marker for those who are both cold and dead.[110] The evidence is not yet compelling, because many of their subjects were buried in avalanches, which causes musculoskeletal trauma that may contribute to hyperkalemia.

Airway

With severely hypothermic patients, the muscles may be very stiff from the cold. As a result, the head-tilt method of opening the airway may be impossible because of a stiff neck. Pulling upward on the jaw is often a more effective way to provide an airway because you're pulling against a smaller group of muscles.

Breathing

At very cold temperatures, the metabolic rate is slow, and adequate respirations may not be detectable by looking, listening, or feeling. Sometimes, fogging of a mirror or piece of glass held at the mouth may show respiration. Determining the adequacy of respiration in severe hypothermia is controversial, but most experts now advise supplemental oxygen and artificial respiration if there is any question of inadequate respirations. Some experts advocate that you ventilate hypothermia patients at less than normal rates, to prevent alkalosis.[111] It used to be thought that alkalosis makes ventricular fibrillation more likely. However, we now have good evidence that increasing ventilation, and thus decreasing the amount of CO_2 in the blood, protects against ventricular fibrillation in hypothermic patients.[112-114] Therefore we recommend normal ventilation rate and volume, but no more than normal.

There is a controversy in the anaesthesia community about how to ventilate hypothermic patients (i.e., those with induced hypothermia for heart operations).[115] Some argue for increased carbon dioxide levels, arguing that the resulting increased CO_2 and acidosis causes

increased cerebral blood flow, and thus protects against the possibility of stroke during the operations. (Those who argue for this do not argue for decreased ventilation, however; they add CO2 to the inhaled gases rather than decreasing ventilation. Decreased ventilation may lead to atelectasis or hypoxia, they say.) However, the best evidence is that strokes during such operations are due to emboli from the operative site, and that increasing cerebral blood flow is of no benefit. The argument is compounded by questions as to what we should consider a "normal" blood gas for a hypothermic patient. Delaney provides a succinct overview of these controversial issues, and argues persuasively for normal ventilation.[116] *One study shows an increased incidence of ventricular fibrillation in dogs when their CO_2 levels fell precipitously. This might seem to favor slower rates for artificial respiration, but really only might argue for starting artificial respiration slowly, not continuing it slowly.*[117]

Oxygen is likely of benefit to any hypothermia patient. Give high-flow O_2, preferably warmed and humidified. (Oxygen toxicity is reportedly worse in patients who are hypothermic, but this would likely be a problem only for patients treated with high concentrations of O_2 for many days, or treated in a hyperbaric chamber.[118]

Circulation

Those with severe hypothermia may be so volume-depleted and vasoconstricted that you cannot detect peripheral pulses at all. The heart rate may be very slow (down to the 30s, or in one case, 6), but this is likely enough to supply the metabolic needs at these low temperatures.[119] Metabolic rate decreases rapidly with decreasing temperature (about 5% per degree C).[116] When severely hypothermic, the brain and heart are in a "metabolic icebox," and their need for perfusion is markedly decreased. There are well-documented cases of hypothermic patients surviving long periods of asystole when hypothermic.[120, 121] Therefore, for patients with **any** signs of life, the goal is to provide artificial respiration and to replenish fluid volume if an IV is available, but not to start external cardiac compression. Why not start external cardiac compression?

First, external cardiac compression can only provide a fraction of the normal cardiac output, and the patient is probably doing better than that on his or her own, even if the heart rate is very slow.

Second, a hypothermic patient with a barely-detectable pulse stands a fair chance of survival, but a hypothermic patient put into ventricular fibrillation by external cardiac compression has much less hope of survival.

Third, rescuers' fingers are often cold and therefore insensitive, making it difficult to find a pulse even in a normothermic patient.

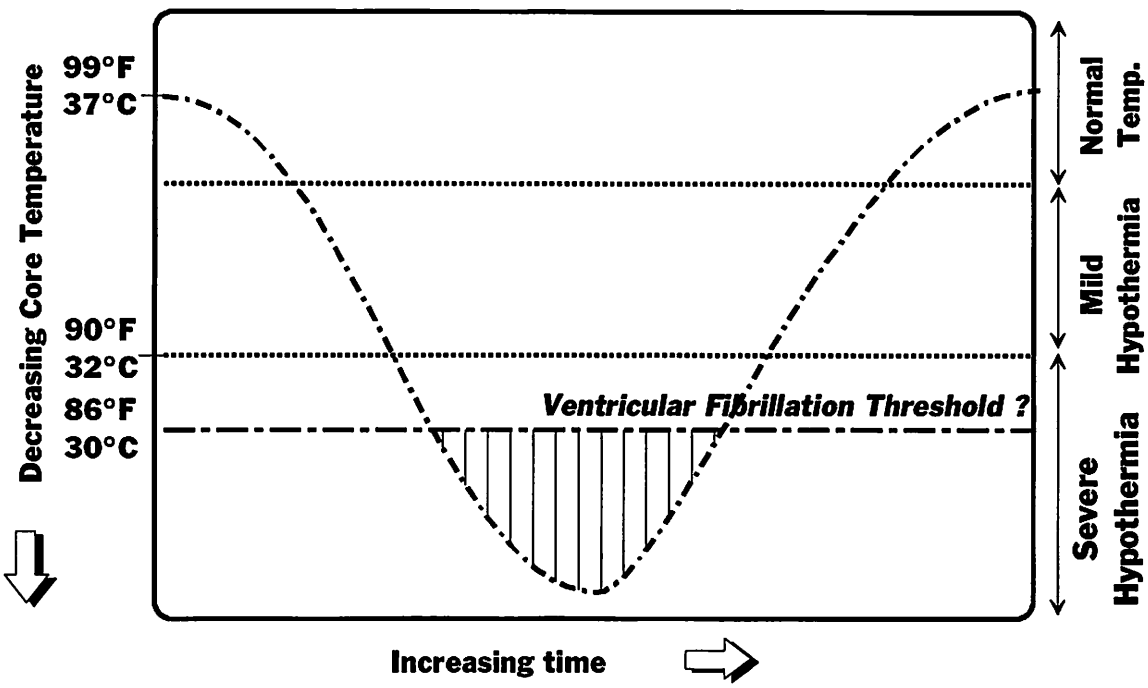

Figure 2: Temperature vs. Risk of Cardiac Arrest

Because of this, most experts recommend checking for up to three minutes for a pulse in hypothermic patients.* For hypothermic patients, the standard 4-6 minute rule for CPR ("a patient in cardiac arrest only has 4-6 minutes before brain damage occurs") does **not** apply: a severely hypothermic patient can tolerate long periods of full cardiopulmonary arrest without brain damage. You should use a stethoscope, if you have one, to listen to the heart, as well as palpating the carotid artery. You may want to hold a mirror to the nose to look for fogging, indicating spontaneous respirations.[122]

If there is any evidence of spontaneous pulse, heartbeat, respiration, movement, or any kind of organized rhythm on a monitor other than ventricular fibrillation or asystole, start ventilation (and IV fluids, if available) but **do not** start external cardiac compression. There is reason to think that external cardiac compression may cause ventricular fibrillation in a hypothermic patient, though no controlled studies are available.[123-129] One article quotes an unpublished experiment in dogs that shows no danger of ventricular fibrillation from external cardiac compression, but we are aware of no published evidence to support this contention.[130] Many studies on hypothermic dogs and pigs by the U.S. Army Research Institute of Environmental Medicine have shown that rolling the animal over or pressing on the chest will cause ventricular fibrillation, at least in unventilated animals. Animals which received artifi-

* The Heart Association's standards call for a one minute check for pulse in a hypothermic patient, but other recommendations call for longer checks.

cial ventilation were less sensitive to movement or chest pressure.*

If there are truly no signs of life at all, and you have no monitor, you must begin both artificial respiration and external cardiac compression.[130] Some have argued that there is no reason to start CPR at all on anyone with a core temperature of less than 28° C=82° F.[131] However, we agree with Leavitt and Podgorny[132] that, if there are truly no signs of life, you should start CPR. However, there are several practical reasons not to start CPR in the wilderness. To word this in the positive manner, start CPR in hypothermic patients in arrest unless:

- it places you at danger, or
- there are reliable signs of death (see above), or
- the chest is frozen so hard you cannot compress, or
- the mouth and nose are plugged with ice.

There is good evidence that patients with deep hypothermia may survive long periods of cardiorespiratory arrest without CPR, and may survive long transports with only intermittent CPR. Therefore, if you are evacuating a hypothermic patient in arrest, and you must stop CPR going through a narrow crawlway in a cave, or down a difficult pitch on a cliff, you should begin CPR again as soon as possible.

CPR Rate

Some authors recommend CPR at half the normal rate, either because of the slow metabolism of hypothermic patients, or because surgeons have observed hard hearts that are slow to fill when trying to resuscitate frozen patients.[89, 111, 133] CPR provides only about 30% of normal perfusion even if done perfectly in a normothermic patient. One study shows that external cardiac compression at normal rates is effective in severe hypothermia, though only providing about half of the blood flow of CPR on a normothermic patient.[134] And, since there is no solid evidence that slower rates will improve circulation, we recommend normal rates for both respiration and external cardiac compression. Normal CPR rates are also supported in the literature.[83, 130] (Also see the comments about CO_2 levels and artificial respiration under *Breathing*, above.) However, in deeply hypothermic patients, you may find that the chest is so "stiff" that you are unable to perform external cardiac compression at normal rates. Both compression and spontaneous re-expansion of the chest may be slowed. In such cases, you may have no choice but to compress at a slower rate.

Advanced Cardiac Life Support (ACLS) and Hypothermia

Airway

Victims of severe hypothermia may be so stiff that you can't hyperextend the neck; positioning the torso and limbs may be difficult for the same reason. The digital intubation technique, possibly with a lighted stylet, may be useful in such a situation.[135, 136] (See the chapter on *Introduction to/Review of Advanced Techniques* for more on these methods of intubation.) In at least one unpublished case (April, 1990, STAT helicopter service, Pittsburgh, PA) paramedics could not intubate a patient with a core temperature of 70° F (21° C) until they gave her midazolam (Versed®): a very short-acting muscle-relaxing benzodiazepine similar to diazepam (e.g., Valium®).

You might think that intubating a hypothermic patient could provoke ventricular fibrillation. There are indeed isolated case reports of hypothermic patients going into ventricular fibrillation during endotracheal intubation.[86, 137] One major paramedic text flatly recommends against intubating hypothermic patients.[138] However, several large studies show that endotracheal intubation is **not** likely to send a hypothermic patient into ventricular fibrillation.[88, 139] One large animal study shows no association between intubation and ventricular fibrillation in hypothermic animals. (As expected, it did show danger of spontaneous ventricular fibrillation from hypothermia).[140] The patient must have an adequate airway to survive. If the airway is inadequate and an endotracheal tube will improve it, and you have the equipment and training, you must intubate. The bottom line is that **the criteria for intubating a patient are**

* Information from Murray Hamlet, D.V.M.

the same whether the patient is normothermic or hypothermic.

Ventricular Fibrillation, Defibrillation, and Hypothermia

The available scientific evidence seems to show that direct stimulation of the heart, as from an intracardiac catheter, causes ventricular fibrillation. One dog study shows that simply rolling the patient over, endotracheal intubation, central IVs that don't reach the heart, and placing NG tubes don't actually cause ventricular fibrillation. However, this same study showed a significant risk of spontaneous ventricular fibrillation simply from the cold.[141] *One suspects that many case reports of ventricular fibrillation "caused" by various stimuli are coincidences. External cardiac compression, however, is a direct stimulus to the heart, and was not tested in this study. See also the discussion in the* ***Circulation*** *portion of* ***Basic Cardiac Life Support****, above.*

Treatment of Hypothermic Ventricular Fibrillation — The tremor associated with deep hypothermia can easily be mistaken for ventricular fibrillation on a monitor, so even if the monitor appears to show ventricular fibrillation, you should make a careful one to three-minute check for pulse, heartbeat, or breathing before starting CPR.

Many wilderness rescue teams do not even carry defibrillators; they are heavy and seldom effective for wilderness patients. If a team has medications but no defibrillator, a trial "chemical defibrillation" can be attempted for patients in ventricular fibrillation; see below. For hypothermic patients, there is no evidence that electrical defibrillation is any more effective than chemical defibrillation, so from a weight-to-effectiveness viewpoint, a bottle of bretylium is far more efficient than a defibrillator.

In a true wilderness setting, hypothermic patients in ventricular fibrillation will probably not survive. Exceptions might be if they can be promptly evacuated by helicopter directly from the scene to a hospital facility, or if you can rewarm the patient and do a successful cardiac resuscitation at the scene (unlikely). However, see the comments on survival after prolonged CPR under BCLS, above.

You cannot, as a rule, defibrillate a hypothermic patient successfully until the patient is rewarmed. With a severely hypothermic patient in ventricular fibrillation, you must generally continue basic CPR until the patient can be rewarmed.

The treatment of ventricular fibrillation depends on the core temperature.

If the patient's core temperature is 86° F (30° C) or warmer, you may use standard ACLS protocols.

If the patient's core temperature is colder than 86° F (30° C), ACLS drugs (except bretylium) are unlikely to be of any benefit, and may complicate later resuscitation efforts.

Chemical Defibrillation — Drugs may be given as an adjunct to electrical defibrillation, or by themselves to cause "chemical defibrillation."

Given that recent findings suggest that lidocaine may actually make it **more** difficult to defibrillate, giving bretylium seems the better choice (see below for more about bretylium).[142, 143] Procainamide (e.g., Pronestyl®) will make ventricular fibrillation more likely in hypothermic patients, so **do not give procainamide** to treat hypothermic ventricular fibrillation.[144]

Magnesium has also been suggested for defibrillation;[145] one study that showed an excellent effect simply from the magnesium without electrical defibrillation, the dosage was 0.1g/kg (or about 7 grams for a normal-sized adult).*[146] We could find no more recent data on magnesium and hypothermic ventricular fibrillation.

Some prehospital hypothermic arrest protocols call for lidocaine or bretylium (or no drug) and one to three defibrillation attempts.[147, 148] Since it is possible (though unlikely) that defibrillation might work, this seems a reasonable approach, but you must follow the standing orders established by your medical director, or request medical command from a Wilderness Command Physician.

Some anecdotal reports point to the efficacy of bretylium in the hypothermic arrest as described below. Although protocols for dealing with this situation is the province of the team's Operational Medical Director, the following is a fairly standard protocol:

- Defibrillate once with 200 Joules. If no response,

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* Maclean, apparently erroneously, gives the dose as 0.1 mg/kg.

- Give bretylium IV (5 mg/kg, or 350 mg for an average 70 kg patient). Start CPR and continue for 10 minutes, then:
- Defibrillate again, with 300 Joules; if no response:
- Repeat the dose of bretylium and circulate it with another 10 minutes of CPR, then:
- Defibrillate again with 360 Joules.
- If you are unsuccessful, and CPR cannot be continued throughout the evacuation, continue CPR for up to another 30 minutes. If, after this effort, you still cannot resuscitate the patient, and medical command is still out of touch, terminate resuscitation or continue CPR and evacuation as per your standing orders.

Other Arrhythmias

Deeply hypothermic patients often are in sinus bradycardia, atrial fibrillation, atrial flutter, or various types of atrioventricular block, and may have frequent premature ventricular contractions ("PVCs"). All of these "benign" arrhythmias get better as the patient warms.[85, 149-151] Atropine is ineffective in hypothermia, and using a pacemaker for the "normal" bradycardia of hypothermia actually decreases cardiac output.[145]

ACLS Medications and Hypothermia

Experience has shown that almost all medications administered during routine prehospital cardiac resuscitation are ineffective in hypothermic patients at the time they are administered.[151] However, as the patient is rewarmed, all the medications finally start having their effects. Therefore, don't give routine ACLS drugs to severely hypothermic patients. In particular, bradycardia, with heart rates as low as 20, is expected in severe hypothermia. In a severely hypothermic patient, you should not treat such bradycardia, even with a low blood pressure, unless it fails to respond to rewarming. Expect a variety of transient arrhythmias or heart blocks, but don't be eager to treat them in a hypothermic patient. Most of these arrhythmias and blocks improve with rewarming. The only standard ACLS medication that is likely to help a hypothermic patient is oxygen.[129] If low blood pressure doesn't respond to fluids, an infusion of dopamine may be of some benefit.[145]

Bretylium to Prevent Ventricular Fibrillation

Bretylium may be effective in both preventing and treating ventricular fibrillation in the hypothermic patient. The evidence consists of two case reports of a hypothermic patient who converted from ventricular fibrillation after IV bretylium,[152, 153] and four animal studies that show it effective for preventing ventricular fibrillation.[141, 154-156] Two of these studies also showed that bretylium makes it easier to defibrillate the hypothermic heart.[141, 154] There is no indication that lidocaine is effective. One paper argues against the need for prophylactic antiarrhythmic medications.[85] However, this recommendation applied only to in-hospital rewarming, not during rescue. Dr. Dan Danzl, a well-known authority on hypothermia, recommends prophylactic prehospital bretylium for hypothermia patients.[111] Still, bretylium is controversial, and orders for bretylium will be made by your Medical Director or an online Wilderness Command Physician. Procainamide (e.g., Pronestyl®) will make ventricular fibrillation more likely in hypothermic patients, so **do not give procainamide** to prevent hypothermic ventricular fibrillation.[144]

Bypass Rewarming

When making evacuation and transportation plans, consider the following. Hypothermic patients with little or no detectable vital signs will do best at a facility that can perform cardiopulmonary bypass or a similar form of rewarming. Regardless, please alert the Emergency Department that will receive the patient as soon as possible. Advance preparation may allow them to call in personnel needed for rewarming and resuscitation.

There are many reports of severely hypothermic patients, some with no detectable vital signs, successfully rewarmed by full cardiopulmonary bypass.[111, 157]

Various easier alternatives to cardiopulmonary bypass have been proposed. Some, such as thoracotomy with warm mediastinal irrigation and internal cardiac massage,[158] or continuous warm lavage via chest tubes,[159-161] should be available at any general hospital Emergency Department. Others, such as continuous arteriovenous rewarming without the full cardiac bypass, possibly removing the need for heparin anticoagulation, require special equipment.[162] Lack of

anticoagulation means this last method may also be useful, to prevent bleeding for patients with both hypothermia and trauma.[163]

Hypothermia is known to cause an anticoagulant effect and a decrease in platelets, both reversible.[164-170] *This is thought to worsen bleeding in cases of trauma; the main treatment is rewarming.*[171] *However, severe hypothermia is also known to cause disseminated intravascular coagulation, (small clots throughout the body) which may be unresponsive to heparin, the usual treatment for "DIC."* [172] *Heparin and dextran are also thought to cause problems with polymerization of cryofibrinogens in some elderly patients with chronic hypothermia.*[145]

Cryofibrinogen is not to be confused with the less-common cryoglobulin: "Cryofibrinogen is a cold-precipitable fibrinogen that seems to formed by the partial action of thrombin. Most normal plasma contains only traces of cryofibrinogen but excess amounts may be found in many clinical disorders, for example, infection, especially pneumonia or infections of the urinary tract by coliform organisms, thrombosis, neoplasia, metabolic disturbances, and connective tissue disorders."[150] *One study found about half of elderly patients with chronic illness and hypothermia had elevated cryofibrinogen.*[173] *DIC is discussed in the chapter on* ***Wilderness Trauma.***

Rewarming vs. "Adding Heat"

Adding Heat, Active Insulation, and Thermal Stabilization

Some EMTs have been told "don't rewarm in the field." So, for example, they will not put heat packs on a hypothermic wilderness patient, because that would be "rewarming the patient." This "don't rewarm in the field" rule has led to ridiculous behavior. An apocryphal (we hope) story illustrates. On one winter rescue, rescuers didn't place any insulation around their deeply hypothermic patient. They were afraid of the dangers of "rewarming in the field." This is horse hockey. Or whatever expletive you prefer.

We assume you will wrap your ill, injured, or exhausted patient in thick insulation. However, even the thickest closed-cell foam pad and artificial-fiber sleeping bag allow some heat to escape. And even the best field rewarming methods combined (a charcoal vest plus warm humidified O_2 and warm IVs) will only add a small amount of heat. The danger of a patient becoming more hypothermic during evacuation is real.

There is a good way for you to counteract this mistaken idea: that you should never apply any rewarming methods in the field. Tell people that it's OK to **add heat**; adding heat is **not** necessarily rewarming. Just because you are adding heat (e.g., heat packs, warm IVs, warm O_2 or air), it doesn't mean that you're rewarming the patient. Especially in a cold environment, the patient will still be losing heat, no matter how well you try to insulate. Therefore, you have to add heat, in effect instituting **active insulation**, offsetting unavoidable losses. Do your best to popularize the terms "adding heat," "active insulation,"[174] and "thermal stabilization."

Avoid Rapid Field Rewarming?

Even if you accept that you can add heat without rewarming, a hot topic among EMTs is still: "Do we start rewarming in the field or not?" Many EMTs have heard that rapid rewarming can cause shock or sudden death in some hypothermia patients. (See below for more on rewarming shock.) They've also heard doctors say "don't rewarm rapidly in the field." EMT textbooks warn against the dangers of rapid rewarming in the field.[175] But, there **is** no way to rapidly rewarm someone in the field! Even using all our field rewarming methods at once, we cannot add enough heat to rapidly rewarm. Unless you happen to have a folding hot tub or portable cardiopulmonary bypass machine in your pack. The best you can do in the field is to slowly rewarm someone, and that only if you have excellent insulation, and if: (a) the person has plenty of energy reserves and can shiver, or (b) you use several rewarming methods (i.e., a charcoal vest and warm humidified air).

Avoid Slow Field Rewarming, Too?

Others worry about supposed dangers of slow external rewarming. Some would even transport without insulation, to preserve the "metabolic icebox" effect and "save" the patient for later core rewarming. However, in the opinion of hypothermia experts, dangers of prolonged hypothermia outweigh dangers of slow field rewarming.* By rewarming slowly, you decrease the danger of spontaneous ventricular fibrillation during

* This was the consensus of participants in a workshop entitled "Controversies in Wilderness Medicine: Hypothermia" at the First World Congress on Wilderness Medicine, Whistler, British Columbia, July 1991.

evacuation. (See below for more about afterdrop and rewarming shock.)

Rewarming Recommendations

We use the term "hypothermia" to include a person who is slightly cold on a day-hike, and also someone who has been buried for days in a collapsed snow cave. Sometimes, when a team member on a winter search is briefly soaked in a stream, he or she needs brief rewarming in the field but doesn't need to be taken to a hospital. On the other hand, some hypothermic patients clearly need urgent evacuation: consider the recent (1986) cases on Oregon's Mount Hood, where multiple patients (some severely hypothermic and others, unfortunately, frozen solid) were insulated and evacuated by helicopter to multiple hospitals for cardio-pulmonary bypass rewarming. Some survived.[111]

Because circumstances vary so widely, decisions about rewarming must be made by you at the scene, and if possible, after you consult with your Wilderness Command Physician. However, we will present some general guidelines that may help in making this decision. (Specific rewarming techniques are discussed later.)

If confronted with a healthy person with incipient or mild (higher than 90° F=32° C) acute or subacute primary hypothermia, you should stop and rewarm as soon as possible. Any method is safe, including rapid rewarming, if you're now out of the wilderness. When the person is rewarmed, rehydrated, rested, and had time to digest an adequate meal, he or she can probably walk back to base, with an escort, but with no further medical attention.

If you are confronted with a patient with any other form of hypothermia, you should transport gently and urgently, adding as much heat as you can during transport. Do your best to get fluids into the patient. (See the *Principles of General Medicine* chapter for guidelines for using oral fluids.) Available methods to add heat during a wilderness evacuation, at present, add only enough heat to keep the patient's temperature stable, or to rewarm very slowly.

What if you have patients with chronic or severe hypothermia, cannot start transport to a medical facility, and have facilities such as a bathtub and hot water? (For example, during a cold-weather disaster, or if snowed in at a mountain lodge.) Your best bet is to talk to a trained Wilderness Command Physician. If you cannot contact a Wilderness Command Physician, you should decide whether you should rewarm the patient rapidly or slowly. Based on the incomplete information available to us, we would recommend you use rapid hot bath rewarming for healthy patients with severe, acute, primary hypothermia. (For example, such as from cold water immersion, or from being thrust into a snowstorm when a plane crashes.) You can use rapid hot bath rewarming for severe subacute primary hypothermia, too, but only after rehydrating the patient with a liter or two of oral or IV fluid to prevent rewarming shock. Patients with secondary hypothermia, or chronic hypothermia, however, you should rewarm slowly, and again must take care to replete their fluids during rewarming. You could potentially use a bath for slow rewarming; simply use water that is at or slightly below normal body temperature (99° F=37° C). Please also read the sections below that describe the dangers of rapid rewarming of hypothermia patients.

Should you ever delay evacuation to rewarm a patient? Since field rewarming methods only rewarm slowly, if at all, the answer is generally no. However, if you are facing an evacuation that will require lifting the patient in a vertical position (e.g., raising out of a narrow 200' pit in a cave), you might delay long enough to get 1 or 2 liters of fluid into the patient. Should you delay warming until the patient is up the pit? Theoretically, external rewarming can cause some vasodilation. However, the amount of heat you can add will cause little vasodilation, so you should not delay rewarming. Warm inspired air or O_2 would be ideal for such a situation, because it should cause little vasodilation.

When treating any hypothermic wilderness patient, remember to **give fluids** because the patient is volume depleted, and to **give food calories** because the patient is likely glycogen depleted. In a mildly hypothermic team member, this may be hot cocoa and gorp* or candy. For a severely hy-

* **Gorp:** A word of uncertain derivation. Used in the outdoor community to refer to small bits of various foods mixed together, most often raisins, peanuts, and chocolate. This mix-

pothermic patient, it means an IV with salt and glucose (e.g., D_5NS, D_5LR; see the chapter on *Wilderness Trauma* for more about the types of IV solutions).

If the patient has hypothermia severe enough to mimic death, or has other severe complications, think about evacuating to a center that can perform cardiopulmonary bypass rewarming. Add heat during transport, and perform CPR as needed (see above).

For severely hypothermic patients who will not be transferred for cardiopulmonary bypass, the Emergency Department should use core rewarming instead of peripheral rewarming. Of the available methods, peritoneal lavage seems the best choice, provided the staff are familiar with the technique.[176] *For patients in cardiac arrest who cannot be transferred to a facility with cardiopulmonary bypass, there is one apparently superior alternative. Open the chest, and use direct irrigation of the mediastinum with internal cardiac massage. External CPR combined with peritoneal dialysis is an alternative.*[177]

Rewarming Terminology

"Active" vs. "Passive"

Rewarming is sometimes classified into "passive" and "active" rewarming. **Active rewarming** refers to adding enough heat to make the core temperature rise. **"Passive rewarming"** means insulating the patient and allowing the patient's basal metabolic rate and shivering to bring up the core temperature. For this to succeed, the patient must have adequate energy reserves to generate the requisite heat (questionable in subacute or chronic hypothermia) and must be insulated well enough to eliminate all significant losses. This includes stopping losses from respiration. (Of course, insulating a patient is a lot easier in a 72° F hospital than in a -20° F blizzard). One study showed that many young, healthy Coast Guard midshipmen did not do a good job of rewarming themselves from even mild acute hypothermia, so this method has questionable merit.[178] However, it has the advantage of minimizing sudden changes in the patient's condition that might precipitate a problem such as ventricular fibrillation. Therefore some authors recommended it for in-hospital use for patients with chronic hypothermia, but it has generally been replaced by more aggressive methods such as cardiopulmonary bypass.

Surface vs. Core Rewarming

Active rewarming methods are generally classed as "core" if they rewarm the body core before the skin and limbs. Core rewarming is generally thought to be superior, because it minimizes afterdrop and rewarming shock (see below).

Rewarming Complications

Three problems are associated with rewarming: **afterdrop**, **rewarming shock**, and **sudden death**.

Afterdrop

Afterdrop refers to the common finding that core temperatures continue to go down after rewarming starts, particularly with rapid peripheral rewarming (a hot tub). This is thought to increase the risk for complications, including ventricular fibrillation. However, it is important to note that afterdrop is not likely when "adding heat" to a wilderness patient. Such slow external rewarming is much more like so-called "passive rewarming," in which people are placed in a warm room and insulated. Such patients do not show afterdrop.[179]

With peripheral rewarming in particular, some authors attribute afterdrop to a local reflex in the limbs. Local skin sensors detect the warmth of the external rewarming, and this causes local vasodilation. This results in a sudden rush of cold, stagnant blood from the extremities to the core. This influx has been shown in experiments on humans, but appears to be only a small amount of flow.[180, 181] Because this blood may be acidotic and full of toxic metabolic products, it is thought

ture is eaten by the handful during strenuous exercise such as hiking and climbing. Gorp provides an easily-digested mixture that can be eaten in any arbitrary amount (as opposed to, for instance, a candy bar). It provides glucose for immediate energy (in the raisins), fat (in the chocolate and peanuts) for a sustained energy yield, and some protein (peanuts) to make up for muscle injury from strenuous exercise. Some insist that "true gorp" consists of one part peanuts, one part raisins, and one part coated chocolate bits. Purists insist that the apportionment be by number of bits rather than weight, and decry the addition of bits of Swiss cheese.

to cause even more of a risk for ventricular fibrillation than its temperature alone would suggest.

However, others suggest that vasoconstriction in severe hypothermia is so intense that there is very little blood in the extremities to be shunted to the core, and that the physics of heat transfer show that external rewarming must result in afterdrop even if there is **no** reflex vasodilation. Researchers have shown afterdrop in hypothermic watermelons that are being rewarmed, in large bags of Jell-O, and in a leg of beef (even though this can be predicted from a very basic knowledge of the physics of heat transfer.)[76] The truth seems to be that a combination of the two effects causes afterdrop.

Regardless of the amount of blood pooled in hypothermic extremities, increased blood flow in the extremities will cause rewarming of the extremities at the cost of the critical core regions. Also, core rewarming, even of a watermelon, will decrease afterdrop and increase temperature rise in the core. Therefore, core rewarming is the ideal.

An assumption by many is that afterdrop during rewarming is somehow a cause of sudden death, particularly by ventricular fibrillation. However, there is good reason to suspect that the danger of fibrillation is a simple function of how cold the heart is, and how long it stays that cold. In this view, there is nothing "magical" about an afterdrop of a degree or so; the heart was likely to fibrillate even without the slight extra cooling of the "afterdrop." If it comes to a choice of slow rewarming with an afterdrop versus having the patient stay hypothermic through a long evacuation, the choice is clear: rewarm.[180]

Rewarming Shock

As severely hypothermic patients are rewarmed, the blood pressure, pulse, and respiratory rate generally start at low levels and gradually increase. Sometimes, the pulse gets very fast while the blood pressure goes back down, and the patient shows sign of diminished cerebral perfusion. You should interpret this as mild shock caused by the rewarming process. You will see it particularly when using rapid external rewarming, such as immersion in a hot bath (you may even see it when rewarming people with incipient hypothermia). Consider that all hypothermic patients are volume depleted and vasoconstricted, and you are applying external heat that is known to cause local reflex vasodilation. Thus, **relative hypovolemia** (a small amount of blood in a vascular system that used to be small, but now is big) is an obvious source of shock. To avoid rewarming shock, you could vigorously hydrate with warm IV solutions, slow the rewarming rate, or use core rewarming techniques.

Sudden Death

Sudden death during hot bath rewarming was reported as early as A.D. 320: Forty Christians were exposed to cold for three days, with a warm bath waiting if they would give up their religion; one who gave up and was rewarmed in the bath suddenly died.* Sudden death during rewarming may be from rewarming shock or from ventricular fibrillation, but there is no good evidence pointing to any one particular cause. One paper is widely cited as supporting the idea that rapid rewarming will cause temperature differences inside the heart that lead to ventricular fibrillation.[182] However, on careful reading, the paper has serious defects, and does not truly show that these temperature differences lead to ventricular fibrillation, nor that rapid rewarming will lead to ventricular fibrillation. More recent evidence, including research cited in the section on ventricular fibrillation above, suggests that the chance of ventricular fibrillation is a simple function of the depth of hypothermia and the length of time at that temperature. If we exclude deaths from rewarming shock, which can be treated with IV fluids, then the chance of ventricular fibrillation during rewarming is no more than the chances during hypothermia itself. If this is true, then rapid rewarming with fluid repletion may be no more dangerous than letting patients stay hypothermic. More research is needed, but for now, reasonable ways to minimize the chances of sudden death include the following: warm IV solutions to prevent rewarming shock, core rewarming to prevent afterdrop, and prophylactic bretylium to prevent ventricular fibrillation.

Though not truly a complication of rewarming, many hypothermic patients, especially those with chronic hypothermia, develop pneumonia (or

* *Butler's Lives of the Saints for March 10: The Forty Martyrs of Sebastea.*

perhaps it caused the hypothermia). If your hypothermic patient faces a very long evacuation, and you find evidence of pneumonia on exam, you may wish to administer an antibiotic. Of the oral antibiotics commonly carried in a wilderness medical kit, amoxicillin-clavulanate (e.g., Augmentin®) has been recommended for this use.[145]

Field Rewarming Methods

The following methods are available to you for field rewarming:

Sleeping Bag

For mild or incipient hypothermia among team or hiking party members, standard advice is:

- get out of wind and rain;
- set up a tent or make other shelter;
- help him or her change into dry clothes;
- give hot sugared drinks; and
- place the patient in a sleeping bag alone or with another (presumably warm) person. However, experiments in immersion hypothermia (in which glycogen stores are better than in "mountain" hypothermia) show that many healthy young men cannot effectively rewarm themselves by shivering alone, so another body in the sleeping bag is always appropriate.[178]

Warm Inspired Air or Oxygen

This method adds heat directly to the core. In a cold environment, it eliminates a major source of continued heat loss (respiration). (Since most of the heat carried by air or oxygen is carried by its water vapor, the gas must be humidified to carry any significant heat.) One disadvantage of warm inspired air or O_2 is that, even with humidification, it delivers only a small amount of heat compared with hospital-based methods of core rewarming.

Some warm humidified air/oxygen systems are also heavy and bulky. Although several developers made lightweight systems that use dry chemicals to generate warm humidified oxygen, none of these developers are still in business, and the systems are no longer available. A variety of **soda-lime** systems are available; they use the reaction of carbon dioxide with soda-lime powder to heat and humidify air or oxygen passing through them. There is no good evidence that oxygen is beneficial in severe hypothermia, so warm, humidified air is probably as effective as warm, humidified oxygen. In view of the great weight of oxygen cylinders, air is probably the best choice for most wilderness rescue teams until lightweight solid-state devices that provide warm, humidified oxygen are available.

One recent study compared a charcoal vest (Heat-Pac®, AB Russell) with a particular model of inhalation device (Heat-Treat®, Thermogenesis, Victoria, British Columbia). The experimenters found many safety problems with the Heat-Treat device: "...explosive ignition of the propane canister burned the arms of three operators; the unit became too hot to handle and melted the plastic flooring; difficult-to-control inhalation temperature resulted in a mild first-degree burn to the face in one subject; and high dyspnea was noted by all eight subjects due to water from condensation restricting air flow in the breathing tubes."[183] The manufacturer of the Heat-Treat device has discontinued its production, and now makes several models that are reportedly technically much improved.*

One study showed decreased heat production in patients rewarmed with warm inhalation.[184] However, this only studied mild hypothermia, and in patients who presumably still had glycogen stores. Hypothermia patients who are glycogen-exhausted will likely have little of their own heat production to suppress. Therefore this is not an argument against using inhalation to add heat to severely hypothermic patients.

Warm IV Solutions

Provided you can start an IV, give solutions with glucose to all severely hypothermic patients†.<$

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* FRES-Q Products, Inc., P.O. Box 661, Quathiaski Cove, BC V0P 1N0 Canada; (604) 285-2890

† Some patients with hypothermia will have high blood sugar. As you are taught about diabetics, adding some glucose will not make a hypothermic patient with high blood sugar much,

Intra-osseous infusion is now rapidly gaining acceptance as a method of emergency pediatric IV access for Emergency Department and prehospital use, as taught in the American Heart Association-American Academy of Pediatrics *Pediatric Advanced Life Support* course. This technique is simple and reliable, and might be useful for obtaining an equivalent of IV access in hypothermic adults. However, we are aware of no controlled studies of the technique in hypothermic adults.

Some simple physical calculations based on the maximum temperature of an IV (104° F=40° C; higher temperatures will cause burns) and the amount that can be given shows that warm IVs do little to rewarm a patient. However, warm IVs should be used to prevent cooling the patient with cold IV fluids. Hydration should be vigorous except for patients with medical complications or chronic hypothermia.

For wilderness rescue, central IV lines are much more secure than peripheral IVs. There may be some danger that central lines long enough to reach the heart, such as are used in intensive care units, might initiate ventricular fibrillation. Short central lines, as might be used in wilderness rescue, should not cause any direct heart irritation, and therefore seem unlikely to cause ventricular fibrillation. Wilderness EMT-Ps should go by their standing orders or medical command when attempting to obtain IV access in a hypothermic patient.

You may warm IV solutions by taping IV bags between two heat packs and wrapping them in closed-cell foam or other insulation, and extra IV tubing (as with a blood-warming IV set) may be wrapped around a heat pack and wrapped in insulation, then inserted between the IV bag and the patient. In the winter, you may clip IV bags to a single-length sling over the shoulder; if worn well up in the armpit, under the outer clothing, the bag should stay warm during the hike in to the patient. If a litter team member must carry an IV bag under his or her arm, you may run an extra length of IV tubing through the arm of the litter team member's parka. Keeping the bag in the litter, with pressure from the patient's body on top of it, or from a blood pump or blood pressure cuff, and with heat packs as described as above, is a much neater method, however.[185] Even in a controlled room temperature, even short lengths of uninsulated IV tubing causes IV fluids to cool rapidly.[186] You must insulate all IV tubing if you expect the fluid to still be warm when it reaches the patient.

Hypothermic patients are generally both water and salt depleted, as discussed under *Hypothermia, Fluid Balance, and the Heart*, above. Many of those found by search and rescue teams are also glycogen depleted. D_5NS is an appropriate choice for intravenous fluid. (The chapter on *Wilderness Trauma* discusses types of intravenous fluids in detail.)

In the Emergency Department, IV bags may be warmed in a microwave.[187-192]

Heat Packs

Heat packs add only a small amount of heat, but can be useful when combined with other methods. To make heat packs more of a core rewarming method, you should place packs at six areas of high heat exchange with the core: both sides of the neck, both armpits, and both groins. (In these areas, large blood vessels are near the surface.) Heat packs may cause burns if hotter than 110° F (43° C); you should always wrap heat packs in a towel, spare T-shirt, or something similar, to avoid skin burns.[16] The various types of commercial heat packs have develop different maximum temperatures, and stay warm for different lengths of time.

Hydraulic Sarong

This device, developed by Washington State mountain rescue teams, is a mass of rubber tubing embedded in a blanket you may wrap around a victim; you may then fire up a stove, heat water, and then pump hot water through the tubing. The hydraulic sarong is an active external device that provides more heat than heat packs, but not nearly so much as a hot bath. Although rescuers may use it to rewarm a patient before transport, it is hard to use during the evacuation. (A lit stove with a pot of hot water on it is difficult to manage during a semi-technical evacuation). The hydraulic sarong rewarms slowly enough that rewarming shock will probably not be a hazard. Unlike hydraulic hospital warming blankets that warm the

if any, worse. However, if the patient is glycogen depleted and hypoglycemic, some IV glucose may increase heat production.

extremities as well as the torso, the hydraulic sarong is designed to apply just to the torso.[193, 194]

Charcoal Vest (e.g., Heat-Pac®)

Also sometimes known as a **charcoal sarong**, this device, developed in Scandinavia, uses a fan-driven charcoal warmer to heat a wrap-around vest. The advantages over the hydraulic sarong are that it can be used during transportation and that it is lighter. It gives off carbon dioxide and small amounts of carbon monoxide, and this could theoretically be a danger in caves, mines, or poorly ventilated tents. To minimize the amount of carbon monoxide given off, it is vital that the exhaust tube is properly attached and not obstructed, that the battery running the fan is fresh and inserted in the proper direction, and that the fan is functioning properly. You may want to permanently connect the exhaust tube to prevent detachments and carbon monoxide accumulation in the patient's sleeping bag or other litter packaging. Lighting the charcoal insert may release a significant amount of carbon monoxide, so light it outside or in a well-ventilated area.[183] Unlike the hydraulic sarong, the charcoal vest can be used continuously in a litter patient. If used with proper cautions, it seems to be, at present, the best easily-portable field rewarming device.*

Warm Water Immersion

Warm water immersion is not a wilderness rewarming method. We describe it here for two reasons. First, you might find yourself in a situation where rapid warm bath rewarming is available, yet you cannot transport to a medical facility (e.g., near a hot spring). Second, our current recommendation for all wilderness hypothermia patients is that you add as much heat as possible during evacuation. This is only because we don't have any method that **will** rewarm rapidly in the field. But what if some method for rapid rewarming during wilderness evacuations should appear in the future? If so, you should probably use it according to the principles of warm bath rewarming described here.

*Some have noted that application of warmth to the hands and feet (**not** the arms and legs) allows heat to enter the deep venous circulation. Theoretically, this would provide a good means of rewarming hypothermic patients, but the method does not seem to add enough heat to be worthwhile.*[17]

The roots of the aggressive treatment of immersion hypothermia can be traced to the military, which for years used warm bath treatment for immersion hypothermia with success. Aggressive rewarming in warm water was first recommended by the Luftwaffe based upon the infamous Dachau experiments, even though the studies are scientifically suspect.[193, 194] This treatment has been subsequently recommended and used by the British Royal Air Force and the U.S. Navy, although no recent documentation of survival rates is available.[195-197]

As MacLean and Emslie-Smith say: "Rapid active surface rewarming by immersion in warm water is becoming the accepted management of fit young adults overwhelmed by cold either by immersion or by exposure."[150] Keatinge, in an editorial in the British Journal of Medicine, said "The principles of treating simple hypothermia remain straight forward and generally uncontroversial for victims who still have a carotid pulse and respiration, however slow and difficult these may be to detect. External rewarming in the horizontal position by warm air, or by a warm bath not hot enough to be painful to the rescuer's elbow, is usually effective."[198] A recent report notes that warm bath immersion (40° C=104° F) was successful in a case of severe hypothermia (30.5° C=87° F) caused by a mix of immersion and exposure; there was no afterdrop.[199] One study found that, for mild hypothermia, warm bath rewarming caused little afterdrop.[184] In a study of acutely hypothermic dogs, rapid rewarming in a warm bath caused **less** rewarming shock than slow rewarming.[105] The University of Chicago has used warm bath rewarming on eighteen patients with chronic hypothermia (mostly deep) with no deaths.[200]

Except for cardiopulmonary bypass, warm water immersion is probably the quickest way to rewarm someone. If you are using a hot bath for rapid rewarming, keep the temperature about 105-110° F (41-43° C); keep the arms and legs elevated and out of the warm water; and keep a towel soaked in the warm water around the patient's neck. It seems reasonable to rewarm the trunk

* The "HeatPac" is available in the U.S. through A.B. Russell Co., Box 373, Route 100, Waitsfield, VT 05673; (802) 496-2401.

first, to more nearly approach core rewarming. The one study that looked at this question showed that, for mildly hypothermic volunteers, it made no difference in rewarming rate or afterdrop whether the limbs were in the bath or not.[201] Nonetheless, this only studied mild hypothermia, and for severely hypothermic patients, rewarming the trunk first still seems appropriate. If you have a patient with mild hypothermia and frostbite, this study suggests it would be best to rewarm the torso and extremities at the same time. Monitor carefully for rewarming shock (even in patients who are merely cold but with normal core temperatures); if the blood pressure begins dropping, add cool water to the bath (to slow rewarming), remove the person from the bath and place in the shock position (flat with legs elevated), or give more IV fluids. (One animal study showed that rewarming shock during warm bath rewarming was easily treated with IV fluids.[105]

The danger of trying to defibrillate a patient in a warm bath is cited as a reason to avoid it. However, if the patient is colder than 86° F (30° C), defibrillation is generally useless. On the other hand, trying to manage the patient while in a bathtub, especially with IVs or central lines, is complex at best.

A cold but non-hypothermic person may of course make his or her own decision about a hot bath, but you might point out the problem of rewarming shock and suggest a bath rather than a shower, and suggest leaving the arms and legs out of the bathtub until last.

Radio Frequency Rewarming

RF rewarming shows promise as a possible field rewarming device. Jokes about drying small animals in microwave ovens notwithstanding, radio frequency coils of the right frequency and energy can effectively rewarm hypothermic patients with no detectable ill effects. RF can rewarm at a rate similar to warm bath rewarming, yet preferentially rewarm the core first.[202-209] Though this can potentially become a relatively lightweight field rewarming device, the only currently available apparatus is large and heavy.

Summary

To review major important points about hypothermia:

- Hypothermia is divided into deep and mild by the temperature 90° F (32° C). Those with mild primary hypothermia and no medical problems can be rewarmed by any method and will do well.
- Hypothermia affects mental function, and may do so without the sensation of being cold.
- You must suspect hypothermia in any person exposed to the cold who acts intoxicated or confused. Even if the core temperature is normal, the person may have "incipient hypothermia" and need rewarming.
- Many medications and illnesses predispose to hypothermia.
- Hypothermic patients must be handled gently to prevent ventricular fibrillation, should not exert themselves during rescue, and should be carried flat or in the slightly head-down position.
- Hypothermia can be acute, subacute, or chronic.
- Acute hypothermia, as from immersion, even if severe, is best treated by rapid rewarming by whatever means are available.
- Those with subacute (mountain, exhaustion) hypothermia need fluid and food calories as well as rewarming.
- Those with chronic hypothermia, generally secondary to drugs or illness, tend to do poorly and must be managed carefully.
- Cold water submersion (near-drowning) is **not** the same as acute (immersion) hypothermia; the treatment for the two is very different.
- Hypothermic patients, especially those with subacute hypothermia, are very fluid depleted, and need IV fluids. Some of the fluid is lost into the tissues, and dextran (a type of IV fluid) will help bring this fluid back into the blood where it belongs.
- Those who have been hypothermic for a long time may have trouble handling large fluid loads, because of "stiffness" of the heart. In such cases, you must monitor carefully for signs of fluid overload.

- "Nobody is dead until they are warm and dead." This is true unless there are reliable signs of death even for hypothermic patients.
- If you come upon a cold an apparently dead person, you may start artificial respiration but should not start external cardiac compression if there are any signs of life. Check for 3 minutes for pulse, heartbeat, and respiration. Check for a rhythm with an EKG monitor if you have one. If there is an organized rhythm, even as slow as 20, you may start artificial respiration but don't start external cardiac compression.
- Use normal rates for artificial respiration and external cardiac compression. Give O_2 if available.
- Hypothermic patients in cardiac arrest may survive long periods without cardiopulmonary resuscitation, if necessary for rescue.
- The criteria for endotracheal intubation are the same for normothermic and hypothermic patients. Intubation does not put hypothermic patients at any more risk of ventricular fibrillation than normothermic patients.
- It is difficult or impossible to defibrillate deeply hypothermic patients. Use standard ACLS procedures if above 30°. C (86° F). If the patient is colder, try bretylium and defibrillation, but don't give other ACLS drugs, particularly procainamide.
- If your hypothermic patient has atrial fibrillation, atrial flutter, PVCs, or various kinds of atrioventricular block, don't treat them; specifically, don't give atropine and don't use a pacemaker.
- If you must transport a hypothermic patient, and you can give intravenous medications, give a single dose of bretylium to prevent ventricular fibrillation during evacuation.
- If you have a hypothermic patient who has no detectable signs of life, consider transport to a facility that can perform bypass rewarming; call ahead to alert the facility.
- When caring for a hypothermic patient in the field, **add as much heat as you can, using any and every method available.** However, don't delay evacuation to rewarm the patient. Also provide fluids and food calories.

A variety of good reviews and reference texts are available to those who would like to do further reading about hypothermia.[36, 57, 79, 145, 150, 176, 210-216]

Glossary

:Apocryphal: Referring to a story; means that it illustrates an important point but isn't necessarily true in detail.

:Benzodiazepine: A class of drugs that relax muscles, mask anxiety, and cause sedation. Diazepam (e.g., Valium®) is a well-known example. Others include chlordiazepoxide (e.g., Librium®), lorazepam (e.g., Ativan®), alprazolam (Xanax®), and midazolam (Versed®).

Angiogram: An X-ray of blood vessels made by injecting a special X-ray dye into the vein or artery

Barbiturate: A class of sedative drugs. Phenobarbital is an example.

Dictum: rule.

Etiology: the causes of a disease, or the science of studying such causes.

Gangrene: Dry gangrene refers to death of tissue (necrosis) with shriveling and blackening, as after frostbite. Wet gangrene refers to infected dead tissue, and gas gangrene refers to a specific and deadly type of infection that occurs in dead tissue and causes gas to form in the tissue.

Hypothyroidism: Low thyroid hormone levels. Thyroid hormone regulates the metabolic rate.

Intercellular Fluid: Interstitial fluid.

Interstitial Fluid: Same as intercellular fluid. Fluid found between the cells. Interstitial fluid comes from plasma leaking out of small veins and capillaries. The fluid then drains into lymph vessels (lymphatics), which then drain back into the large central veins in the chest.

Midazolam: Trade name Versed®. A very short-acting muscle-relaxing benzodiazepine, similar to diazepam (e.g., Valium®).

Necrosis: Death of tissue.

Neuropathy: Damage to nerves.

Nuclear Scan: A type of diagnostic study made by injecting very slightly radioactive particles into the blood, then using a special camera to make a picture of where the radioactivity deposits. Examples include a bone scan, looking for areas of infection or cancer, or a lung scan, looking for clots in the pulmonary arteries.

Prognosis: The outlook for progression or resolution of a disease; how a person with the disease may reasonably expect to do.

Prophylactic: "Tending to ward off disease." E.g., lidocaine is given to patients with a myocardial infarction as a prophylactic measure, hoping to prevent ventricular fibrillation.

Raynaud's Syndrome: A disease characterized by spasm of the finger and toe arteries in the cold, causing cold, blue digits.

Relative Hypovolemia: Too little volume to fill the vascular system; refers to a normal amount of volume in an expanded vascular system.

Tinnitus: ringing in the ears. Seen as a toxic effect from high aspirin levels, or from ear infection or ear trauma.

Vasodilator: any medicine that causes vasodilation. Nifedipine (e.g., Procardia®, Adalat®) is a vasodilator sometimes used in the prehospital setting.

Vasospasm: Spasm of blood vessels, especially small arteries. Found in a variety of conditions, including Raynaud's Syndrome, chilblain, immersion foot, and the late phase of frostbite.

Versed®: Midazolam. A very short-acting muscle-relaxing benzodiazepine, similar to diazepam (e.g., Valium®).

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