

WILDERNESS MEDICAL SOCIETY PRACTICE GUIDELINES

Wilderness Medical Society Practice Guidelines for the Prevention and Treatment of Frostbite: 2014 Update

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The Wilderness Medical Society convened an expert panel to develop a set of evidence-based guidelines for the prevention and treatment of frostbite. We present a review of pertinent pathophysiology. We then discuss primary and secondary prevention measures and therapeutic management. Recommendations are made regarding each treatment and its role in management. These recommendations are graded on the basis of the quality of supporting evidence and balance between the benefits and risks or burdens for each modality according to methodology stipulated by the American College of Chest Physicians. This is an updated version of the original guidelines published in *Wilderness & Environmental Medicine* 2011;22(2):156–166.

Key words: frostbite, frostbite prevention, hypothermia, rewarming, aloe vera, thrombolysis

Introduction

The Wilderness Medical Society (WMS) convened an expert panel to develop a set of evidence-based guidelines for prevention and treatment of frostbite to guide clinicians and disseminate knowledge about best practices in this area of clinical care. We present the main prophylactic and therapeutic modalities and make recommendations about their role in injury management. Recommendations are graded on the basis of the quality of supporting evidence and balance between the benefits and risks or burdens for each modality. We then provide suggested approaches for prevention and management that incorporate these recommendations.

The expert panel was convened at the 2010 Annual Winter Meeting of the WMS in Park City, UT. Members were selected on the basis of their clinical or research

experience. Relevant articles were identified through the MEDLINE database using the search terms frostbite, frostbite management, prehospital frostbite treatment, prehospital frostbite management, frostbite prevention, first aid frostbite treatment, and first aid frostbite, and were restricted to the English language. Studies in these categories were reviewed and level of evidence was assessed. The panel used a consensus approach to develop recommendations regarding each modality and graded each recommendation according to criteria stipulated by the American College of Chest Physicians (ACCP) statement on grading recommendations and strength of evidence in clinical guidelines (see online ACCP [Supplementary Table 1](#)).¹ This is an updated version of the original guidelines published in *Wilderness & Environmental Medicine* 2011;22(2):156–166.

Pathophysiology of Frostbite

The freezing injury of frostbite may be divided into 4 overlapping pathologic phases: prefreeze, freeze-thaw,

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vascular stasis, and late ischemic. The prefreeze phase consists of tissue cooling with accompanying vasoconstriction and ischemia, but does not involve actual ice crystal formation. Neuronal cooling and ischemia produces hyperesthesia or paresthesia. In the freeze-thaw phase, ice crystals form intracellularly (during a more rapid-onset freezing injury) or extracellularly (during a slower freeze), causing protein and lipid derangement, cellular electrolyte shifts, cellular dehydration, cell membrane lysis, and cell death.² The thawing process may initiate ischemia-reperfusion injury and the inflammatory response. In the vascular stasis phase, vessels may fluctuate between constriction and dilation; blood may leak from vessels or coagulate within them.³⁻⁵ The late ischemic phase results from progressive tissue ischemia and infarction from a cascade of events including inflammation mediated by thromboxane A₂, prostaglandin F_{2α}, bradykinins, and histamine; intermittent vasoconstriction of arterioles and venules; continued reperfusion injury; showers of emboli coursing through the microvessels^{6,7}; and thrombus formation in larger vessels.⁸ Destruction of the microcirculation is the main factor leading to cell death.⁹ The initial cellular damage caused by ice crystals and the subsequent postthawing processes are made worse if refreezing follows thawing of injured tissues.^{10,11}

Classification of Frostbite

Frostbite has been divided into 4 tiers or “degrees” of injury, historically following the classification scheme for thermal burn injury. These classifications are based on acute physical findings and advanced imaging after rewarming.¹² These categories can be difficult to assess in the field and before rewarming because the still-frozen tissue is hard, pale, and anesthetic. An alternate 2-tiered classification that is more appropriate for field use is suggested after the 4-tier classification.

Frostnip is distinct from frostbite but may precede it. Frostnip is a superficial nonfreezing cold injury associated with intense vasoconstriction on exposed skin, usually cheeks, ears, or nose. Ice crystals, appearing as frost, form on the surface of the skin. By definition, ice crystals do not form in the tissue nor does tissue loss occur in frostnip. The numbness and pallor resolve quickly after covering the skin with appropriate clothing, warming the skin with direct contact, breathing with cupped hands over the nose, or gaining shelter that protects from the elements. No long-term damage occurs. The appearance of frostnip signals conditions favorable for frostbite and appropriate action should be undertaken immediately to prevent injury.

First-degree frostbite presents with numbness and erythema. A white or yellow, firm, slightly raised plaque

develops in the area of injury. No gross tissue infarction occurs; there may be slight epidermal sloughing. Mild edema is common.

Second-degree frostbite injury results in superficial skin vesiculation; a clear or milky fluid is present in the blisters, surrounded by erythema and edema.

Third-degree frostbite creates deeper hemorrhagic blisters, indicating that the injury has extended into the reticular dermis and beneath the dermal vascular plexus.

Fourth-degree frostbite injury extends completely through the dermis and involves the comparatively avascular subcutaneous tissues, with necrosis extending into muscle and to the level of bone.

To simplify classification, after spontaneous or formal rewarming but before imaging, we favor the following 2-tier classification scheme:

- *Superficial*—no or minimal anticipated tissue loss, corresponding to 1st- and 2nd-degree injury
- *Deep*—deeper injury and anticipated tissue loss, corresponding to 3rd- and 4th-degree injury

Severity of frostbite may vary within a single extremity.

Prevention

The adage that “prevention is better than treatment” is especially true for frostbite, which is typically preventable and often not improved by treatment. Risk of frostbite can also be related to underlying medical problems, and prevention must address both environmental and health-related aspects. Frostbite injury occurs when tissue heat loss exceeds the ability of local tissue perfusion to prevent freezing of soft tissues (blood flow = heat). One must both ensure adequate perfusion and minimize heat loss to prevent frostbite.

MAINTAINING PERIPHERAL PERFUSION

Preventive measures to ensure local tissue perfusion include: 1) maintaining adequate core temperature and body hydration; 2) minimizing effects of known diseases or medications and drugs that may decrease perfusion; 3) covering all skin and the scalp to avoid vasoconstriction; 4) minimizing restriction in blood flow, such as constrictive clothing, footwear, or immobility; 5) ensuring adequate nutrition; and 6) using supplemental oxygen in severely hypoxic conditions (eg, >7500 m). Recommendation grade: 1C.

EXERCISE

Exercise is a specific method to maintain peripheral perfusion because it enhances the level and frequency of

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