15 From the Mountain Top to the Sea Bottom: Training and Preparing for Wilderness, Environmental Medical Events
WILLIAM BINDER, MD, MA, FACEP
GUEST EDITOR

16 Wilderness Dermatology: Bugs, Plants, and Other Nuisances That May Ruin Your Hike
DOMINIC J. WU, MD; JENNIFER LEE, MD; AFTON CHAVEZ, MD; JOHN C. KAWAOKA, MD

23 Sailing Injuries: A Review of the Literature
ANDREW NATHANSON, MD, FACEP, FAWM

28 Accidental Hypothermia: ‘You’re Not Dead Until You’re Warm and Dead’
JOHN L. FOGGLE, MD, MBA, FACEP

33 A Free Diver with Hemoptysis and Chest Pain
CHANA RICH, MD; KRISTINA MACATEER, MD; VICTORIA LEYTIN, MD; WILLIAM BINDER, MD

37 Learning from the Outside In: Incorporating Wilderness Medicine into Traditional Emergency Medicine Education
DANIEL COLEMAN, MD; CHANA RICH, MD; HEATHER RYBASACK-SMITH, MD

42 Climate Change: A Review of a Public Health Opportunity for the Northeast
REENE N. SALAS, MD, MPH, MS

Cover photo was taken at the Brown Wilderness Medicine Race held in 2018 in Lincoln, RI. Twenty-five residents and eight Brown medical students, as well as volunteer faculty and members of the Warwick Fire Department Technical Rescue Team in Warwick, RI; and the Cumberland EMS in Cumberland, RI; participated in the event. It was funded by the Department of Emergency Medicine.
In this issue of the *Rhode Island Medical Journal* (RIMJ) we are pleased to present several wide-ranging topics in wilderness and environmental medicine. Human interaction with the natural environment has led to a burgeoning interest in wilderness medicine, a multi-specialty area of interest that requires an ability to diagnose and improvise in areas where resources may be scarce. **DOMINIC WU**, et. al. discuss dermatologic misfortunes that may impact outdoor travel. Sailing, one of the most ancient means of transport through the most vast, uninhabited regions on earth, is frequently the source of injury. **ANDREW NATHANSON** discusses the wide range of injury and illness incurred while sailing, when resources and space are limited and definitive care may be days to weeks away. **JOHN FOGGLE**, who recently toured Antarctica, reviews the modern management of hypothermia, and **DANIEL COLEMAN**, et. al. provide a fascinating discussion of the impact of wilderness medicine on medical education. **CHANA RICH**, et. al. provides a wonderful clinical contribution from the Brown University Emergency Medicine Department of a case of free diving off the coast of Rhode Island and alveolar hemorrhage.

In a slight departure from previous issues of RIMJ, we are fortunate to have a non-Rhode Island author contributing to this issue. **RENEE SALAS**, who completed her fellowship in Wilderness Medicine at Massachusetts General Hospital, and who was a former colleague of mine, has become increasingly involved with the Harvard Global Health Institute. Dr. Salas was the recent lead author of the *Lancet’s Countdown U.S. Brief on Health and Climate Change*, and reviews the impact of climate change on health and economies here in the northeast. Her sobering discussion will help us plan for the impact of climate on the health of our patients in New England and in Rhode Island.

We hope you enjoy this RIMJ focus section issue on the rapidly developing field of wilderness medicine that is unfolding on myriad educational, research, and technical fronts across the medical, societal and global climatological spectrum as these articles outline.
ABSTRACT
Spending time outdoors can be rewarding. However, exposure to the sun, insect bites, and plant exposures may result in a wide range of dermatologic manifestations. In this article, we describe potential cutaneous manifestations of common wilderness exposures in New England including photodermatoses from prolonged sun exposure, phytodermatoses from plant exposures, and arthropod-bite reactions from common insects (mosquitoes, spiders, ticks, hymenoptera, mites, and chiggers). The article will also address preventive and treatment strategies which may help physicians and their patients better prepare for spending time in the wilderness.

KEYWORDS: dermatology, wilderness, phototoxicity, arthropod reactions, plant exposure

INTRODUCTION
Although hiking, biking, skiing, and other outdoor pursuits are wonderful physical activities and can enhance one’s mental wellbeing, it is vital to take appropriate preventative measures to ensure the most enjoyable experience possible. Environmental exposure to the sun and altitude puts one at risk of sunburn and both acute and chronic skin damage. Insect bites can induce an array of dermatologic manifestations that, if not appropriately managed, may result in serious medical complications. Plant exposures can result in multiple cutaneous manifestations including allergic contact dermatitis, phytophotodermatitis, irritant contact dermatitis, and contact urticaria.

SUN EXPOSURE
Prolonged ultraviolet (UV) radiation exposure may result in cutaneous manifestations including sunburn, aging, and malignancies. Approximately 1 in 5 people in the United States may develop skin cancer in their lifetimes. Both UVA and UVB radiation may cause skin cancer, with UVA primarily responsible for chronic skin changes such as photoaging, wrinkling, and lentigines. Even on cloudy days, up to 80% of harmful UV radiation can reach the skin. As one reaches higher in altitude, the total amount of UV radiation exposure increases as much as 30% for every 1000 meters.

a. Sun Protection
All people older than 6 months of age, regardless of skin color, should apply a broad-spectrum and water-resistant sunscreen of at least SPF 30. It is also important to re-apply sunscreen at least every 2 hours, and after each water exposure. Patients should seek shade when possible, avoid sun at peak hours between 10am–2pm, and wear sun protective clothing including broad-brimmed hats and sunglasses. The American Academy of Pediatrics recommends that children younger than 6 months of age should have minimal exposure, wear appropriate clothing and have adequate shade.

b. Drug-induced Phototoxicity
Some medications can induce phototoxic rashes that resemble sunburns after sun exposure. Common offenders include NSAIDs, quinolones, tetracyclines, and diuretics. This side effect is thought to be due to UV light absorption by the medication, leading to free radical formation and cellular damage in sun-exposed areas. The severity of the effect is often dose-dependent and linked to the duration and intensity of sun exposure. The phototoxic rash often occurs within 30 minutes of UV exposure and begins with burning and pruritus, with or without wheals, and further develops into a bright red edematous photodistributed rash. This may progress to erythema, hyperpigmentation, edema, and even blistering.

Management is supportive and involves prevention strategies such as sun protection, limiting sun exposure, and dose reduction or elimination of the offending medication. Cool compresses and ice packs may offer significant relief. Similar to sunburns, the patient should maintain hydration and use gentle emollients.

INSECTS
In the northeast United States, the most common cutaneous complaints from insect exposure are due to mosquitoes, mites and chiggers, gypsy moths, fleas, spiders, hymenoptera, and ticks. Their bites may contain toxins and irritants that can cause a wide range of dermatologic manifestations.
a. Mosquitos
Mosquitos are vectors for many diseases including West Nile virus, encephalitis, and more. Mosquito bites classically present as pruritic wheals and papules, often with a central punctum. Depending on the victim’s immune response, these bites may appear as urticaria, vesicles, or even as granulomatous lesions.5

Prevention strategies include avoiding sites with stagnant water, wearing light-colored long-sleeved clothing to easily identify mosquitos, utilizing mosquito nets, and wearing insect repellant. Insect repellant containing DEET 10–35% or picaridin 20% can be quite effective,6 and clothes can also be washed with permethrin.

Mosquito bites are generally self-limited. Ice packs, topical steroids, and over-the-counter topical products such as pramoxine 1% cream, menthol-containing products, and oral antihistamines may help patients with more severe and symptomatic reactions.7 First-generation antihistamines are more sedating than second-generation agents, but tend to be more effective for pruritus.

b. Spiders
Most spider bites encountered in the U.S. are harmless and may induce a brief localized skin reaction that spontaneously resolves. However, black widow [Latrodectus mactans] and brown recluse [Loxosceles reclusa] spider bites may require more directed medical care (Table 2).

Female black widow spiders are more commonly found in the South up to Southern New England and in the West. Their bites transmit a venom containing α-latrotoxin, which leads to a large release of acetylcholine. The bite site typically appears as a painful pink edematous papule or plaque with central fang marks with possible central clearing. The victim may then experience muscle spasms and cramps within an hour in the chest or abdomen that may mimic a myocardial infarction. Treatment options include intravenous benzodiazepines for muscle spasms and narcotic pain medications. Antivenom can be considered up to 48 hours after a bite if severe pain persists after routine therapy.8

Brown recluse spiders are rare in the Northeast. The bite itself may be only slightly painful. However, soon after the bite, a tender erythematous halo rash may develop. In many cases, this progresses to central necrosis, sometimes even requiring a skin graft. It is thought that sphingomyelinase D in the venom triggers platelet aggregation and activates thromboxane B2 which leads to skin necrosis.6,9 Treatment options are controversial; however, most experts suggest that overly aggressive management, such as excising the bite site to prevent necrosis, is harmful and not indicated. Most suggest proper wound care and minor debridement, with or without antihistamines or dapsone.10

c. Ticks
Ticks can carry a multitude of diseases including Lyme disease [Table 1]. Tick bites are typically not painful, as their saliva contains anesthetic and anticoagulant factors.

Lyme disease typically presents with the erythema migrans rash (red macule or thin plaque that slowly expands outwards with central clearing resembling a target). The incidence of Lyme disease has increased since 2007, and the geographic distribution has broadened from primarily New England, the Mid-Atlantic States, and Wisconsin to include adjacent states.11 If the tick is identifiable as I. scapularis, has been attached to the host for more than 36 hours and is engorged in a Lyme-endemic area, prophylactic treatment with a single dose of doxycycline 200 mg orally may be administered within 72 hours of tick removal (if no other contraindications).12

Ticks should be removed carefully using a pair of forceps or a tick-removal device by grasping the tick as close to the skin as possible and removing the parasite with gentle, steady traction perpendicular to the skin without twisting.6,13

Tick bites may be prevented by wearing clothing treated with permethrin. Applying DEET-containing insect repellants, tucking pants into socks, wearing long-sleeved clothing and pants, and performing daily tick checks can help to reduce potential exposure.

d. Hymenoptera (Bees, Wasps)
Stings from bees and wasps can be incredibly painful, and may even provoke anaphylaxis. Their stings often produce immediate burning and pain at the site, followed by an erythematous wheal. This usually resolves spontaneously within a few hours. However, some individuals have a more exaggerated response, such as swelling greater than 6 inches in diameter lasting up to 7 days. More severe local reactions in select individuals may be due to venom-specific IgE antibodies in sensitized victims.8

To remove a bee or wasp sting, the victim should ideally use a straight, hard surface such as the edge of a credit card to nudge out and remove the stinger. One should avoid using fingers or tweezers to remove the stinger, as this may squeeze additional venom into the stinging site. Some stings, such as that of the honeybee, have venom sacs and attached musculature that may continue to pump venom if the sac is not removed. Intradermal skin allergy testing can be performed with dilute quantities of venom to identify individuals at high risk of anaphylaxis to venom. These individuals at risk for hymenopteran anaphylaxis have the option of undergoing venom immunotherapy which has been shown to be effective. They should always carry a preloaded epinephrine device for emergency administration.5,14 (Table 3)
<table>
<thead>
<tr>
<th>Disease/Organism/Vector</th>
<th>Vector Image</th>
<th>Cutaneous findings and clinical photo</th>
<th>Non-cutaneous findings/Tests</th>
<th>Treatments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Rocky Mountain Spotted Fever</strong></td>
<td><img src="image" alt="Dermacentor variabilis" /></td>
<td><img src="image" alt="Rocky Mountain Spotted Fever rash" /></td>
<td>Non-cutaneous findings: -Fever -Myalgias -Nausea, vomiting -Headaches Tests: -Serology with indirect immunofluorescence assay (IFA) -ELISA</td>
<td>Doxycycline (even in children &lt;8) May use chloramphenicol if pregnant</td>
</tr>
<tr>
<td><strong>Lyme Disease</strong></td>
<td><img src="image" alt="Ixodes scapularis" /></td>
<td><img src="image" alt="Erythema migrans" /></td>
<td>Non-cutaneous findings: A) Early localized disease -flu like symptoms B) Early disseminated disease -facial nerve palsy -joint pain -carditis C) Chronic disease -persistent neurologic and rheumatologic symptoms Tests: -Enzyme immunoassay for IgM and IgG antibodies -Western blot of enzyme immunoassay positive or equivocal</td>
<td>Doxycycline in adults and children &gt; 8 yrs old: 14–21 days Amoxicillin in pregnant women, children &lt; 8 yrs old: 14–21 days For severe disseminated disease - Ceftriaxone IV Prophylaxis if 1) Tick is Ixodes scapularis tick and has been attached for approximately 36 hrs 2) Post exposure prophylaxis within 72 hours of tick removal 3) Local rate of infection with Borrelia at least 20% 4) Doxycycline isnot contraindicated When these criteria are met, treat with: Doxycycline 200mg in single dose w/I 72 hours of tick bite for adults Doxycycline 4mg/kg in children &gt; 8 yrs old (max dose 200mg) w/I 72 hrs of tick bite</td>
</tr>
<tr>
<td><strong>Babesiosis</strong></td>
<td>See Lyme disease</td>
<td></td>
<td>Non-cutaneous findings: -flu like symptoms -fatigue -malaise -fevers -chills -myalgias -occasionally mild hepatomegaly or splenomegaly -Dark-colored urine due to hemolytic anemia Tests: -CBC to look for hemolytic anemia -Reticulocyte count -Definitive diagnosis by Giemsa or Wright stains of blood smears which show ring forms and tetrads</td>
<td>Atovaquone and Azithromycin for 7–10 days For severe disease treat with clindamycin IV and quinine</td>
</tr>
</tbody>
</table>
**Table 1. Tick-borne Illnesses (continued)**

<table>
<thead>
<tr>
<th>Disease/Organism/Vector</th>
<th>Vector image</th>
<th>Cutaneous findings and clinical photo</th>
<th>Non-cutaneous findings/Tests</th>
<th>Treatments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Disease:</strong> Anaplasmosis</td>
<td>See Lyme disease</td>
<td>Usually no exanthem, so presence of cutaneous findings should raise suspicion for other diseases or coinfection</td>
<td>Non-cutaneous findings: -Fever -Malaise -Myalgias -Headaches -Nausea, vomiting -Confusion Tests: -serology by IFA -ELISA -PCR</td>
<td>Doxycycline in adults and children &gt; 8 yo</td>
</tr>
<tr>
<td><strong>Organism:</strong> <em>Anaplasma phagocytophilium</em></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Tick vector:</strong> <em>Ixodes scapularis</em></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Ixodes pacificus</em></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Disease:</strong> Ehrlichiosis</td>
<td></td>
<td>Exanthem present in 10% of cases -Faint, blanching generalized erythema -Erythematous macules, papules, petechiae</td>
<td>Non-cutaneous findings: -Fever -Malaise -Myalgias -Headaches -Nausea, vomiting -Confusion -Meningoencephalitis -Cranial nerve palsies Tests: -serology by IFA -ELISA -PCR</td>
<td>Doxycycline in adults and children &gt; 8 yo</td>
</tr>
<tr>
<td><strong>Organism:</strong> <em>Ehrlichia chaffeensis</em></td>
<td>Female lone star tick¹</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Tick vector:</strong> <em>Amblyomma americanum</em></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

e. Mites and Chiggers

Mites are small arthropods with eight legs whose bites may cause pruritic papules. Its six-legged larval form is called the chigger, which appear as tiny red insects that crawl around until they reach a barrier such as lining of a sock. The mites’ saliva sometimes provokes an allergic reaction from the human host, producing a pruritic papule. Mite and chigger bites classically present as grouped 1–2 mm pruritic red papules with an abrupt demarcation line around borders of clothing such as around the ankles.

These bites tend to be self-limited and may be treated symptomatically for more severe reactions. Prevention and treatment strategies are similar to those for mosquitoes.

**PHYTODERMATOSES**

Exposure to plants may result in a variety of cutaneous manifestations including phytophotodermatitis, irritant contact dermatitis, allergic contact dermatitis, and contact urticaria.

a. Phytophotodermatitis

Phytophotodermatitis is a cutaneous eruption resulting from the exposure of skin to photosensitizing agents from plants such as furocoumarins (eg. psoralen) which react with UV radiation. Some plants that are known to cause phototoxicity include lemon, lime, celery, carrots, dill, and anise. A common presentation is a patient who squeezed limes out-doors and subsequently developed a burning, erythematous rash. Wild parsnip (*Pastinaca sativa*) is found throughout New England and may cause a severe phytophotodermatitis reaction.

Cutaneous manifestations include burning and painful, bullous, erythematous eruptions, which may result in chronic skin hyperpigmentation.¹⁵ Treatment is mostly symptomatic (with cool compresses and oral antihistamines if needed), and prevention involves avoidance of triggers. With appropriate sun protection and time, the post-inflammatory hyperpigmentation should resolve spontaneously.

b. Irritant contact dermatitis

There are two main categories of irritant contact dermatitis (ICD) from plant exposures: mechanical (physical injury), and chemical.

Mechanical ICD is a result of physical trauma to the skin caused by a plant’s trichomes (hairs), spines, glochids (barbed hairs), or thorns. These defense mechanisms may breach the epidermis and trigger a papular eruption. A classic example of this are cacti which contain spines and sometimes glochids, which, if a person is exposed, may present as erythematous papules and nodules that spontaneously resolve with time.

Chemical ICD is often provoked by plant chemicals such as calcium oxalate that may be transferred to the epidermis after physical contact with a plant, resulting in subsequent inflammation. Daffodils commonly cause erythema, dry skin, as well as scaling of the finger tips among florists due...
### Table 2. Arthropod and Snake Reactions

<table>
<thead>
<tr>
<th>Organism and Image</th>
<th>Presentation</th>
<th>Treatment</th>
</tr>
</thead>
</table>
| Scorpion ²         | - Pain and paresthesias of bite site  
   - May have neurological or cardiopulmonary complications | - Remove stinger  
   - Supportive care  
   - Ice  
   - Antihistamines |
| Bees, wasps, hornets | - Ranges from pain and local edema at site of bite to urticaria, respiratory distress, anaphylaxis | - Remove stinger, symptomatic care |
| Fire ants ²        | - Line or ring of pustules with surrounding red or hemorrhagic halo  
   - May have neurologic systemic symptoms, anaphylaxis | Symptomatic care |
| Snake bite  
   - Crotalidae: rattlesnake, copperhead ³, and cottonmouth moccasin  
   - Elapidae: coral snake (touching red and yellow bands) ³ | - Fast onset pain, swelling, hemorrhage, necrosis | Emergency: visit closest emergency department  
   - Antivenom, tetanus prophylaxis, possible antibiotics |
| Gypsy moth | - Eczematous, pruritic dermatitis  
   - Urticaria | - Strip bite site with adhesive tape to remove caterpillar hairs  
   - Wash site with soap and water  
   - Topical or oral steroids for severe reactions |
| Io moth ⁴ | - Immediate pruritus and stinging of involved skin | Same as gypsy moth |
| Puss caterpillar ⁴ | - Intense burning pain  
   - Hemorrhagic linear track marks | Same as gypsy moth |
| Saddle back caterpillar ⁴ | - Immediate painful stinging  
   - Redness  
   - Edema | Same as gypsy moth |
| Black widow spider ⁵ | - Hourglass-shaped marking on abdomen  
   - Acute edema  
   - Pain  
   - Symptoms resembling a surgical abdomen | - Antivenom  
   - Benzodiazepines  
   - IV calcium gluconate |
| Brown recluse spider ⁵ | - Violin-shaped marking on head  
   - Erythema  
   - Bullae  
   - Necrosis  
   - Possible disseminated intravascular coagulation | - Ice  
   - Elevation  
   - Possibly dapsone |

**References**

to contact with calcium oxalate crystals. Reactions tend to be self-limited and require no medical treatment.

c. Allergic contact dermatitis

Allergic contact dermatitis (ACD) is a type of delayed or type IV hypersensitivity reaction. Urushiol found in poison ivy, poison oak, and poison sumac, is a common cause of ACD in the United States. Although poison ivy may appear as clusters of three leaves, poison oak and sumac have anywhere from three to 13 leaves. Clinically, these lesions appear as erythematous pruritic patches and plaques within 48 hours of contact, which often progress to vesicles in a linear distribution, most often on extremities. Crusted plaques and even bullae, along with significant edema may also be seen. A key exam finding is lesions in a linear configuration where the leaves have brushed against the skin.

Immediate management involves removal of the plant oils using soap and water or rubbing alcohol. All plant oils should also be washed from clothing and gear that may have touched the oils. High potency topical corticosteroids such as clobetasol 0.05% cream or betamethasone dipropionate 0.05% cream or ointment may be applied on the trunk and extremities for limited skin disease. For lesions on the face and skinfolds, low potency topical corticosteroids such as hydrocortisone 2.5% cream or ointment are preferred. In more severe cases, long taper of oral steroids of 2–3 weeks may be necessary. Shorter courses of oral corticosteroids may result in a rebound flare.

**CONCLUSION**

Hiking and spending time outdoors is a healthy and potentially therapeutic pastime. Exposures to insects, plants, and the weather, however, can result in a multitude of symptomatic dermatologic manifestations. Equipping oneself with the knowledge of potential exposures and prevention and treatment strategies can help physicians prepare patients to make the most out of their time in the wilderness.

**References**


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Sailing Injuries: A Review of the Literature
ANDREW NATHANSON, MD, FACEP, FAWM

ABSTRACT
Sailors are at risk for acute injuries, overuse injuries, environmental injuries, and sailing-related illnesses. Sailing-related injury rates vary from 0.29 to 5.7 per 1,000 hours which is lower than many other land-based sports. However, the fatality rate of 1.19 per million sailing-days is relatively high. The most common injuries are contusions and lacerations predominantly to the upper and lower extremities. Falls and impacts from various parts of the sailboat are the most common mechanisms of traumatic injury. High winds, operator inexperience, and operator inattention are the most common contributing factors for injury. Among Olympic-class sailors, overuse injuries to the back (29–45%) and knees (13–22%) are commonly reported. As many as seventy-three percent of sailing-related deaths are due to drowning as a result of falls overboard (39–44%) or capsizing the vessel (20–40%). Eighty-two percent of sailing-related drowning victims in US waters were not wearing a lifejacket. Leading contributing factors to fatal sailing accidents are high winds (12–27%), alcohol use (10–15%), and operator inexperience (8%).

KEYWORDS: sailing, injury, illness, fatalities

INTRODUCTION
By far the largest wilderness areas on earth are oceans and lakes which cover over 71% of the planet’s surface. For over 5,000 years, humans have used sailing craft as an efficient means of traversing these waterways in order to fish, transport goods, and explore distant lands. Although the invention of steam engines in the late 18th century gradually made commercial sailing obsolete, sailing soon transitioned into a recreational pastime, first in Europe, then in the United States. Sailboat racing was first formalized in 1851, when the schooner America defeated 14 British entries in a race around the Isle of Wight in what has since become the America’s Cup, considered the oldest international sporting event in the world.1

Present-day sailing encompasses a broad spectrum of activities from day-sailing on a lake, to around-the-world racing. Sailboats range in size and complexity from single-handed 2.4 m Optimist training dinghies, to 30 m “maxi” racing boats sailed by a crew of 22, and capable of speeds in excess of 30 mph. In 2011 the US Coast Guard (USCG) estimated that 3.7% of all US households owned a sailboat, and that there were 154 million sailing person-hours in US waters.2

The physical demands of sailing vary greatly depending on the type of vessel, the windspeed, whether cruising or racing, as well as the crew member’s position on the boat. In dinghies, the sailor spends significant energy levering his or her body “hiking” over the side of the boat to keep it from capsizing. In keelboats the most demanding activity is turning winches to pull in lines under tension.

Sailors are at risk of injury and illness from a variety of causes. Environmental risks include solar exposure, hypothermia, immersion injuries, and motion sickness. Acute injuries are often caused by falls, direct impact from various parts of the boat, and from lines under tension. Finally, overuse injuries can be sustained by repetitive activities such as “grinding” winches, steering the vessel, and hiking.

Medical care for injury or illness aboard a sailboat presents some unique “wilderness” challenges, particularly when far offshore. Definitive medical care may be days away, supplies are limited, space below-deck is often cramped and poorly lit, and the motion of the vessel can be violent and unpredictable. Evacuation at sea is often dangerous and not always an option. Compounding these challenges is the fact that injuries are most likely to occur during stormy weather when crew members are fully engaged in sailing the boat and may be fatigued or seasick.

Injury rates, mechanisms of injury and types of injury have been found to vary significantly according to the type of sailboat and type of sailing. For ease of study, the medical literature and the US Coast Guard generally categorize sailboats into two groups; vessels greater than 6m in length equipped with a weighted keel for stability and usually motorized, known as keelboats, and smaller, non-motorized dinghies and catamarans which are dependent on crew weight for stability and are easily overturned. Most studies focus on a particular class of sailboat or regatta, so findings may not be generalizable, and comparisons of injury-rates between studies is difficult due to differing definitions of injury and methodologies.
METHODS
A search was performed using PubMed with the terms ‘sail’, ‘sailing’, ‘yacht’, ‘yachting’, and ‘injury’, ‘injuries’, or ‘medical’. Studies were limited to the English language literature between 1990 and 2018. A total of 27 articles were retrieved, from which 14 were selected. Public access USGC Databases were also reviewed.

SAILING INJURIES
Injury Rates, Types of Injury and Mechanisms of Injury

Dinghies
In a study of the 2016 Olympic summer games (raced in 10 classes of smaller boats), 21 out of 360 sailors (5.5%) suffered an injury; only two of those injuries resulted in time-loss from sports participation. This compares favorably to an average injury rate of 9.5% for all Olympic athletes, ranking sailing 26th out of 40 sports regarding risk of injury. Injury rates at an international 2014 Olympic-class regatta, and among elite dinghy sailors in New Zealand, and novice dinghy sailors in Germany have been reported as 0.59 per 1,000 hours, 0.2 injuries per year and 0.29 per 1,000 hours, respectively. The newer, faster, and less stable Olympic-class boats, the 49er and Nacra 17, have higher rates of acute injuries than the other classes.

The most frequent but least severe acute injuries aboard dinghies sailed by elite as well as novice sailors are contusions (9–55%) which are often caused by falls and contact with various parts of the boat during rapid turning maneuvers in confined quarters. Hand injuries (6–31%) including lacerations, fractures, finger dislocations and rope burns from handling lines, the tiller or the center-board are also common. Head injuries, which appear to be more common among intermediate (11%) than more advanced (3%) sailors, are often caused by impact from the low-hanging boom and are among the most severe. Boom-related trauma can cause scalp or facial lacerations, contusions, fractures, and concussions. Capsizing (13–52%) and collisions with other boats or objects (3–23%) are other common mechanisms of injury.

Overuse injuries, particularly among elite competitive racers, are very common and, in some studies, outnumber acute injuries. Hiking in a straight leg position places high static and dynamic loads on the extensor mechanism of the leg, particular the knees, as well as the back. Low back pain (29–45%) and knee pain (13–22%) are consistently the most common chronic conditions reported by dinghy racers.

Table 1. Summary of Dinghy Sailing Injury Studies

<table>
<thead>
<tr>
<th>Study Population (Study Design)</th>
<th>Soligard3</th>
<th>Schaefer6</th>
<th>Tan4</th>
<th>Nathanson7</th>
</tr>
</thead>
<tbody>
<tr>
<td>Population (Study Design)</td>
<td>2016 Olympic Games (Prospective)</td>
<td>Novice sailors (Survey)</td>
<td>Elite Dinghy Racing (Survey)</td>
<td>Dinghies mixed population (Internet Survey)</td>
</tr>
<tr>
<td>Average Age (SD)</td>
<td>24 (6)</td>
<td>238</td>
<td>341</td>
<td>397</td>
</tr>
<tr>
<td>Male Gender</td>
<td>57%</td>
<td>57%</td>
<td>83%</td>
<td></td>
</tr>
<tr>
<td># Injuries</td>
<td>21</td>
<td>238</td>
<td>341</td>
<td>397</td>
</tr>
<tr>
<td>Injury Rate</td>
<td>5.5% of sailors/Olympics</td>
<td>0.29/1,000 hours</td>
<td>0.59/1,000 hours#</td>
<td>4.6/1,000 days</td>
</tr>
<tr>
<td>Type of injury</td>
<td>Laceration/Abrasion 29%</td>
<td>33%</td>
<td>5%</td>
<td>31%</td>
</tr>
<tr>
<td></td>
<td>Sprain/Strain 29%</td>
<td>1%</td>
<td>45%</td>
<td>16%</td>
</tr>
<tr>
<td></td>
<td>Dislocation 4%</td>
<td>4%</td>
<td>2%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Concussion 4%</td>
<td>1%</td>
<td>2%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Burns 4%</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Other 9%</td>
<td>2%</td>
<td>8%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Chronic/overuse 14%</td>
<td>19%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body Part Injured</td>
<td>Head/Neck 0</td>
<td>32%</td>
<td>6%</td>
<td>12%</td>
</tr>
<tr>
<td></td>
<td>Upper extremity 24%</td>
<td>39%</td>
<td>28%</td>
<td>39%</td>
</tr>
<tr>
<td></td>
<td>Other/unknown 5%</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

# Injury rate for sailing and land-based training

Figure 1. Part of dinghy associated with injury, n = 397

With permission from Wilderness and Environmental Medicine
Keelboats

A study of the 2003 America’s Cup reported an injury rate of 2.2 per 1,000 hours of sailing, while the injury rate for land-based conditioning was 8.6 per 1,000 hours. Bowmen and grinders had the highest rates of injury while sailing, and helmsmen the lowest.9 A study of an amateur around-the-world race, and an Internet-based survey of intermediate/advanced sailors found injury rates of 9 and 4.6 per 1,000 sailing days, respectively.10,7 [See Table 2.] By comparison, in studies using similar definitions of injury, recreational alpine skiing was found to have an injury rate of 4 per 1,000 hours, and men’s collegiate soccer 17 per 1,000 hours.11,12

As with dinghy sailing, contusions are common among keel-boat sailors (12–40%), as are lacerations (5–26%).9,7 Many of these injuries are the results of falls (30%) which can be attributed to walking on a wet, lurching, and steeply angled deck, often cluttered with lines, winches, and hatches.7 Impact from rigging, flogging sails and other crewmembers is another cause of injury. [Figure 2] In a study of a mixed population of keelboat sailors, leg contusions (11%) and hand lacerations (8%) were most common.7 In the 635-nautical mile Newport to Bermuda race, 47% of injuries were found to be to the hand/fingers, mostly from operating winches and handling lines under high tension.13 Probably due to the heavier forces involved, fractures have been found to be more common on keelboats (5.7%) as compared to dinghies (2.4%).7 Burns from hot liquids, foods and the engine have also been widely reported in off-shore sailing.10,13 During the 2001–02 Volvo around-the-world race, one-third of injuries occurred below-decks likely due to the forceful and erratic movements of those yachts in heavy weather.14

In offshore racing, helmsmen/women can develop carpal tunnel syndrome from gripping the wheel for prolonged periods of time. Chronic/overuse injuries to the shoulder, elbow, and back are common among grinders in both near and offshore sailing.14

Risk Factors for Injury and Severe/Fatal injuries

Studies of recreational and competitive dinghy and keelboat sailing have consistently identified high winds as a leading contributing factor for injury.4,6,7,13 Not only do stronger winds place exponentially higher loads on rigging, and increase risk of capsize, they can also create large and hazardous seas. Turning maneuvers [i.e. tacks, jibes] have also been identified as contributing factors for injury in sailing craft of all sizes, likely because the boom crosses the boat overhead, and sails and lines must be released on one side of the boat and pulled in on the other. 6,7

### Table 2. Summary of Keelboat Sailing Injury Studies

<table>
<thead>
<tr>
<th>Study</th>
<th>Neville⁹</th>
<th>Nathanson⁷</th>
<th>Price¹⁰</th>
</tr>
</thead>
<tbody>
<tr>
<td>Population (Study Design)</td>
<td>America’s cup keelboats (Prospective)</td>
<td>Keelboats (Internet Survey)</td>
<td>Amateur Around-the-world race (Prospective)</td>
</tr>
<tr>
<td>Average Age (SD)</td>
<td>33 (5)</td>
<td>40 (13)</td>
<td>21-60 (range)</td>
</tr>
<tr>
<td>Male Gender</td>
<td>100%</td>
<td>83%</td>
<td>78%</td>
</tr>
<tr>
<td># Injuries</td>
<td>220</td>
<td>1,226</td>
<td>299</td>
</tr>
<tr>
<td>Injury Rate</td>
<td>5.7/1,000 hours</td>
<td>4.6/1,000 days at sea</td>
<td>9/1,000 days at sea</td>
</tr>
<tr>
<td>Type of injury</td>
<td>Laceration/Abrasion</td>
<td>Fracture</td>
<td>Sprain/Strain</td>
</tr>
<tr>
<td></td>
<td>5%</td>
<td>2%</td>
<td>68%^</td>
</tr>
<tr>
<td></td>
<td>26%</td>
<td>6%</td>
<td>17%</td>
</tr>
<tr>
<td></td>
<td>12%</td>
<td>10%</td>
<td>36%</td>
</tr>
<tr>
<td>Contusion</td>
<td>12%</td>
<td>40%</td>
<td>Dislocation</td>
</tr>
<tr>
<td></td>
<td>10%</td>
<td>2%</td>
<td>1%</td>
</tr>
<tr>
<td>Concussion</td>
<td>3%</td>
<td>7% *</td>
<td>Burns</td>
</tr>
<tr>
<td></td>
<td>7%</td>
<td>16%</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>13%</td>
<td>5%</td>
<td>6%</td>
</tr>
<tr>
<td>Chronic/overuse</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body Part Injured</td>
<td>Head/Neck</td>
<td>Back/trunk</td>
<td>Upper extremity</td>
</tr>
<tr>
<td></td>
<td>14%</td>
<td>20%</td>
<td>40%</td>
</tr>
<tr>
<td></td>
<td>11%</td>
<td>11%</td>
<td>40%</td>
</tr>
<tr>
<td></td>
<td>12%</td>
<td>21%</td>
<td>22%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Lower extremity</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>25%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>38%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>17%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Other/unknown</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>27%</td>
</tr>
</tbody>
</table>

# Injury rate includes land-based training. ^ Including chronic/overuse injuries, * “Head injuries No LOC”

Figure 2. Part of keelboat associated with injury, n = 1,226

*With permission from Wilderness and Environmental Medicine*
While the majority of sailing injuries in most studies are of relatively minor, severe injuries do occur and sailing-related fatalities are more common than in most land-based sports. In a study of 841 sailing injuries reported to the USCG requiring more than first aid, operator inattention (22%) and inexperience (11%) were identified as the leading preventable factors. An analysis of 70 severe injuries requiring evacuation or hospitalization, found that high winds were a contributing factor in 35% of cases, and that 25% of these injuries were fractures, 16% soft tissue injuries, and 14% concussions. Injury patterns for severe injuries included: planned and accidental jibes where the sailor was struck by the boom, or mainsheet; collisions with other boats; dismastings; and falls through open hatches or companionways.

A study of annual USCG Boating Accident Reports from 2000–11 found that there were 271 sailing-related fatalities in US waters during the time period, as compared to 197 football-related deaths. Drowning was found to be the most common cause of death (73%) followed by trauma (10%), and hypothermia (4%). Falls overboard (41%), capsizing (29%), sinking (7%), and collisions (5%) were leading accident mechanisms. Among those who drowned, 82% were not wearing a lifejacket. Using exposure data from the 2011 USCG National Recreational Boating Survey (a large-scale, population-based, weighted survey), a fatality rate of 1.19 deaths per million sailing person-days was calculated, which is similar to a calculated rate of 1.06 deaths per million skiing/snowboarding-days.

The leading operator-preventable contributing factors for fatal sailing accidents were alcohol use (keelboats 15%, dinghies 10%), operator inexperience (8%), and improper lookout/inattention (keelboats 10%, dinghies 5%). The leading non-preventable contributing factors were high winds (keelboats 12%, dinghies 27%), hazardous waters (keelboats 9%, dinghies 10%), and equipment failure (keelboats 4%, dinghies 6%).

### SAILING-RELATED ILLNESSES/ENVIRONMENTAL INJURIES

Although any illness which occurs on land may also occur at sea, prolonged exposure to the elements, confined living quarters, poor hygiene, and the motion of the vessel predispose sailors to a number of disorders. Seasickness is the most common illness directly attributable to sailing (8%–15% of all illnesses) and is directly correlated to stormy conditions. Though almost never fatal, it is often incapacitating which can result in safety concerns among short-handled crews in heavy weather. Seasickness usually resolves after 3 days of a constant sea-state and its symptoms can be moderated or prevented by the use of medications which are most effective when taken before embarkation or at the very first onset of symptoms. Scopolamine, the most effective medication, has strong anticholinergic side effects and can cause urinary retention, psychosis, blurred vision, and dry mouth.

Among 360 novice, round-the-world sailors, dermatologic conditions accounted for 21% of medical cases. Sunburn, boils to the buttocks (from sitting on deck), cellulitis, and tinea infections featured prominently. Upper respiratory infections were reported to cause 18% of illnesses, and gastroenteritis, including a cluster of cases on one boat, caused 15% of illnesses.

### CONCLUSIONS

Although the majority of sailing injuries are minor and injury rates are low, the risk of death, predominantly from drowning, is higher than that of many land-based competitive sports. While many sailing-related injuries are soft tissue injuries that require nothing more than first aid, some are more severe. Among this latter group, some injuries could be prevented by more ergonomic sailboat design, use of gloves, and use of helmets. Fatal sailing accidents often occur when sailors unexpectedly find themselves in the water after falling overboard or capsizing. Falls overboard on keelboats can largely be prevented by wearing tethers which should be worn on-deck in stormy conditions, or whenever sailing at night. Dinghy sailors should be cautious when sailing in high winds, and wear insulating clothing in cold water with the expectation that they will capsize. Lifejackets should be worn when sailing, as there is compelling evidence that lifejackets save lives. A before-and-after study in Australia showed a significant decrease [U-26; p = .04] in boating-related drownings once lifejackets were mandated by law. Alcohol use, though likely underreported in many studies, has been found to be a contributing factor to drowning, sailing injuries, and sailing fatalities. Though current RI laws prohibit a boat operator to be intoxicated, the law should be broadened to include passengers, as they are just as likely to fall overboard and drown as is the skipper.

### References


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Accidental Hypothermia: ‘You’re Not Dead Until You’re Warm and Dead’

JOHN L. FOGGLE, MD, MBA, FACEP

INTRODUCTION
The classic teaching in medical school regarding hypothermia is “you’re not dead until you’re warm and dead.” More precise definitions than the medical school axiom exist. Hypothermia is a drop in core body temperature <35°C, or per history or trunk palpation when an initial core temperature measurement is unavailable due to cold exposure which, if not reversed, can lead to mental and physical impairment, hypoxemia, hypotension, acidosis, unconsciousness, arrhythmia, and death. Modern approaches to rewarming have improved survival from accidental hypothermia. This article reviews the epidemiology and classification of accidental hypothermia and reviews traditional and recently developed warming techniques that reduce morbidity and mortality in the setting of severe accidental hypothermia.

EPIDEMIOLOGY
The exact incidence of hypothermia deaths in the United States is unknown, and existing data is likely underestimated. The Centers for Disease Control and Prevention (CDC) reports nearly 17,000 hypothermia-related deaths in the United States between 1999 to 2011. Of the almost 1500 annual deaths, approximately 2/3 are male and nearly half of those who succumb are elderly. Fifty percent of deaths are from accidental causes. [1]

Common risk factors for accidental hypothermia include homelessness, poverty, mental illness, extremes of age, diabetes, cardiovascular disease, and alcohol and illicit drug use. Recreational activities related to climbing and skiing, resulting in cold exposure, and water sports, resulting in cold immersion, make up another large cohort of hypothermia cases.

CLASSIFICATION
There are a number of different classification systems used to organize the physiologic response to hypothermia. All are based on core temperatures, which can be determined by low-reading rectal or esophageal thermometers, or through epitympanic measurements. Epitympanic measurements may be considerably lower than the actual core temperature if the environment is very cold, or if there is water or snow in the external auditory canal. [2,3] Rectal temperatures can lag behind the true core temperature, especially in severe hypothermia. [4,5,6] The most accurate measurement, especially during rewarming, is an esophageal probe in the lower one-third of the esophagus, but this is only an option in an intubated patient.

The most common hypothermia classification system used in the United States is a three-stage system relying on the single lowest core temperature measurement:

- MILD 32–35°C (90–95°F)
- MODERATE 28–32°C (82–90°F)
- SEVERE <28°C (<82°F)

The Four-stage Swiss System [see Table 1] is used to estimate core temperature at the scene, with stages based on clinical signs that roughly correlate with the core temperature. [7] The Swiss classification splits the severe group into “unconscious” (24–28°C) and “no vital signs” (<24°C) and can be used to guide treatment once a core temperature is measured. One of the limitations of both the Swiss model, as well as other systems, is that there is much overlap and clinical findings may not correspond to classification. [8]

Each of the classification systems correspond to the physiologic response to hypothermia. In mild hypothermia, the initial response to a decrease in core temperature is peripheral vasoconstriction and increased metabolic heat production from shivering. [9] Cardiac output and respiratory effort

<table>
<thead>
<tr>
<th>Stage</th>
<th>Core Temperature</th>
<th>Clinical Findings</th>
<th>Treatment Approach</th>
</tr>
</thead>
<tbody>
<tr>
<td>HT-I</td>
<td>32–35°C</td>
<td>Conscious, shivering</td>
<td>Warm environment and clothing, warm liquids</td>
</tr>
<tr>
<td>HT-II</td>
<td>28–32°C</td>
<td>Impaired consciousness, not shivering</td>
<td>Active external, warmed fluids, minimally invasive internal rewarming</td>
</tr>
<tr>
<td>HT-III</td>
<td>24–28°C</td>
<td>Unconscious, vital signs present</td>
<td>All of the above plus airway control; consider ECMO or CPB if patient deteriorates</td>
</tr>
<tr>
<td>HT-IV</td>
<td>&lt; 24°C</td>
<td>No vital signs</td>
<td>Restore vital signs via CPR or defibrillation if possible, followed by ECMO or CPB</td>
</tr>
</tbody>
</table>

ECMO denotes Extracorporeal membrane oxygenation, CPB is Cardiopulmonary bypass, and CPR is Cardiopulmonary resuscitation
increase, as does oxygen consumption. When the core temperature drops below 32°C, metabolic activity decreases and bradycardia and diminished myocardial contractility is noted. Additionally, hypoventilation with concomitant carbon dioxide retention, hypoxia, and respiratory acidosis can occur.

As core temperature drops below 30°C, multiple organ systems are affected. Cardiac irritability results in dysrhythmias, and diminished brain metabolism results in increased irritability, confusion, apathy, and lethargy, and can proceed to somnolence and coma. [10] Other systems are impacted as well. Coagulopathy, renal dysfunction and cold diuresis occur, and endocrine and immunologic changes are seen in hypothermic patients. [9,11]

**MANAGEMENT OF ACCIDENTAL HYPOTHERMIA**

The rationale for aggressive treatment in accidental hypothermia is that the brain can tolerate cardiac arrest for up to 10 times longer at 18°C than at 37°C. [12] A full neurological recovery may be possible even after prolonged cardiac arrest as long as asphyxia does not precede the development of severe hypothermia. It is essential to recognize that unless there are obvious lethal injuries, a fatal illness, prolonged asphyxia, or if the chest is incompressible, survivability is possible in a hypothermic victim even when there are fixed pupils or early signs of rigor mortis.

**PASSIVE REWARMING**

The principles of treating mild hypothermia in victims who still have a carotid pulse and active respirations, [Swiss Hypothermia Stage I], with core body temperatures of 32-35°C, remain straightforward and uncontroversial. Such a patient is conscious and is likely shivering, although they may be lethargic and bradycardic. Passive external rewarming should begin in the field, even before emergency department arrival. It constitutes removing wet garments along with insulating the victim by using dry clothing, sleeping bags, or blankets. It is important to note that victims will continue cooling, which is known as “afterdrop,” after removal from a cold environment. This can result in a worsening Swiss Hypothermia Stage with life-threatening cardiac arrhythmias.

**ACTIVE EXTERNAL REWARMING**

Warm blankets and warm bags of saline especially to the core (axilla, back, chest, and groin), as well as warm (40°C), humidified oxygen delivery by mask, are the initial recommended emergency department treatments for Swiss Hypothermia Stages I and II. Impaired consciousness and the absence of shivering are expected in Stage II. Passive adjuncts like raising the ambient room temperature to >32°C and contact rewarming with a Bair Hugger® also facilitate external rewarming, while warm water immersion (40°C) is mostly impractical in an emergency department setting.

**ACTIVE INTERNAL REWARMING**

Active internal rewarming starts initially with warm parenteral fluids. Large volumes of IV fluids are essential during the rewarming process as vasodilation causes expansion of the intravascular space. It is at this stage, [Swiss Hypothermia Stage II], where cardiovascular instability may occur and the patient’s heart rhythm may progress from bradycardia to atrial fibrillation.

The first step in an unconscious patient who has vital signs [Swiss Hypothermia Stage III] is endotracheal intubation to protect the airway and to oxygenate the patient with warmed, humidified oxygen by ET tube (40–50°C). Nasogastric tube and bladder catheter lavage with 500cc NS at 40°C usually follows. Although there is a risk that overstimulation of an unconscious, severely hypothermic patient with endotracheal intubation may trigger ventricular fibrillation, the minimal risk outweighs the obvious benefits. [13]

Rhythm deterioration into ventricular fibrillation and asystole [Swiss Hypothermia Stage IV] will force a change in management. Defibrillation should be attempted, but it may not be successful until the core temperature rises to greater than 30°C. Similarly, the hypothermic heart may be unresponsive to cardiovascular resuscitation medications.

Centrally rewarming the heart helps prevent peripheral vasodilatation and cardiovascular collapse. Invasive treatments include delivery of warm fluids (1L NS at 40°C) via peritoneal lavage and/or via pleural lavage following bilateral tube thoracostomy, or with mediastinal irrigation after a thoracotomy. While case reports demonstrate successful resuscitations after prolonged CPR and active internal warming with peritoneal and pleural lavage, these techniques have shown relatively limited improvements in morbidity and mortality. [14,15,16]

Conventional techniques of rewarming are no longer the standard of care in level 1 trauma centers. Extracorporeal assisted warming (ECAR) has supplanted traditional methods of blood warming. ECAR, also called extracorporeal life support or ECLS, is a technology related to cardiopulmonary bypass (CPB), which was first utilized in 1953 by Gibbon to close an atrial septal defect in an 18-year-old. [17] ECAR has been successfully utilized in humans since 1967. [18,19] ECAR using extracorporeal membrane oxygenation (ECMO) provides vascular rewarming and augments oxygenation, ventilation, and cardiac output using portable mechanical circulatory support systems. Veno-arterial ECMO and CPB can raise body temperature by 6°C/hr and 9°C/hr, respectively, and have been shown to be much more effective than other rewarming techniques.[20] In one small subgroup analysis of patients suffering hypothermic cardiac arrest,
survival among those warmed with conventional techniques was 14%, while those undergoing ECAR had an over 80% survival rate. [21] ECMO has numerous advantages over earlier cardiopulmonary bypass approaches to rewarming: 1) it is usually instituted using femoral cannulation in severe hypothermia under conscious sedation; 2) ECMO may be used for greater than a week whereas CPB is usually used for only a few hours; and 3) ECMO allows time for recovery of the lungs and heart in severe hypothermia and appears to reduce ischemia-reperfusion injury and prevents diastolic dysfunction. [22, 23]

Veno-arterial ECMO, usually with deoxygenated blood from the femoral vein going through the circuit with oxygenated blood being delivered to the femoral artery, has become the preferred ECMO method over veno-venous ECMO when there is severe hypothermia and refractory cardiac arrest (see Figure 1). Percutaneous vascular access can be obtained during traditional CPR via femoral cannulae connected to a circuit that pumps blood through an oxygenator and heat exchanger, then back into the patient. It facilitates chest wall access for ongoing CPR, allows for cardiovascular support with a stepwise, swift, controlled increase in the patient’s body temperature, and it can be instituted rapidly using a groin incision in the emergency department. [23,24]

ECMO can be ended when the core temperature is greater than 37°C and there is a spontaneous, stable cardiac rhythm. It also can be terminated when there is failure to wean from ECMO, specifically when there is persistent asystole or refractory ventricular fibrillation and a core temperature of greater than 36°C. That patient is often defined as “warm and dead.” Patients who suffer cardiac arrest prior to hypothermia and those who asphyxiate, as well as those who are frozen solid and have a non-compressible chest, have very poor outcomes regardless of the means used to rewarmand, and they can be considered dead if they are without vital signs. While core temperature is not necessarily predictive of outcome, poor prognostic signs include potassium levels greater than 10 mmol/L in adults, and >12 mmol/L in children, severe acidosis (pH< 6.5), coagulopathy, as well as severe traumatic injury. [21,25]

CASE REPORTS OF SEVERE HYPOTHERMIA

The lowest recorded temperature [13°C] with full recovery involved a sea immersion of a 7-year-old girl in Sweden in 2011. [26] There are numerous other case reports, with age ranges from 2½ to 65, showing successful resuscitation with neurologically intact survival from Swiss Stage Hypothermia Class IV and with no vital signs initially and extremely low core temperatures after prolonged CPR. All made full recoveries after treatment and rehabilitation, overcoming enormous odds against survival. The following are several case reports of patients with extremely low core temperatures, all believed to be incompatible with possible survival (<20°C).

Case 1: Core temperature of 13.7°C in cold water immersion in Norway

A 29-year-old female skier fell into a waterfall gully flooded by icy water. She was lifeless for approximately 45 minutes. CPR and endotracheal intubation were initiated shortly after rescue. She had 9 hours of resuscitation and rewarming, with ECMO needed for 5 days, but had a full recovery. [27]
Case 2: Core temperature of 16.9°C in avalanche burial in Poland
A 25-year-old woman was buried under 40 cm of snow in a vertical position for nearly 2 hours, but was able to breathe. She had a GCS of 11 upon extrication, and then developed ventricular fibrillation (VF) cardiac arrest. Three unsuccessful shocks were delivered and manual CPR was started and continued during evacuation, followed shortly thereafter with endotracheal intubation, with persistent VF. ECMO was implemented upon hospital arrival and the patient was successfully defibrillated after rewarming to 24.8°C. ECMO support was required for 91 hours. [28]

Case 3: Median core temperature of 18.4°C in seven boating accident victims in Denmark
Thirteen teenagers and two adults, ages 15–45, were immersed in 2°C seawater after a boating accident. One drowned, and seven patients had severe accidental hypothermia circulatory arrest. All were successfully resuscitated using a treatment approach that included extracorporeal rewarming, followed by intensive neuro-rehabilitation. Seven other hypothermic victims, with core temperatures as low as 23°C, did not suffer circulatory arrest and survived the accident with non-invasive management. All of the survivors who received extracorporeal rewarming made near-complete recoveries except for one who has severe cognitive dysfunction felt to be the result of asphyxia from near-drowning prior to the onset of severe hypothermia. [29]

Numerous case series and reviews support these anecdotes and case reports and conclude that patients with severe hypothermia and cardiac arrest treated with extracorporeal rewarming have successful resuscitation rates of up to 50%. [30,31,32] Although there are no reliable tools available to predict who will eventually survive after ECLS, these case series and a recent meta-analysis suggest that success rates might be even higher if certain exclusion criteria are used to determine candidates for prolonged CPR and ECMO, including: 1) asphyxia that precedes severe hypothermia with cardiac arrest and 2) severe hyperkalemia (>12 mEq/L), as extremely high potassium levels have been associated with a poor prognosis and death. [33,34] The highest recorded serum potassium in a successful severe hypothermia resuscitation is 11.8 mEq/L.[35] Age greater than 65 has also been used as an exclusion criteria in some Level 1 Trauma Centers.

CONCLUSIONS
While the data to support when to use ECMO may be limited, data suggest: 1) the duration of CPR does not predict outcome; and 2) patients with core temperatures below 14°C have been and can be successfully resuscitated. Therefore, aggressive management with CPR and rapid extracorporeal blood rewarming is indicated in any patient who has a cold exposure or cold water immersion and presents to an emergency department in severe accidental hypothermia with no vital signs and no lethal signs of injury, and has no evidence that asphyxia preceded the hypothermia. That patient should be seen as having the potential for a full recovery. Unless a patient has findings incompatible with life, then the axiom is true: “You’re not dead until you’re warm and dead.”

References


A Free Diver with Hemoptysis and Chest Pain

CHANA RICH, MD; KRISTINA MCATEER, MD; VICTORIA LEYTIN, MD; WILLIAM BINDER, MD

Case Reports from the Alpert Medical School of Brown University Residency in Emergency Medicine

DR. CHANA RICH: Today’s patient is a 24-year-old man who presented to the Emergency Department (ED) with a cough and hemoptysis. The patient was freediving (breath-hold diving) in order to spearfish and had submerged to 50 feet while using an 11-pound belt. The patient ascended without expiration and developed chest pain. Upon surfacing, he coughed up approximately 5 tablespoons of bright red blood. After a brief rest on the boat, he dived to a depth of 30 feet in order to catch a large striped. Upon surfacing the second time, the patient had pleuritic chest pain and mild shortness of breath.

Due to his symptoms, the patient presented to the ED. He did not complain of headache, visual changes, ear pain, nausea, joint or muscle pain, and he had no additional episodes of hemoptysis. Vital signs revealed BP 127/68 mm Hg, heart rate 53, respiratory rate 16, and the patient had an oxygen saturation of 98% on room air. On exam, he was comfortable and non-toxic appearing and was in no distress. His lungs were clear to auscultation bilaterally; his cardiac exam revealed a normal s1s2 and he had a regular rate and rhythm with no murmurs. There was no crepitus. The remainder of his physical exam was unremarkable.

DR. WILLIAM BINDER: Free-dive spearfishing at 50 feet must cause significant pressure changes as our bodies are subject to almost 3 atmospheres of pressure. What disorders occur at increased atmospheric pressure?

DR. RICH: Spearfishing and other underwater activities may be done while freediving, snorkeling, or scuba diving. Our patient and his friends were freediving, or breath-hold diving. Unlike scuba diving, breath-hold divers do not use supplemental air underwater. Divers face a unique set of underwater hazards in addition to the general aquatic problems such as drowning, hypothermia, water-borne infectious diseases, and interactions with hazardous marine life. When diving deep, free divers are exposed to increased pressure, causing a spectrum of injuries to the body.

Pressure contributes either directly or indirectly to the majority of serious diving-related medical problems. As a diver descends underwater, absolute pressure increases much faster than in air. The pressure change with increasing depth is linear, although the greatest relative change in pressure occurs nearest the surface, where it doubles in the first 33 feet of sea water from 1 to 2 atmospheres of pressure. The body behaves as a liquid and follows Pascal’s law; pressure applied to any part of a fluid is transmitted equally throughout the fluid. When a diver submerges, the force of the tremendous weight of the water above is exerted over the entire body. The body is relatively unaware of this change in pressure.

While this is true of the body, the spaces within the body containing gas (air), including the lungs, sinuses, intestines, and middle ear, follow a different law. The gases in these spaces obey Boyle’s law; the pressure of a given quantity of gas at constant temperature varies inversely with its volume. Therefore, as one dives deeper, the volume of air in the middle ear, sinuses, lungs, and gastrointestinal tract is reduced, but upon ascent, the volume expands. Inability to maintain gas pressure in these body spaces equal to the surrounding water pressure leads to barotrauma.

Barotrauma can potentially involve any area with entrapment of gas in a closed space. In addition to sinuses, lungs and the GI tract, barotrauma can occur in the external auditory canal, in the teeth, the portion of the face under a face mask, and skin trapped under a wrinkle in a dry suit. The tissue damage resulting from such pressure imbalance is commonly referred to as a “squeeze.”

Illustration 1. Pascal’s Law: pressure applied to any part of a fluid is transmitted equally throughout the fluid.
Source: https://upload.wikimedia.org/wikipedia/commons/thumb/2/27/Pascals-law.svg/2000px-Pascals-law.svg.png
DR. ELIZABETH SUTTON: How does this account for the patient’s hemoptysis?

DR. RICH: There are two possibilities accounting for the patient’s chest pain and hemoptysis. Pressure-related injury to the lung can occur on descent, or as a diver ascends to the surface. If one were able to completely exhale, the absolute minimum lung volume remaining is called the residual volume (RV). Lung squeeze occurs when the diver descends to a depth at which the total lung volume is reduced to less than the residual volume. The increased pressure on the pulmonary vascular bed can damage the integrity of the pulmonary capillaries. At this point, transpulmonic pressure exceeds intraalveolar pressure, leading to transudation of fluid or blood from ruptured pulmonary capillaries. [1] Patients can exhibit signs of pulmonary edema and hypoxemia. This is the reason that many free divers cough up blood after a deep dive, although there are a number of case reports of lung squeeze with repetitive shallow dives with brief surface intervals. [2]

Despite this presumed mechanism of barotrauma of descent, free divers are able to dive to depths beyond those that should cause mechanical damage to the lungs. Other physiologic mechanisms must play a role, although the exact pathophysiology of this condition remains unclear. When diving deep, the chest cavity itself gets smaller and there is central pooling of blood in the chest from the surrounding tissues. The central pooling of blood in the chest equalizes the pressure gradient when the RV is reached and thereby decreases the effective RV. These mechanisms allow the lungs to be compressed down to about 5% of total lung capacity in highly trained breath-hold champions. [3] An individual’s anatomy, physiologic reserves, underlying pathology and the conditions of the day all play a role in the development of pulmonary barotrauma. [3]

Alternatively, and perhaps more likely, the patient suffered alveolar injury upon ascending. As a diver ascends, the pressure within the alveoli of the lung increases as the pressure around the diver decreases. If intrapulmonary gas is trapped behind a closed glottis, as the diver ascends and the surrounding pressure decreases, the volume of the intrapulmonary gas increases in accordance with Boyle’s law. Increased pressure within the lung causes an increase in trans-alveolar pressure leading to overexpansion injury, alveolar rupture, and intraparenchymal hemorrhage. [4] A situation of rapid ascent to the surface, such as if a diver runs out of air, panics, or drops his weights, is often the cause of pulmonary barotrauma of ascent. Divers who hold a breath as they ascend and those with obstructive airway diseases, such as asthma or chronic obstructive pulmonary disease, are at increased risk. This was likely the case with our patient – he did not exhale and relieve the building pressure as he ascended, causing his pulmonary barotrauma.

DR. KRISTINA MCATEER: What were your next diagnostic steps for this patient?

DR. RICH: Laboratory studies were normal and an ECG revealed sinus tachycardia without signs of ischemia or right heart strain (no ST depression or T-wave inversions in V1-V4). The chest X-ray [Figure 1] reveals bilateral, patchy, airspace disease.

DR. JAMES RAYNOR: Did the patient require positive airway pressure or other airway management?
DR. RICH: The patient was placed on supplemental oxygen and was admitted to the ICU for observation and supportive care. A CT scan [Figure 2] was performed in order to evaluate for any underlying pulmonary parenchymal disorders and a repeat chest X-ray was obtained the following morning which demonstrated no appreciable change.

DR. OTIS WARREN: It appears that this patient did quite well. However, did he risk other consequences from his pulmonary barotrauma?

DR. RICH: Yes, this patient was quite fortunate. The intrapulmonary pressure can become so elevated that air is forced across the pulmonary capillary membrane. Pulmonary interstitial air can dissect along bronchi to the mediastinum causing pneumomediastinum. Air from the mediastinum can track down the esophagus and great vessels causing retroperitoneal emphysema or pneumoperitoneum. [5] Mediastinal air can track superiorly to the neck, resulting in subcutaneous emphysema. Rarely, air may reach the visceral pleura, causing a pneumothorax.

One of the more concerning consequences of pulmonary barotrauma is due to an air embolism. If air enters the pulmonary vasculature, it can travel to the heart and embolize to other parts of the body. While a frequent cause of air embolism is iatrogenic secondary to interventional procedures, many other causes, including blunt and penetrating trauma, childbirth, and diving can lead to an air embolism. Clinical manifestations and severity of illness can range from asymptomatic to sudden onset circulatory collapse and are dependent upon the amount of air as well as the location of the air bubble. [6]

In our patient, due to his chest pain and hemoptysis suggesting alveolar injury, air embolism was a primary concern. Venous air embolism is less likely to cause severe symptoms. However an arterial gas embolism [AGE] can be devastating. Approximately 4% of divers who suffer an arterial gas embolism die immediately from total occlusion of the central vascular bed. [7,8] Furthermore, AGE patients who survive and present to the hospital frequently suffer neurologic deficits. Air entering the pulmonary vasculature can result in a cerebral air embolism [CAE] which can cause seizures, stroke, and death. [9]

DR. VICTORIA LEYTIN: What treatments are available for patients suffering from an air embolism?

DR. RICH: All cases of AGE must be considered for hyperbaric oxygen treatment as rapidly as possible. Treatment is required even if manifestations resolve prior to reaching an ED in order to prevent progression of subtle neurologic deficits that are not immediately detected. [10] Our patient showed no signs of right heart strain and had no neurologic deficits. He remained hemodynamically stable and had no respiratory distress. He was discharged on hospital day 2. The patient had no shortness of breath and was at his baseline after 2 weeks, and a follow up X-ray at that time revealed complete resolution of the bilateral infiltrates.
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Learning from the Outside In: Incorporating Wilderness Medicine into Traditional Emergency Medicine Education

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KEYWORDS: Wilderness Medicine [WM], outdoor education, resident education, wellness, emergency medicine

INTRODUCTION

Experiential education is a well-established method of learning, in which learners are purposefully engaged in direct experiences and focused reflection to promote the development of meaningful knowledge, skills and values essential to their jobs and place in the community.1 The link between experience and education was formally introduced and written about by John Dewey in the early 20th century, but one of the most well-known leaders in this field was Kurt Hahn, the founder of Outward Bound. Even into the present day, Outward Bound programs revolve around wilderness activities, such as hiking, mountaineering, and paddle sports, among many others. Hahn used the principles of experiential education as the cornerstone of these experiences, hoping the endeavors would build leadership among its participants, develop them as a whole person, and create citizens who could make right, just, and independent decisions.2

Wilderness and austere medicine [WM] is the discipline of medicine characterized by the provision or improvisation of patient care in the remote setting, or when resources and manpower are limited.3 This encompasses not just the provision of care at altitude or in the backcountry, but travel and expedition medicine, dive medicine, military medicine and EMS/search and rescue. WM has developed over the last few decades into an academic specialty in its own right with societies, conferences, research base and journal, textbooks, certifications and fellowship training. WM is a recognized sub-specialty of emergency medicine with 15 fellowships as of 2017.4 While often associated with emergency medicine, the principles of WM are multi-disciplinary and come from the fields not only of emergency medicine but surgery, toxicology, sports medicine, military and travel medicine, to name a few. Practitioners of WM come from all medical backgrounds and practice in a variety of settings, limited not just to backcountry travel but international travel, disaster and relief work, cruise ship medicine and EMS. WM education focuses on the gaps in traditional medical curriculum and relies heavily on out-of-classroom coursework and experiential education.

Experiential learning has formed the foundation of most WM courses [e.g. Wilderness First Responder, Advanced Wilderness Life Support, etc.] through the use of standardized patients, simulation dummies, and moulage. Such methods are essential because WM typically involves small numbers of patients and rarely encountered scenarios. Whereas traditional medical education provides ample opportunity to manage conditions like heart failure and asthma in the hospital, there are
very few, if any, opportunities to manage patients with wilderness-specific conditions such as envenomations, or to treat patients in an austere environment. WM also encompasses many sub-disciplines of emergency medicine, including toxicology, disaster medicine, and EMS/pre-hospital care, which often involve scenarios not usually encountered during typical emergency medicine training. Most providers are not involved in the medical decision making or practical aspects of patient evacuation, especially the lengthy and arduous transports needed for remote patients.

Over the past two decades, the Medical Wilderness Adventure Race (MedWAR) has become an increasingly popular way for medical providers to expand their knowledge base and put their wilderness/austere medicine knowledge and skills to the test. Since the early 2000s, these races have brought the simulated scenarios of WM courses into the context of a competition. Race participants engage in various outdoor activities, such as orienteering, paddling, and biking, while being intermittently challenged with a variety of WM situations that encompass the entire breadth of wilderness practice. With its inception, there was also the hope that the MedWAR races would lead to interagency alliances, including search and rescue (SAR) teams, park rangers, outdoor activity guides, and WM education organizations.

A two-year retrospective in 2003 noted that participants felt these races provided not only a boost in medical confidence, but also engendered teamwork and effective communication. Interestingly, while a number of racers felt that they had somewhat increased their knowledge base, they felt strongly that the experience itself was more important for their growth as medical professionals. Other lessons learned included difficulties with the written portions of the race and comments on the physical activities as being either too difficult or easy, and the medical challenges too complex or simple (even within the same race). In short, these races offer unique opportunities for providers to practice in a pressured, time-sensitive situation, acquire new skills, and develop relationships that are essential to providing timely, high-quality care.

**THE BROWN WILDERNESS MEDICINE COMPETITION**

The Warren Alpert Medical School of Brown University has a four-year emergency medicine residency. All accredited residency programs have an Accreditation Council for Graduate Medical Education (ACGME) mandate for resident education, which is met predominantly through weekly conferences and didactic education, although there is significant variation among different programs. In an effort to promote resident education and engagement, one conference day per month is spent in the simulation center. This offers residents a chance to develop life-saving skills in an appropriately controlled setting, often with rarely encountered conditions and scenarios. In 2017, a group of residents at Brown developed an off-campus wilderness medicine event adapted from the MedWAR model, to replace a monthly simulation day.

This race was established with five main goals: 1) Increase learner engagement through experiential learning. 2) Exposure to the basic tenets of wilderness medicine. 3) Development of relationships across multiple learning levels, including medical students, residents, and attending physicians. 4) Deepen relationships between our program and local EMS and SAR resources. 5) To promote resident wellness through engagement in wilderness activities.

**HOW THE RACE WORKS**

For both 2017 and 2018, the competition has been held at Lincoln Woods State Park (LWSP) in Lincoln, RI. LWSP was chosen for its close proximity to the main residency teaching site and many options for outdoor activities, including hiking/running, rock climbing, paddle sports and mountain biking – perfect for the physical variety that is key to these wilderness medicine races. Residents, mid-level providers and medical students are split into teams of four and given a
very basic map of the park with the general location of 10–12 “stations,” multiple choice questions and a variety of physical challenges. They must decide as a team how to find and complete as many stations and questions as possible within the allotted time.

Teams are required to plan and prepare a pre-packed medical kit, which must include all equipment, supplies, and medications (pre-labeled syringes/bottles) they anticipate requiring to complete the stations as well as to care for real-world minor injuries. Following the competition, a debrief cookout allowed participants and facilitators to reflect on the experience, lessons learned and discuss the details of the scenarios. Small prizes were awarded to the top three teams.

The core of the event is the stations, each of which presents participants with a hands-on austere-medicine scenario. These stations are created and run by attending emergency medicine physicians and local EMS personnel, often with the assistance of medical student volunteers. Examples of scenarios from this past year included a drowning requiring water rescue, a penetrating eye injury while skiing, a fall on steep terrain actually requiring low-angle rescue by a high-angle tech rescue team, and a crush injury requiring extrication and management of a threatened limb. These stations are designed to have five critical actions, each of which must be completed to get full marks, one point for each critical action. For example, the drowning requiring water rescue station required:

1) Evaluate for the scene safety: Is it OK to enter water and rescue victim?
2) Improvised spinal immobilization
3) Control bleeding from leg injury w/improvised tourniquet
4) Splint fracture
5) Treat for hypothermia

Additional points were awarded to the teams that completed their stations the fastest.

For the written portion of the competition, multiple choice questions on a variety of wilderness/austere medicine topics were placed along the course. Many questions utilized visual stimuli. Example questions included picture ID of poisonous plants and physical exam findings and wilderness survival knowledge.

INTEGRATION OF LEARNING AND WELLNESS
Overall, the goals set forth by organizers were accomplished; learners were engaged in the outdoor classroom and exposed to a variety of austere scenarios including search and rescue and tactical medicine. There was a great deal of engagement and camaraderie between EMS and physicians/learners and wellness was promoted in the pursuit of outdoor recreation and physical activity, teambuilding and friendly competition.

A cornerstone of the Brown Wilderness Medicine Race is integration of senior and junior providers/students within teams and scenarios, as well as with EMS personnel and EM faculty in unexpected and unfamiliar scenarios. Whereas typical simulation days are held indoors in a controlled environment, this race enabled cross-functional interaction during stressful medical scenarios. For example, during the low-angle rescue scenario mentioned above, patient was in a simulated ravine, unable to be initially assessed by the physicians. Personnel from the Fire Department taught and assisted residents with harness and safety gear, and provided a safe way for them to reach the patient and begin evaluation. The residents had to begin the scenario with remote patient evaluation as they awaited safe transport to the patient. During the debrief, participants had the opportunity to ask questions about the intricacies of technical rope rescue, Fire Department experience with such rescues and discuss how knowledge about the extrication could improve hospital care. The Fire Department also learned from the providers and were given hands-on instruction on patient care and assessment during the scenario. Both parties reported a great deal of benefit from working together and learning a different perspective on the scenario. EMS work and rotations notwithstanding, there are few times during training where residents may have access to such insight.

Another benefit of the model was the interaction between
LEARNERS AND FACULTY, which were both EM attendings and senior EMS providers. The MedWARs event facilitated both casual interaction and more camaraderie than is usually found in the hospital setting. Additionally, EMS providers and physicians readily interacted and learned from each other during the training event, which is uncommon in traditional residency training and was described in feedback to be mutually beneficial.

From a health and wellness standpoint, the Brown Wilderness Medicine Race allowed all involved to break out of their usual routines, the demands of which often lead to stress. Burnout is prevalent among residents, and is particularly high among emergency physicians. In a prospective study, 45% of 2nd year residents report some symptoms of burnout, and 65% of attending emergency physicians reported symptoms of burnout, often associated with unhealthy coping behaviors such as overeating, alcohol, and drug use.7,8

Outdoor activities often provide an important outlet for health care providers, who often pursue adventurous endeavors outside of the hospital. Regular outdoor activity – even just walking – has shown a variety of health benefits, including weight management, blood pressure control, and lowering the risk of vascular catastrophes, such as myocardial infarction and stroke.9 Increasingly, interventions are sought by health care organizations and residency programs to promote wellness and reduce burnout and stress.10,11 The MedWAR race enabled attending physicians and residents to engage in, and share, their knowledge regarding activities they found personally and professionally rewarding. Simultaneously, the race contributed to the participant’s work-life balance, an essential component of a long and healthy career in medicine. Gawande has reported on the deleterious effect of computers impact on the physician’s work day; experiential learning may be an antidote to physician burnout and stress.12

LESSONS LEARNED

The response to the first Brown Wilderness Medicine Race in 2017 was very positive. Participants greatly valued the realism of the faculty-developed scenarios and enjoyed learning outside of the confines of weekly conference and the simulation center. As with the MedWAR races, there was also a general feeling that the experience itself was as important as the educational aspect of the day. The race also did a great job of fostering provider bonding and camaraderie.

In 2018, the race was run in a similar manner, with some changes based on first-year course feedback. Improvements included assignment of a starting station expanding the course to different areas of the park. In the 2018 race, local EMS agencies created and ran stations and incorporated technical rescue components run by the Fire Department to foster additional exposure to the unique pre-hospital environment. The physical aspects of the race were extended by increasing the distance between some stations. Feedback regarding the physical activity part of the race was mixed; some groups were very tired by the end, whereas others wanted even more of a physical challenge.

The addition of technical rescue also had some unintended consequences including longer wait times for scenarios and more down time for both teams and faculty. Participant teams were found to cluster around the technical stations, both because they took longer to complete and because teams were very keen on completing those stations. While it was not expected that all teams complete all stations, this did result in inequity felt amongst faculty at less popular stations. Setting clear expectations for faculty including expected down time proved very important to keeping faculty both engaged and feeling appreciated for time spent.

CONCLUSIONS

With the overwhelmingly positive response from these first two years, the Brown Wilderness Medicine Race has become a permanent yearly fixture in the educational curriculum. Regarding race specifics and logistics, we fully expect this to vary year-by-year, not only because race leadership will turn over, but also because feedback will continue to vary. A key difference between this race and the MedWAR races is that all residents who would normally attend weekly conference are expected at this event. Whereas MedWAR self-selects participants who have a specific interest in wilderness medicine and physical activity; these might not be important to the participants in the Brown Wilderness Medicine Race. Based upon all of these variables, it will be difficult to develop an experience that caters to all tastes, but perhaps the most important aspect of the race is that it develops teamwork and communication skills necessary during all medical scenarios.
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Climate Change: A Review of a Public Health Opportunity for the Northeast

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KEYWORDS: climate change, public health, heat illness, global warming

INTRODUCTION

The connection between climate change and health has been called the greatest public health threat of our time.1 The Northeast (NE) is especially vulnerable. Average temperatures in the NE are expected to rise >3.6°F (2°C) higher than in the pre-industrial era and far more quickly than in the other contiguous states in North America. The region faces higher sea level rise and worse flooding than other regions of the United States (U.S.) with negative human health and economic impacts in the region. At present, the key mitigating mechanisms to ameliorate the health effects of climate change is through decreasing greenhouse gas (GHG) emissions and developing evidence-based adaptation interventions.

STATE OF CLIMATE CHANGE IN 2018

The consensus among 97% of climate scientists is that climate change is occurring and is human caused.2 Most contrarian research has significant methodological flaws.3 The planet is already 1.8°F (1°C) warmer than the pre-industrial baseline. Meanwhile, 2018 is on track to be the fourth hottest year on record, surpassed only by the preceding three years. The warmest 20 years have occurred in the past 22 years.4

Climate change leads to the following exposure pathways, among others: extreme heat, rising sea levels, extremes of precipitation, and more extreme weather [Figure 1].5-7 The U.S. had 16 billion-dollar weather and climate disasters in 2017 with an estimated total of $313 billion dollars in damages.5 Worldwide, a number of catastrophic extreme weather events are related to climate change.6

During the 2016 Paris Agreement, 194 countries joined together to declare action on climate change. This body reconvened in December 2018 at the 24th Conference of the Parties (COP24) to outline the specifics of implementing the reduction of GHGs. While a consensus was reached, the current committed action will not be sufficient to reduce global GHG emissions to a level that will keep the planet below the necessary 2.7°F (1.5°C) as outlined by a special report in October 2018.9 This report by the Intergovernmental Panel on Climate Change (IPCC) gave an urgent call that global GHG emissions must be cut in half by 2030 to attempt the goal of 2.7°F (1.5°C). We are currently on track for 5.4°F (3°C).

The healthcare system is part of the problem given the energy intensity of immense 24-7-365 facilities. The U.S. health sector is responsible for approximately 10% of U.S. GHG emissions.10 It has been estimated that the mortality burden from the U.S. health sector’s air pollution production is equal to the mortality burden of medical errors.11

CLIMATE CHANGE AND HEALTH

Climate change leads to a broad range of negative health impacts [Figure 1].12 Recent emerging health concerns, such as an association between increased heat and microbial

Figure 1. Sample Climate Change Exposure Pathways, Mediators, and Health Implications for the United States (figure created by R. Salas).
resistance to antibiotics, reveal that our understanding of these health risks is still in its infancy. Importantly, climate change disproportionately harms the health of the most vulnerable, including children, the elderly, and those with chronic medical conditions, as well as people of lower socioeconomic classes. Recent reports from key agencies, such as the Lancet Countdown, World Health Organization (WHO), and U.S. Global Change Research Program (USGCRP), outlined these health impacts globally and for the U.S.\textsuperscript{5–7,13}

The Lancet Countdown is an interdisciplinary research consortium tracking the impact of climate change on health in 41 indicators over five domains.\textsuperscript{6} It also stresses the potential for cascading disruptions to public health infrastructure that may overwhelm existing health services. The WHO report highlights that air pollution from the burning of fossil fuels leads to seven million deaths annually, suggesting that climate change is a public health emergency.\textsuperscript{11} Worldwide, the U.S. has the second highest carbon emission from the burning of fossil fuel, creating disproportionate health impacts in developing countries.\textsuperscript{14,15}

Two recent reports stress that the health impacts of climate change are not only occurring in distant lands, but also in the U.S. The companion 2018 Lancet Countdown Brief for the U.S. highlights that Americans have increased exposure to more frequent and longer heat waves, increasing extreme weather, and worsening climate-sensitive vector-borne diseases.\textsuperscript{5} In addition, the USGCRP’s second volume of the National Climate Assessment, released in November 2018, also outlines that the health of every American is at risk and emphasizes the unique geographic vulnerabilities.\textsuperscript{7}

**THE NORTHEAST: UNIQUE CLIMATE CHANGE AND HEALTH CHALLENGES**

**Extreme Heat**

The NE is predicted to have the largest temperature increase for the contiguous U.S. – nearly 3.6°F (2°C) warmer by 2035 and over 5°F (2.8°C) by 2050 compared to present day averages.\textsuperscript{7} This temperature rise will be nearly 20 years ahead of the predicted global rise to 3.6°F (2°C).\textsuperscript{14} therefore, benefit from understanding regional consequences of limiting the global mean temperature increase to well below 2°C above pre-industrial levels, a limit agreed upon at the United Nations Climate Summit in Paris in December 2015. Here, we analyze climate model simulations from the Coupled Model Intercomparison Project Phase 5 (CMIP5 Annual temperatures in New England have already increased by about 3°F (1.7°C) since the beginning of the 20th century.\textsuperscript{7} The seasons in the NE are also becoming less distinct as the winter months are warming three times faster than summers, while the health risks for heat appear to be most significant at the start of the summer. In addition, those residing in cities are especially at risk due to the phenomenon of urban heat islands – areas of increased temperatures in urban areas in comparison to outlying suburban, exurban and rural locations.

The connection between extreme heat and health is well established, as elevated temperatures can lead to heat stroke, cardiovascular, renal, and respiratory disease, worsening mental health issues, and adverse birth outcomes.\textsuperscript{5,7,12} One study of Rhode Island (RI) found increased morbidity, mortality, and emergency department (ED) utilization with increased temperatures.\textsuperscript{17} The increase in ED visits occurs rather abruptly when maximum ambient temperature is > 80°F. Between 2005–2012, all cause ED visits increased by 1.3% with the strongest association at the extremes of age. Heat-related ED visits increased by 23.9% when the daily maximum temperatures increased by 10°F (from 75°F to 85°F), while all cause mortality increased by 4%. It is predicted that the NE will experience 650 excess deaths annually by 2050 and then upsurges from approximately 960 in a moderate climate model to 2,300 excess deaths in the extreme model per year by 2090 from heat.\textsuperscript{7}

**Ocean Warming**

The ocean is warming three times faster along the Northeast Continental Shelf compared to the global rate, with the Gulf of Maine increasing 99% more than the global average the last decade.\textsuperscript{7} In 2012, the NE experienced the most intense warming event as the ocean rose to 3.6°F (2°C) above average. As ocean temperatures increase, conditions for harmful algae blooms are growing more favorable and have been increasing in both frequency and duration in the Gulf of Maine.\textsuperscript{7} Conditions for Vibrio are also more favorable and have expanded to the north. Both of these potentially leave more Americans exposed to toxins and bacteria through direct contact or ingestion of contaminated seafood.\textsuperscript{5,7}

**Sea Level Rise and Extreme Weather**

Ocean levels are not rising equally due to a variety of factors, and the NE has experienced some of the highest rates of sea level rise.\textsuperscript{7} It is predicted that by the end of the century, sea levels could rise to more than 11 feet on average in this region. As sea levels rise, high tide flooding has increased by 100–200% in some areas with more nuisance flooding. Over the past 20 years, the NE has also experienced some of the largest increases in precipitation during the spring and fall seasons.\textsuperscript{10} Increased precipitation and flooding risks, coupled with more frequent power outages, create ideal mold conditions, which has implications for respiratory conditions.\textsuperscript{7}

The NE experiences nearly year-round storms from Nor’easters, occurring September to April, and from Atlantic hurricanes, occurring June to September.\textsuperscript{7} This is especially concerning as Atlantic hurricanes are of higher intensity and produce increased precipitation.\textsuperscript{10} Extreme weather can result in healthcare issues related to direct trauma and disruptions in the healthcare system.\textsuperscript{11} For instance, due to the aging infrastructure of NE urban centers, the region is
increasingly vulnerable to flood dynamics. It is predicted that climate-related hazards may cause upward of $11–17 billion dollars per year of damage.7 Many northeast cities – Boston, New York, Philadelphia, Portland, Providence, and others – have begun to plan for climate change as they seek to upgrade aging infrastructure, including water systems. Many NE cities have combined sewer systems, which can lead to drinking water contamination and subsequent gastrointestinal illness during flood events. Massachusetts has seen an increase in gastrointestinal illness related to heavy rains and runoff and Maryland has noted increased cases of Campylobacter and Salmonella during heavy precipitation.19, 20 These vulnerabilities reinforce that healthcare systems must be prepared for contingencies and remain resilient during extreme weather.

**Air Degradation**

Air quality has improved in the NE over recent decades, yet climate change threatens to unravel this achievement. For example, as the NE experiences higher temperatures, it will worsen ground-level ozone concentrations and lead to hundreds of excess deaths by 2050 from the baseline estimate in 2000.7 While the NE is immune to wildfires, climate change is intensifying wildfires in the western U.S. and Canada, and smoke can travel thousands of miles. Worsening air quality in the NE from increased smoke particulate matter, worsening pollen levels due to higher CO2 levels, and increased ozone all pose significant health risks, especially for those with underlying lung pathology.

**Climate-sensitive Vector Borne Diseases**

As the conditions for vectors, especially arthropods, are altered by climate change, the risk of transmission to humans is heightened. The ticks and mosquitoes known to transmit two key climate-sensitive vector-borne diseases in the NE, Lyme disease and West Nile virus, are expected to continue moving further north.7 By 2065–2080, the transmission of Lyme in the NE could start nearly three weeks earlier in Maine and Pennsylvania (compared to baseline 1992–2007). Cases of neuro-invasive West Nile may increase by 210 to 490 cases per year.

**OPPORTUNITIES TO MINIMIZE MORBIDITY AND MORTALITY IN THE NORTHEAST**

**Mitigation**

The reduction of GHG emissions, notably through converting from fossil fuels to renewable energy sources like wind and solar, can positively impact healthcare by reducing air pollution and minimizing climate change. In a best-case scenario, mitigation can result in a moderate climate change model and could save $21 billion and prevent nearly 1,400 premature deaths annually from heat alone. Given the current lack of federal commitment to this issue, action will likely reside on local and state levels. However, the current local and state commitments are only meeting half of the U.S. Paris Agreement commitments.5 While states in the NE are reducing the carbon intensity of the energy system [Figures 2 and 3]21, urgent and aggressive action is needed in order to maintain global temperature rise below 2.7°F (1.5°C).9

The health sector can assume leadership in the field by modeling the behavior needed to reduce morbidity and mortality. Healthcare systems can honor U.S. Paris Agreement commitments and reduce GHG emissions by reducing...
fossil fuel use. A physician messenger transmitting the key message that climate change is first and foremost a public health issue has been found to increase the engagement of the public and depoliticize it.10

Adaptation
Adaptation interventions can help minimize the harm climate change is causing to health. Philadelphia and New York City have implemented policies and alterations in its infrastructure, including opening cooling centers, protecting utility services from overload, and planting over one million trees to develop an urban canopy – that will reduce mortality from heat.22 In addition, health systems must focus on climate resiliency to ensure that these systems can face the numerous challenges of climate change as it threatens to disrupt efficiency, infrastructure, supply chains, and function.

CONCLUSION
Climate change is the greatest public health opportunity of this generation. Advocating for renewable energy and research into evidence-based adaptation interventions is in line with the Hippocratic oath. There is nothing political about protecting the health of patients.

References

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