Hawai‘i Journal of Medicine & Public Health
A Journal of Asia Pacific Medicine & Public Health

November 2014, Volume 73, No. 11, Supplement 2, ISSN 2165-8218

WILDERNESS MEDICINE IN HAWAI‘I AND THE PACIFIC

OUT OF THE WILDERNESS: FLIPPING THE CLASSROOM TO ADVANCE SCHOLARSHIP IN AN INTERNAL MEDICINE RESIDENCY PROGRAM 2 Dale S. Vincent MD

HIGH ALTITUDE ILLNESSES IN HAWAI‘I 4 Terry Shin MD

IS HIGH ALTITUDE PULMONARY EDEMA RELEVANT TO HAWAI‘I? 7 Seth Lewis Cornell MD

PREVENTION AND TREATMENT OF INJURIES FROM CAVE EXPLORATION IN HAWAI‘I 10 David W. Cowart MD; John B. Halleck BS; and Benjamin R. Park DO

THE RISKS OF SCUBA DIVING: A FOCUS ON DECOMPRESSION ILLNESS 13 Jennifer Hall DO

HAZARDS OF HAWAI‘I VOLCANOES NATIONAL PARK 17 Gregory M. Sprowl MD

LEPTOSPIROSIS: THE MICROSCOPIC DANGER IN PARADISE 21 William A. Londeree MD

CIGUATERA FISH POISONING IN HAWAI‘I AND THE PACIFIC 24 Nathanial K. Copeland MD; Wyatt R. Palmer DO; Paul K. Bienfang PhD

THE POTENTIAL DANGER OF EATING WILD LETTUCE: A BRIEF REVIEW OF HUMAN RAT LUNGWORM INFECTION 28 Evan C. Ewers MD and Sarah K. Anisowicz MD

HEAT ILLNESS IN HAWAI‘I 33 Sarah Gordon MD

FOREIGN BODY SYNOVITIS IN THE PACIFIC 37 Caleb Anderson MD; Rodger Stitt MD; and Jefferson Roberts MD

CENTIPEDE ENVENOMATION: BRINGING THE PAIN TO HAWAI‘I AND PACIFIC ISLANDS 41 Joshua L. Fenderson MD

TROPIC LIGHTNING: MYTH OR MENACE? 44 John McCarthy MD
Out of the Wilderness: Flipping the Classroom to Advance Scholarship in an Internal Medicine Residency Program

Dale S. Vincent MD

Abstract
Residents in an internal medicine residency program “flipped the classroom” in a series of learner-centered activities which included the creation of a medical student interest group, a continuing medical education symposium, and a journal supplement focused on wilderness medicine topics in Hawai‘i and Asia Pacific. The project encompassed both scholarly activities (discovery, integration, application, and teaching) as well as scholarship (writing for publication). The project advanced the professional formation of residents by developing competencies and producing outcomes that are key features of the ACGME Next Accreditation System.

Introduction
This Wilderness Medicine Supplement started with a brainstorming session with residents just before the start of the academic year. How do we encourage scholarly activity and also produce more tangible scholarship? Indeed, the Next Accreditation System (NAS) of the Accreditation Council for Graduate Medical Education (ACGME) has begun to shift from emphasizing structure and process (scholarly activities) to outcomes (peer-reviewed publications).1

Here was the idea: focus on activities that the residents have learned to love by living in Hawai‘i, and create a collection of scholarly reviews of common and not-so-common wilderness medicine topics for the community. Several residents had already established a popular Wilderness Medicine Interest Group at the University of Hawai‘i John A. Burns School of Medicine. The supplement would be a natural fit in the truest sense.

Professor Ernest Boyer reimagined the range of scholarly activities in medicine to encompass the elements of discovery, integration, application, and teaching.2 By discovery, he meant a disciplined, methodical pursuit of new knowledge. Integration refers to the reworking of isolated facts to present a coherent, illuminating narrative. The scholarship of application applies knowledge to important social and institutional problems. Teaching transmits knowledge, and also transforms knowledge in creative and stimulating ways. Medical professional organizations such as the Association of Professors of Medicine have embraced this broad definition of scholarly activity,3 and participation in scholarly activities has been an integral feature of residency training.

The ACGME has recently begun to ask residencies to report the outcomes of scholarship in residency programs.4 By scholarship, the ACGME means the creation of published works in the peer-reviewed medical literature. Internal medicine and other primary care residents already have grueling schedules and a daunting body of knowledge to master during their short, three-year residencies, so this new expectation represents a special challenge.4

In order to overcome barriers to participation in this project, we identified and leveraged several sources of motivation for the residents. Some residents are motivated to publish by a desire to be more competitive for employment and fellowship applications, an extrinsic source of motivation. We believed that we could also harness powerful internal motivation by having the residents choose fun and interesting subjects with which they were already familiar.

Thus, the supplement became an example of a “flipped classroom.” The “flipped classroom” has been described as a “disruptive” educational model in which learners acquire knowledge outside of the classroom, and consolidate that knowledge during mentoring that takes place inside the classroom.5 The characteristics of the flipped classroom have been succinctly summarized as: a flexible learning environment, a learner-centered culture, intentional content, and guidance from a professional educator.6 This Wilderness Medicine Supplement exemplifies all of these characteristics.

Residents developed new skills and consolidated their learning during the supplement’s preparation. All of the papers were written online using Google collaboration tools. Residents with publishing experience mentored more junior residents. Many of the authors reached out to experts in the Hawai‘i medical and public health communities, anchoring their papers in local tradition and experience. They had regular meetings with deadlines and updates throughout the academic year, and gained experience with the peer review process.

The residents also presented a six hour Wilderness Medicine Symposium with Continuing Medical Education credit. They wrote learning objectives using Bloom’s taxonomy, completed nondisclosure forms, and engaged in other activities that are an integral part of the CME accreditation process.

For many, participation in this project represented a sentinel event in their personal professional formation. We hope you sense their enthusiasm, and enjoy their papers.

Disclosures
The Henry M. Jackson Foundation for the Advancement of Military Medicine, Inc. provided financial support for this supplement. Dr. Vincent reported no conflicts of interest.

Disclaimer
The views expressed in this abstract/manuscript are those of the author and do not reflect the official policy or position of the Department of the Army, Department of Defense, or the US Government.
Author’s Affiliation:
Department of Medicine, Tripler Army Medical Center, Honolulu, HI 96859

Correspondence to:
Dale S. Vincent MD; Program Director; Internal Medicine Residency Program
Tripler Army Medical Center, Honolulu, HI 96859;
Ph: (808) 433-6793; Email: dvincenmd@gmail.com

References

Wilderness Medicine in Hawai‘i and the Pacific

Guest Editors

Seth L. Cornell MD
William A. Londeree MD
Gregory M. Sprowl MD
Dale S. Vincent MD

Nu‘uanu from Pali Notches (Photo: William Harner MD)
High Altitude Illnesses in Hawai’i

Terry Shin MD

Abstract
High Altitude Headache (HAH), Acute Mountain Sickness (AMS), and High Altitude Cerebral Edema (HACE) are all high altitude related illnesses in order of severity from the mildly symptomatic to the potentially life-threatening. High altitude illnesses occur when travelers ascend to high altitudes too rapidly, which does not allow enough time for the body to adjust. Slow graded ascent to the desired altitude and termination of ascent if AMS symptoms present are keys to illness prevention. Early recognition and rapid intervention of AMS can halt progression to HACE. Pharmacologic prophylaxis with acetazolamide is a proven method of prevention and treatment of high altitude illness. If prevention fails then treatment modalities include supplemental oxygen, supportive therapy, hyperbaric treatment, and dexamethasone. Given the multitude of visitors to the mountains of Hawai’i, high altitude illness will continue to persist as a prevalent local condition. This paper will emphasize the prevention and early diagnosis of AMS so that the illness does not progress to HACE.

Relevance to Hawai’i and Asia Pacific
High altitude illnesses typically occur with ascent above 2,500 m (8,202 ft) but can occur at altitudes as low as 2000 m (6,562 ft).¹ There are three mountain peaks on the Hawai’i islands that exceed this height threshold. Mauna Kea, the largest mountain on the island of Hawai’i rises to 4205 m (13,796 ft) from sea level. Mauna Loa, also on the island of Hawai’i, peaks at 4,169 m (13,679 ft).² Haleakala, the third highest mountain, is on the island of Maui and rises to 3,055 m (10,023 ft).³ Both tourists and island residents are permitted to travel via a motorized vehicle to the peaks of these mountains. AMS is a common presentation in individuals who reach the summit of these mountains due to rapid changes in altitude. One survey of visitors to Mauna Kea found that 30 percent of tourists and 69 percent of astronomy staff developed symptoms consistent with AMS.⁴

Clinical Description
The most common and initial presentation in high-altitude illness is a headache. This headache is described as high altitude headache (HAH) and will occur in approximately 80% of individuals that ascend to altitude.⁵ HAH can present as a generalized tension or migraine headache. AMS is thought to be the progression of HAH where the headache persists with additional systemic symptoms. At the opposite of the end of the illness spectrum from HAH is the potentially lethal condition known as high altitude cerebral edema (HACE). HACE is considered the end stage of neurologic high altitude sickness. Early recognition of AMS can provide an opportunity for intervention and treatment so that the condition does not progress to HACE.

The exact mechanisms of high altitude illness are still unclear. For the last 20 years, the “tight-fit hypothesis” was the predominant explanation. The tight-fit hypothesis suggests that inadequate cerebral spinal fluid buffer places individuals at risk for increased intracranial pressure from edema formation and increased blood flow.⁶ Recent research, suggests that the tight-fit hypothesis is an oversimplification of complex underlying processes. Current theory describes a complicated networking of hypoxic ventilator response, vessel permeability, multiple chemical mediators, and sodium/potassium ATPase failure.⁷ AMS symptoms can occur as early as an hour after ascent, but typically occur 6 to 10 hours following ascent to higher altitudes.⁸ There are no known diagnostic modalities that reliably confirm the diagnosis of AMS. AMS is a clinical diagnosis based on history and symptoms. At the Hypoxia and Mountain Medicine Symposium in Lake Louise, Canada a consensus was established for the diagnosis. This group defined AMS as an unacclimatized person who recently ascended an altitude above 2500 m who presents with headache, plus the presence of at least one of the following conditions: gastrointestinal symptoms (anorexia, nausea, vomiting), fatigue/weakness, dizziness/lightheadedness, or difficulty with sleep.⁹ A scoring table was created to diagnose and predict the severity of AMS. (Table 1) The Lake Louise Scoring System Table includes a total of 5 questions. Any individual with a self reported score of at least 3 or more is considered to have AMS. A score of 3-5 is considered mild AMS and a score of 6 or more is considered to be moderate to severe.

Table 1. Lake Louise Score for AMS

<table>
<thead>
<tr>
<th>Self Reported Score</th>
<th>Severity</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headache</td>
<td>No headache</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Mild headache</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Moderate headache</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Severe headache, incapacitating</td>
<td>3</td>
</tr>
<tr>
<td>Gastrointestinal Symptoms</td>
<td>None</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Poor appetite or nausea</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Moderate nausea&amp;/or vomiting</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Severe nausea &amp;/or vomiting</td>
<td>3</td>
</tr>
<tr>
<td>Fatigue/Weakness</td>
<td>Not tired or weak</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Mild fatigue/weakness</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Moderate fatigue/weakness</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Severe fatigue/weakness</td>
<td>3</td>
</tr>
<tr>
<td>Dizziness/Lightheadedness</td>
<td>Not dizzy</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Mild dizziness</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Moderate dizziness</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Severe dizziness, incapacitating</td>
<td>3</td>
</tr>
<tr>
<td>Difficulty Sleeping</td>
<td>Slept as well as usual</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Did not sleep as well as usual</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Woke many times, poor sleep</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Could not sleep at all</td>
<td>3</td>
</tr>
<tr>
<td><strong>TOTAL SCORE</strong></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

AMS score >= 3 MILD AMS
AMS score >= 6 MODERATE/SEVERE AMS

HAWAI’I JOURNAL OF MEDICINE & PUBLIC HEALTH, NOVEMBER 2014, VOL 73, NO 11, SUPPLEMENT 2
Other diagnoses should be pursued if symptoms persist greater than three days, there is a lack of response after descent, or there is an absence of a headache. A differential diagnosis for AMS includes dehydration, migraine, hypoglycemia, CNS infection, transient ischemic attack, carbon monoxide exposure and substance abuse.6

High altitude cerebral edema is a rare condition which is considered the end stage of high altitude illnesses. HACE is rare and the incidence is roughly seen in only 1% to 2% of people ascending to heights greater than 4500 m (14,763 ft).9 HACE, however, can occur at elevations as low as 2750 m (9022 ft).6 HACE can be visualized with MRI, which presents as reversible white matter edema within the corpus callosum and splenium.10 The two primary clinical presentations of HACE are altered consciousness and ataxia. The condition can progress rapidly from hallucinations and irrational behavior to lethargy and coma. Death will typically occur as a result of brain herniation. Physical exam findings include truncal ataxia, papilledema, retinal hemorrhage, and other focal neurologic deficits. Due to its lethality, patients presenting with any such symptoms should be treated as if diagnosed with HACE until proven otherwise.

Example Case
A male Kailua resident drove up to the top of the volcano, Mauna Loa. At approximately 11,000 feet, he hiked along a trail up to his destination of 13,200 feet.11 During his ascent, he began to develop headache, nausea, and lightheadedness that eventually progressed to bouts of persistent emesis. Despite oral fluid hydration and attempting to stay warm, his symptoms persisted. He was eventually air lifted from his location and was subsequently transferred to Hilo Medical Center. He was discovered to have lost 10 pounds in a 24-hour period. He was administered intravenous fluids and prescribed analgesic medications for his headache. He reportedly recovered without significant sequelae.

Treatment in Resource Constrained Environments
Prevention is the cornerstone to averting high-altitude illnesses, especially when ascending mountains of high altitude. One of the simplest and safest ways of preventing AMS is by ascending to higher altitudes slowly along graded elevations. Two such methods are staged ascent and graded ascent. In staged ascent, an individual will ascend at an average pace to a moderate altitude and remain there for three or more days to allow acclimatization before further ascension. Graded ascent limits the gain in altitude with partial acclimatization throughout the ascent.13 For prolonged habitation at altitudes above 3000 m, it is recommended that individuals limit sleeping altitude to no more than 300 meters above the previous sleeping altitude.13 A combination of the two methods is considered optimal in preventing high altitude illness. Graded ascent would be the most practical method in high-altitude illness prevention given the ability to rapidly obtain high altitudes in Hawai‘i.

Pharmacologic prophylaxis with diuretics is an option that can be utilized to limit the effects of altitude sickness. Acetazolamide, a carbonic anhydrase inhibitor, is the drug of choice for prevention of AMS. Multiple trials have demonstrated the effectiveness of acetazolamide for AMS prevention and it is currently a Grade 1A recommendation per the Wilderness Medical Society.14 Acetazolamide is indicated for rapid ascent to altitudes greater than 3000 m (9843 feet).15 Prophylaxis with acetazolamide would be most applicable to individuals traveling to Mauna Kea and Mauna Loa who plan prolonged stays at high altitudes. Other indications for prophylaxis include rapid gain in sleeping altitude in a day and a history of AMS. The common dose regimen for acetazolamide ranges from 125 mg to 250 mg twice a day.5

If an individual develops AMS, the treatment should include halting ascension and awaiting acclimatization. A clear indication for immediate descent is a severe neurologic change. The existence of only a few reported incidents in Hawai‘i of serious altitude illness might be explained by the ability to descend rapidly from the mountains such as on Mauna Kea, leading to prompt reversal of any adverse effects.5

Lightweight portable hyperbaric chambers commonly known as Gamow bags can be used when immediate descent is not possible. They consist of lightweight fabrics that serve as a pressure bag with manual air pumps to create a hyperbaric chamber. Prospective studies have demonstrated hyperbaric chambers are effective for immediate relief of AMS and HACE.16 They are usually limited to mountaineering expeditions and are typically not utilized in lower altitudes.

Treatment in Non-Austere Environments
After halting ascent any unnecessary physical exertion should be kept to a minimum. Treatment typically involves supportive care and initiation of low-flow supplemental oxygen to maintain arterial oxygen saturation to greater than 90%. Even though in other geographic locations oxygen tanks are typically carried by climbers on ascents that involve high altitudes, in Hawai‘i, supplemental oxygen will most likely be available only to EMS or in hospital settings. Supplemental oxygen should be administered in the inpatient setting for approximately 1 to 2 days or until AMS symptoms resolve.8 Patients often present dehydrated and should receive oral or intravenous hydration as appropriate. They can also be treated with Acetazolamide, but it should be administered as early as possible to capture any benefit. Symptomatic therapy typically includes analgesics such as NSAIDs or acetaminophen for headaches. Antiemetics should be provided to treat nausea and vomiting. Treatment with dexamethasone can also relieve symptoms but it is usually limited to emergency cases where progression to HACE is probable. The usual dosage is 4 mg every 6 hours until resolution of symptoms.8 A stationary hyperbaric chamber is an additional treatment option but is not required unless there is a concern for refractory HACE.
Conclusion

High Altitude Illness is recognized as a notable concern in high altitude medicine, and public awareness is vital for prevention. Education of healthcare providers to readily recognize and appropriately treat individuals presenting with symptoms is crucial to preventing fatalities. Acute Mountain Sickness does occur on the islands of Hawai‘i, but the progression of AMS to HACE is uncommon given the local topography which allows for quick descent to lower altitudes. Nevertheless, the case presented in this article illustrates the susceptibility of climbers to AMS with the potential progression to HACE in Hawai‘i. The key to preventing high altitude illnesses ultimately relies on an established system to properly educate travelers and residents who visit the mountains of Hawai‘i.

Disclosures

The author reported no conflicts of interest.

Disclaimer

The views expressed in this paper are those of the author and do not reflect the official policy or position of the Department of the Army, Department of Defense or the U.S. Government.

Author’s Affiliation:
Department of Medicine, Tripler Army Medical Center, Honolulu, HI 96859

References

12. Mountain Operations, Field Manual 3-97.6 (90-6), Headquarters, Department of the Army. 28 November 2000.

Mauna Kea Road (Photo: Jacob M. Mathew DO)
Is High Altitude Pulmonary Edema Relevant to Hawai‘i?

Seth Lewis Cornell MD

Abstract
High altitude clinical syndromes have been described in the medical literature but may be under recognized in the state of Hawai‘i. As tourism increases, high altitude injuries may follow given the easy access to high altitude attractions. Visitors and clinicians should be aware of the dangers associated with the rapid ascent to high altitudes in the perceived comfort of a vehicle. This paper will review the basic pathophysiology, prevention, and treatment of the most serious of the high altitude clinical syndromes, high altitude pulmonary edema.

Introduction
The physiologic effects of high altitude are complex and not simply limited to hypoxia. This is reflected by the varying ability of individuals to acclimate to changes in altitude. High altitude clinical syndromes are diverse and can range from life threatening high altitude cerebral and pulmonary edema to less severe acute mountain sickness. The rate of ascent is often directly related to the severity of the clinical syndrome. High altitude pulmonary edema (HAPE) is the most frequent cause of altitude related fatalities. Although the pathophysiology is not completely understood, HAPE is easily treatable; unrecognized, it may be fatal.

Relevance to Hawai‘i and Pacific Region
Mauna Kea is the highest point in the Pacific Basin and if measured from base to summit, the tallest mountain in the world. From its origins deep within the Pacific Ocean, to its high altitude peak, this dormant volcano rises approximately 32,000 feet and stands at 13,796 feet above sea level. This sacred mountain, once regarded as “the abode of gods,” is now visited by several hundred visitors daily, usually by vehicle. Poli‘ahu, the ancient snow goddess of Mauna Kea and rival of Pele, now keeps watch over the world’s largest collection of observatories, which occupy the summit of the hallowed peak. The Hawaiian Islands offer lowlanders the rare opportunity of ascending from near sea level to elevations in excess of two and a half miles. In fact, many of these volcanic peaks are readily accessible by automobile, allowing people of all backgrounds to rapidly visit areas previously limited to the determined mountaineer. Although easy access affords many the opportunity to experience the wonders of the volcanic goddess, Pele, it also exposes many tourists to the adverse effects of high altitude physiology.

Case Report from the Medical Literature
One of the first reported cases of high altitude pulmonary edema appeared in a 1960 article by Dr. Charles S. Houston. Dr. Houston described a healthy 21 year old male who made a cross country ski trip from Aspen, Colorado (elevation 7,890ft) over a 12,000 foot mountain pass. On the third day of the expedition, the patient noted dyspnea, weakness, and cough that subsequently worsened over the fourth day. The patient was transported to the nearest hospital where he was found to have a temperature of 99.6°F, a blood pressure of 120/80, a pulse of 96 and respirations of 30. His physical exam was remarkable for moderate cyanosis, with bilateral coarse pulmonary rales noted on auscultation. There was no cardiomegaly, peripheral edema, or jugular venous distention. The patient had a leukocytosis of 13,000 and a chest radiograph that revealed bilateral pulmonary edema. The patient experienced rapid resolution of his symptoms within 36 hours of admission. On further questioning, the patient described similar symptoms during a previous mountain expedition three years earlier with complete resolution of his dyspnea after descent from altitude.

Discussion
Hypoxia is the primary insult of high altitude exposure. Barometric pressure and the partial pressure of inspired oxygen (PIO2) decreases in a logarithmic manner as altitude is increased. At altitudes below 10,000 feet, the changes in PIO2 and resultant partial pressure of arterial oxygen (PaO2) routinely have little effect on the arterial oxygen saturation (SaO2). However, above 10,000 feet small decreases in PaO2 can have significant effects on SaO2. Fortunately, the human body has innate physiologic mechanisms that allow for altitude acclimatization. The process of acclimatization is complex and its success varies from individual to individual. The rate of altitude gain directly correlates with the severity of high altitude pathology and therefore it is recommended that altitude gains be incremental, allowing time for physiologic adjustment. The recommended rate of ascent and time allowed for acclimatization is dynamic and based on an individual’s overall risk for developing a high altitude illness.

Hyperventilation is an early response to low PaO2 and works to enhance oxygen delivery to tissues. Hyperventilation causes a respiratory alkalosis which triggers renal excretion of bicarbonate as a compensatory mechanism to normalize serum pH. Maximum ventilation with renal adjustment is typically reached within one week at a given altitude. The degree of an individual’s hyperventilatory response may correlate with overall acclimatization success and the prevention of high altitude illness.

Renal secretion of erythropoietin also occurs in response to hypoxia. Increased proliferation of red blood cells begins within four days, however, the complete effects of erythropoiesis may take several weeks. The success of acclimatization does not seem to correlate with the degree of erythropoiesis. Changes in mitochondrial architecture and quantity have also been proposed as another mechanism of altitude acclimatization.
When acclimatization to altitude does not occur, several high-altitude syndromes may happen. The high-altitude syndromes (acute mountain sickness, high altitude cerebral edema, and high altitude pulmonary edema) likely represent a spectrum of pathophysiology. Symptoms often overlap making an accurate clinical distinction difficult.

HAPE is the leading cause of death related to high altitude syndromes. Symptom onset is often encountered within 2-4 days of arrival at high altitude (approximately 10,000 ft), however, incidents have occurred sooner and at lower altitudes. Affected individuals typically complain of malaise, cough, dyspnea, and fatigue. Physical examination may reveal a cyanotic appearance with vital signs demonstrating tachypnea and tachycardia, along with a possible fever. Predicting which individuals will be most susceptible to HAPE is difficult, though previous episodes seem to be the strongest predictor of future susceptibility. Certain underlying medical conditions such as lung disease, heart disease, diabetes, and pregnancy, may also put individuals at increased risk of developing HAPE.14-17

The pathophysiology of HAPE is not completely understood, although increased pulmonary arterial pressure (PAP) seems to be uniformly present. Pulmonary vasoconstriction and resultant increased PAP is a known response to hypoxia. However, increased PAP alone cannot explain HAPE, as hypoxia and PAP are common findings among asymptomatic mountaineers at altitude.18 Increased capillary permeability, perhaps secondary to increased capillary stress from PAP, has been proposed as a possible mechanism in HAPE.19

**Treatment in Resource-Constrained Environment**

Simple awareness and education of HAPE often allows for appropriate preventive techniques to be used. All individuals should be encouraged to slow their rate of ascent once above 8,500 ft. For prolonged expeditions, most experts recommend no greater than an increase of 1,500 ft per day for sleeping elevations, with no elevation gain every third or fourth day. For individuals with a prior history of HAPE, pharmacologic prophylaxis should be considered. Numerous pharmacologic agents have been used to prevent HAPE (nifedipine, salmeterol, tadalafil, dexamethasone, acetazolamide), however, current guidelines only recommend the use of nifedipine.20 Most experts recommend 30 mg of sustained release nifedipine every 12 hours which should be started on the day prior to ascent and continued until descent is initiated.

Prompt recognition of HAPE is essential, as most cases improve dramatically with descent from altitude. Emergent cases require rapid descent, whereas less severe symptoms may improve with a moderate decrease of 1,500ft to 3,000ft. Since patients frequently suffer from hypoxemia during HAPE, exertion during the descent should be minimized as much as possible and supplemental oxygen administered. Euthermia should also be maintained to lessen any increases in PAP, which may worsen the pulmonary edema. If attempts at re-ascent are made, additional time should be allowed for proper acclimatization.

**Treatment in Non-Austere Environments**

As most cases of HAPE resolve with descent from altitude, additional treatment is not typically required, however, severe cases of HAPE may require hospitalization with more intensive medical care. In addition to descending from altitude, supplemental oxygen is a mainstay of therapy. Hypoxia frequently resolves with low flow oxygen, though more severe cases may require high flow or even continuous positive airway pressure (CPAP). Portable hyperbaric chambers have also been used for HAPE treatment. Endotracheal intubation with mechanical ventilation is infrequently required but may be necessary.

There are several proposed medications often used in the adjunctive treatment of HAPE. Nifedipine, a dihydropyridine calcium channel blocker, is the most well studied of the treatment modalities and works through vasodilation which acts to lower PAP. Likewise, phosphodiesterase inhibitors such as sildenafil may blunt the hypoxic pulmonary vasoconstriction response, thus improving PAP. Other previously used adjuncts include diuretics, corticosteroids, carbonic anhydrase inhibitors, and inhaled beta-agonists, though current guidelines do not support their routine use in the treatment of HAPE.20

**Conclusion**

High altitude pulmonary edema is an easily treatable, though potentially fatal, syndrome of the acute mountain illnesses. With education and implementation of proper preventive techniques, such as a judicious rate of ascent above 10,000ft and nifedipine when indicated, HAPE can often be avoided. Several of the volcanic peaks found among the Hawaiian Islands rise well above 10,000ft and many are directly accessible by vehicle and overly rapid ascent. Proper education and recognition of HAPE is paramount in decreasing its incidence and reducing its morbidity.

**Disclosures**

The author reported no conflicts of interest.

**Disclaimer**

The views expressed in this manuscript are those of the author and do not reflect the official policy or position of the Department of the Army, Department of Defense, or the US Government.

**Author’s Affiliation:**
Department of Medicine, Tripler Army Medical Center, Honolulu, HI 96859

**Correspondence to:**
Seth Cornell MD; 1 Jarrett White Rd, Honolulu, HI 96859;
Email: seth.l.cornell.mil@mail.mil
References
Prevention and Treatment of Injuries from Cave Exploration in Hawai‘i

Abstract
Cave exploration is a popular activity in the United States that can be challenging, thrilling, and dangerous. In addition to common risks associated with caves on the mainland, caves in Hawai‘i may be filled with tidal water, or contain large pools of water that are accessible only through underwater entrances. This paper will discuss common injuries in caves on the mainland United States, as well as cave related injuries in Hawai‘i as reported to the National Speleological Society from 1984-2013.

Introduction
Cave exploration is a popular activity among well-trained cavers as well as untrained members of the general population. Over two million people visit thousands of caves in the United States each year.1-4 Caves are often cold, dark, and contain confusing tunnels with tight and twisting spaces. An unfamiliar environment has inherent dangers, and injuries can be serious, especially in the inexperienced cave explorer.

Relevance to Hawai‘i
The Hawaiian islands are home to numerous caves, many formed by lava tubes during the islands’ development. Hawai‘i attracts millions of tourists annually and this brings increased risks and dangers to visitors who explore them and are unfamiliar with their challenges. From 1980 through 2008, 877 nationwide incident reports were sent to the National Speleological Society (NSS) Accident Reports, involving 1356 individual cavers, and eighty one (6%) had fatal outcomes. From 1984 through 2013, a total of 33 incidents involving 35 people were reported from Hawai‘i, and 18 (51.4%) died as a result of injuries sustained during cave exploration. Cave rescue and medical management are clearly pertinent topics for Hawai‘i in view of these serious injuries and outcomes.

Illustrative Case
Caves often present rough terrain and tight passageways, and may allow for passage of only one individual at a time. This may prolong the time to reach an injured patient, and to manage an extraction. Primary interventions such as securing an airway and monitoring for breathing and circulation are more difficult. For example, if a patient vomits during transport, airway and monitoring for breathing and circulation are more challenging. One case report describes an incident that took place in Yorkshire in which an individual became trapped and pinned by a dislodged boulder while crawling through a cave.3 The patient, with two friends, had entered the cave at 1000 and after 2 hours was approximately one-half mile from the cave entrance. It took his friends one hour and forty five minutes to reach the surface and call for help. The rescue team, including a physician, arrived on site at 1630 and reached and released the patient at 1720. It then took 10.5 hours to get the patient to the surface using a drag stretcher, requiring periodic off loading from the stretcher to maneuver crawl spaces and obstacles. He reached the surface at 0550 the next morning and was admitted to the hospital at 0815. This case highlights the prolonged time that may be required to extract an injured person from a cave.

Treatment in Austere Environment
Treatment can be broadly classified as self-aid, rescuer treatment, and definitive hospital care. Training and preparedness are both extremely important preventative measures which significantly decrease the risk and severity of injury.2 The most common reported incidents in Hawai‘i are falls (41.7%), drowning (30.6%), and being lost or stranded (8.3%) (Table 1). A national survey suggested that experienced cavers are much less likely to sustain serious injuries and are more likely to take preventative measures, such as appropriate clothing to prevent hypothermia and also wearing of helmets.2 Self-aid is the initial treatment strategy immediately following an injury. Since most caving injuries occur to inexperienced cavers, resources (ie, water, batteries, heat source, tourniquet, compression bandages) are likely to be limited. Falls, the most common type of reported injury in Hawai‘i, can result in multiple injuries, usually involving sprains or fractures but also head trauma. Injured cavers need to be resourceful during this initial phase of treatment. One Hawai‘i incident reported to the NSS described a photographer who fell and fractured his leg while at the entrance of a cave. He used clothing to brace his leg with the camera tripod, enabling him to walk a sufficient distance to seek help. The majority of cases that result in successful rescue are typically non-life threatening injuries.

Stabilization of a sprain or fracture with a simple improvised brace or splint is often sufficient until further medical care is available. If immediate, life-threatening concerns (bleeding or hemorrhage) are present, compression should be applied continuously during the rescue. Use of improvised tourniquets is controversial but may provide life-saving control of hemorrhage at the expense of a limb in prolonged extractions.5 A list of useful items in a rescue kit is shown in Table 3.

Although the Hawaiian climate is tropical, hypothermia is a real threat in a cave, which may be significantly cooler and wetter than the surface. Injured cavers should attempt to remain dry and rescuers should remove any wet clothing, wrap the patient in waterproof garments and warm with blankets or body contact whenever possible during prolonged extractions.5 The injured caver can also be given warm fluids by mouth, as injuries allow, helping to increase the core body temperature.
Table 1. Mechanism of Incident (Data from NCRC Incident Reports)

<table>
<thead>
<tr>
<th>Incident</th>
<th>Number (% of total # incidents)</th>
<th>Fatality (%)</th>
<th>Injury and aid (%)</th>
<th>Injury without aid (%)</th>
<th>Aid only, no injury (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Caver Fall</td>
<td>15 (41.7)</td>
<td>3 (21.4)</td>
<td>8 (50)</td>
<td>3 (21.4)</td>
<td>1 (7.1)</td>
</tr>
<tr>
<td>Drowning</td>
<td>11 (30.6)</td>
<td>11 (100)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Lost or Stranded</td>
<td>3 (8.3)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>3 (100)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Near Drowning</td>
<td>2 (5.6)</td>
<td>0 (0)</td>
<td>2 (100)</td>
<td>0 (0)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Fell and Drown</td>
<td>1 (2.8)</td>
<td>1 (100)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Laceration</td>
<td>1 (2.8)</td>
<td>0 (0)</td>
<td>1 (100)</td>
<td>0 (0)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Cardiac Arrest</td>
<td>1 (2.8)</td>
<td>1 (100)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Unknown</td>
<td>1 (2.8)</td>
<td>1 (100)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Found Murdered Victim</td>
<td>1 (2.8)</td>
<td>1 (100)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
</tr>
<tr>
<td></td>
<td>36</td>
<td>18</td>
<td>10</td>
<td>6</td>
<td>1</td>
</tr>
</tbody>
</table>

Table 2. Months of Reported Incidents (Data from NCRC Incident Reports)

<table>
<thead>
<tr>
<th>Month of Year</th>
<th>Number (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>January</td>
<td>3</td>
</tr>
<tr>
<td>February</td>
<td>6</td>
</tr>
<tr>
<td>March</td>
<td>2</td>
</tr>
<tr>
<td>April</td>
<td>2</td>
</tr>
<tr>
<td>May</td>
<td>2</td>
</tr>
<tr>
<td>June</td>
<td>1</td>
</tr>
<tr>
<td>July</td>
<td>5</td>
</tr>
<tr>
<td>August</td>
<td>1</td>
</tr>
<tr>
<td>September</td>
<td>3</td>
</tr>
<tr>
<td>October</td>
<td>1</td>
</tr>
<tr>
<td>November</td>
<td>5</td>
</tr>
<tr>
<td>December</td>
<td>2</td>
</tr>
<tr>
<td>Winter</td>
<td>1</td>
</tr>
<tr>
<td>Spring</td>
<td>1</td>
</tr>
<tr>
<td>Unknown</td>
<td>1</td>
</tr>
</tbody>
</table>

Table 3. Suggested items of a Cave Medic Kit

<table>
<thead>
<tr>
<th>Contents of a Basic Cave Medic Kit</th>
<th>Additions for Personnel with Higher Level of Training/Licensing</th>
</tr>
</thead>
<tbody>
<tr>
<td>● Scissors</td>
<td>● Narcotic/pain medications</td>
</tr>
<tr>
<td>● Adjustable Cervical Collar</td>
<td>● Intramuscular antibiotics</td>
</tr>
<tr>
<td>● Duct Tape</td>
<td>● Injectable 1% lidocaine syringes and needles for regional nerve blocks</td>
</tr>
<tr>
<td>● Gauze bandages</td>
<td>● Povidone-iodine prep pads</td>
</tr>
<tr>
<td>● Flexible splints</td>
<td>● Scalpel blades</td>
</tr>
<tr>
<td>● Elastic wrap</td>
<td></td>
</tr>
<tr>
<td>● Personal Protective Equipment</td>
<td></td>
</tr>
<tr>
<td>● Stethoscope</td>
<td></td>
</tr>
<tr>
<td>● Blood pressure cuff</td>
<td></td>
</tr>
<tr>
<td>● Pencil with waterproof paper</td>
<td></td>
</tr>
<tr>
<td>● Digital thermometer</td>
<td></td>
</tr>
<tr>
<td>● Safety pins</td>
<td></td>
</tr>
<tr>
<td>● Nasal and oral airways</td>
<td></td>
</tr>
</tbody>
</table>

Conclusion
The type of injuries sustained in caves can vary greatly and, in many cases, inexperienced cavers are underprepared for injuries and rescuers have limited medical training. Bleeding should be controlled, fractures and sprains stabilized, and the injured patient kept warm, dry, and hydrated. Cervical spine stabilization should be considered in any patient that has sustained a fall. Most people with life-threatening caving injuries do not survive due to prolonged time to definitive care. Therefore, life-threatening injuries such as heavy bleeding, hypothermia, crush injuries, and respiratory distress must be recognized and treated to avoid progression to fatal outcomes. Many patients will be cold, scared and experience panic attacks. Rescuers should be prepared to reassure these individuals as much as possible.

Disclosures
The authors reported no conflicts of interest.

Disclaimer
The views expressed in this manuscript are those of the authors and do not reflect the official policy or position of the Department of the Army, Department of Defense, or the US Government.

Acknowledgements
The National Speleological Society (NSS) for allowing access to their incident reports and to John Halleck for his personal experience, authorship and guidance on this project.

Authors' Affiliation:
Department of Medicine, Tripler Army Medical Center, Honolulu, HI 96859

Correspondence to:
David W. Cowart MD; 1 Jarrett White Rd, Honolulu, HI 96859; Email: david.w.cowart.mil@mail.mil
References

Additional Public Resources
The Risks of Scuba Diving: A Focus on Decompression Illness

Jennifer Hall DO

Abstract
Decompression Illness includes both Decompression Sickness (DCS) and Pulmonary Overinflation Syndrome (POIS), subsets of diving-related injury related to scuba diving. DCS is a condition in which gas bubbles that form while diving do not have adequate time to be resorbed or “off-gassed,” resulting in entrapment in specific regions of the body. POIS is due to an overly rapid ascent to the surface resulting in the rupture of alveoli and subsequent extravasation of air bubbles into tissue planes or even the cerebral circulation. Divers must always be cognizant of dive time and depth, and be trained in the management of decompression. A slow and controlled ascent, plus proper control of buoyancy can reduce the dangerous consequences of pulmonary barotrauma. The incidence of adverse effects can be diminished with safe practices, allowing for the full enjoyment of this adventurous aquatic sport.

Introduction
Scuba diving is a sport with exhilaration, beauty, and fascination; however, the risks involved are often not advertised. Two specific conditions can turn a fantastic dive into trouble, with occasional fatal outcomes: Decompression Sickness and Pulmonary Overinflation Syndrome. After spending six years as a US Navy Diving Medical Officer, I will share some case reports and discuss the physiology, clinical presentation, and treatments for these diving-related injuries.

Decompression Illness includes both Decompression Sickness (DCS) and Pulmonary Overinflation Syndrome (POIS), subsets of diving-related injury related to scuba diving. Divers should understand their limitations and how to prevent adverse outcomes. DCS is a condition in which gas bubbles that form while diving do not have adequate time to be resorbed or “off-gassed.” This results in bubble entrapment in specific regions of the body, most commonly in joints such as the shoulder. If adequate decompression time is omitted, the trapped bubbles may lead to DCS.1 POIS is due to an overly rapid ascent to the surface resulting in the rupture of alveoli and subsequent extravasation of air bubbles into tissue planes.2 On rare occasions, the bubbles may traverse the cerebral circulation, causing a potentially fatal condition.3 Divers must always be cognizant of dive time and depth, and be trained in the management of decompression. A slow and controlled ascent, plus proper control of buoyancy can reduce the dangerous consequences of pulmonary barotrauma. Overall, the incidence of adverse effects can be diminished with safe practices, allowing for the full enjoyment of this adventurous aquatic sport.

Relevance to Hawai‘i and Asia Pacific
The Hawaiian Islands and islands throughout Asia Pacific are popular scuba diving venues with an abundance of marine life and warm, comfortable weather conditions year-round. The incidence of diving-related DCS ranges from 1 to 35 events per 10,000 dives, depending on region of the world and specific class of diving (ie, recreational, commercial, military, or scientific).3,4 Assuming an average of fifteen diving-related injuries per 10,000 dives, and approximately 200 daily dives across the Hawaiian Islands, we would predict one DCS or POIS event every week.3,4

Example Cases
Case 1
A 28-year-old US Navy SEAL performed a working dive using scuba to secure an apparatus on the bottom to buoy lines at the water surface. This entailed multiple descents and ascents. The seas had been choppy with swells of two to four feet and currents at roughly one to two knots, thus creating a challenging environment. The diver descended to 60 feet and ascended to the surface, intermittently holding his breath while bobbing up and down on the surface. After obtaining the next piece of work equipment from the zodiac boat, he made another descent to 60 ft, followed by ascent to the surface. This time, he noted blurry vision in his right eye, thinking he had merely gotten seawater under his mask. The diver proceeded to the bottom again to finish his job. Upon ascent, he experienced mild paresthesias to the right side of his face. The diver was brought into the zodiac boat and described his symptoms to the diving supervisor. His symptoms quickly progressed to involve numbness and tingling in his left upper extremity, at which time the duty diving bends team (a specialized and trained group of divers and medical personnel who respond to diving casualties) was activated. The diver was promptly transported to the support team and diving chamber. When the diver arrived approximately five minutes later, the entire right side of his body was numb. He also had difficulty ambulating. The diver was urgently brought to the hyperbaric chamber where he immediately descended to 60 feet of seawater (fsw) and was placed on 100% oxygen. Within minutes, the diver had full resolution of his symptoms. A full treatment per the US Navy standard diving guidelines was completed for the diagnosis of arterial gas embolism (AGE).

This scenario describes pulmonary barotrauma resulting in arterial gas embolism. The diver’s rapid ascent to the surface after repeated dives placed the diver at increased risk for POIS. His symptoms were of quick onset and rapid progression, ultimately requiring emergent treatment with hyperbaric oxygen therapy (HBO). Fortunately, he was in close proximity to a diving chamber and had expeditious treatment.

Following HBO treatment, the resolution occurred quickly after he reached 60 fsw, which supports the diagnosis of arterial gas embolism. In AGE, air bubbles enter into the cerebral circulation and become lodged in the arterial vasculature, resulting in symptoms mimicking a stroke. HBO therapy essentially...
follows Boyle’s Law, wherein the increase in atmospheric pressure causes the volume of the bubble to decrease, allowing for circulation to be restored.

**Case 2**
A 35-year-old man was in the process of becoming certified as an advanced scuba diver with a dive company from O‘ahu. He was to complete multiple dives, including wreck and night dives. The first day, he completed a dive for thirty-five minutes to 65 feet, followed by another dive that night for thirty minutes to 45 feet. The diver felt well and the following morning joined his dive crew for his third dive of the weekend. He made a dive to sixty feet for 45 minutes. The fourth dive, two hours later, had been planned to a maximum depth of fifty feet. The diver was feeling very tired with pain in his right shoulder. Despite his symptoms, he continued with his final dive. Surprising to him, when he reached depth, his shoulder pain resolved. However, when he reached the surface after this final dive, his shoulder pain recurred. This diver did not state any complaints after his dive course and went home. His wife noted that her husband was acting more fatigued than usual and perhaps had some element of confusion and memory loss. Furthermore, he began to complain of worsening shoulder pain. The following day, the diver presented for medical advice, examination and possible treatment. His symptoms raised the suspicion for DCS, even though his dive computer did not show any “omitted decompressions” during his dives. Omitted decompression occurs when a diver fails to make a needed decompression stop. Dive computers are programmed to indicate when decompression stops are required and the duration of these stops. Since DCS was suspected, he was treated in the hyperbaric chamber, and his symptoms resolved after two days of HBO therapy.

This diver likely suffered from decompression sickness, which may be seen in divers who adhere to their dive time and depth and do not omit decompression. Interestingly, this diver had an echocardiogram with agitated saline which revealed a Patent Foramen Ovale (PFO). It is unclear whether the PFO contributed to his DSC.

**Discussion**
Diving compressed gases (ie, scuba diving) can lead to two very serious medical conditions: Decompression Sickness (DCS), otherwise known as “the Bends,” and Pulmonary Over-Inflation Syndrome (POIS). DCS most often is not lethal, but is associated with morbidity, whereas POIS can result in a spectrum of disorders from minor complaints to potentially lethal sequelae unless emergent treatment with hyperbaric oxygen therapy is performed.

There are four distinct disorders as a part of POIS: Arterial gas embolism (AGE), pneumothorax, mediastinal emphysema and subcutaneous emphysema. They all result from overdistention and rupture of the lungs by expanding gases during ascent. The expanding gases cannot escape, leading to one of the myriad of symptoms of POIS. Boyle’s law states that if the temperature of a fixed mass of gas is kept constant, the relationship between the volume and pressure will vary in such a way that the product of the pressure and volume will remain constant. In other words, on ascent, when the atmospheric pressure is decreasing, the volume of gas bubbles will increase ($P_1V_1 = P_2V_2$). This explains why pulmonary barotrauma occurs. Inexperienced divers who panic may ascend rapidly and develop POIS.

Air embolism is the most serious manifestation of POIS, resulting from gas passing from the ruptured lung into the pulmonary veins and then into systemic circulation. Here, it can cause vascular damage or obstruction, hypoxia, infarction, and activation of an inflammatory cascade. Initial presentation includes loss of consciousness or symptoms similar to acute stroke. The symptoms upon ascent are often immediate, but may present up to ten minutes after surfacing. Serious effects may result from blockage of cerebral or coronary vessels by bubbles merely 25 to 50 microns in diameter, or by interrupting flow. Treatment must be immediate, consisting of urgent recompression in a hyperbaric chamber.

The three types of pulmonary barotrauma are typically less serious, but may warrant treatment. The mechanism is similar to the pathophysiology of pulmonary barotrauma. After alveolar rupture, gas escapes into the interstitial pulmonary tissues. This gas may track along the loose tissue planes surrounding the airways and blood vessels, into the hilar regions, and then into the mediastinum, neck and the pleural space. This results in mediastinal emphysema, subcutaneous emphysema or pneumothorax. Symptoms include voice changes, feeling of fullness in the throat, dyspnea, dysphagia, retrosternal discomfort, chest pain, or in extreme cases, syncope and collapse. As in the case of emphysema, crepitus is often palpated on the surface of the skin. Usually these syndromes are self-limited and treated with observation, allowing time for resorption of the gas bubbles. Mild symptoms may be treated with 100% oxygen administered by face mask for approximately 4-6 hours. In severe, albeit rare cases, a shallow decompression treatment may be started, which is usually adequate for resolution. Pneumothorax may require chest tube placement, depending on the size and symptoms present.

Decompression sickness is much more common. Present-day diving often follows dive tables, initially adopted by the US Navy. Modern technology allows for most divers to use dive computers which can track their dive and decompression time accurately, without having to calculate diving time based on depth and the dive tables. Despite advances with dive computers, divers still develop DCS, either from human error or from disobeying their decompression guidelines. Even if a diver is adherent to appropriate dive time and depth, the dive tables are not infallible and DCS may still occur.

During DCS, there is liberation of gas bubbles from solution into the tissue or blood. Symptoms often resolve, but, these bubbles may lead to death or permanent neurological impairment. Bubbles may cause direct mechanical effects such as tissue distortion or disruption, or ischemia by blocking blood vessels or increasing tissue pressure sufficient to impair perfusion. They are also known to precipitate an inflammatory cascade.
via neutrophilic activation as a consequence of endothelial or cellular damage.\textsuperscript{1,2} Basically, when diving, the deeper the depth, the greater the partial pressures of gases, which leads to an increase in bubble formation/extraction into the tissues. The longer one remains at that depth, the more bubbles will “on-gas.” The ability of these bubbles to become resorbed into solution, or “off-gas” will depend on how many gas bubbles have accumulated, which depends on time and depth. Thus, one is required to undergo decompression stops in order to allow time for this “off-gassing” to occur. If decompression is omitted, the result may be decompression sickness.

Furthermore, the severity of DCS will also be determined by the amount of omitted decompression. There are many theories as to how DCS develops, but one unifying mechanism has not been established. It is a multifactorial and multiorgan process. There are established risk factors that may predispose a diver to developing decompression sickness. Lack of physical fitness, increased age, obesity, dehydration, physical injury, alcohol use during diving, repetitive dives, and traveling to altitude to dive are some of the risk factors that may lead to higher incidences of DCS.\textsuperscript{1} The best outcomes after diving are in physically fit individuals who are well-hydrated and who follow their dive tables or dive computers without pushing the limits.

Clinical manifestations are classified into two categories: Type I “pain only,” and Type II “serious.” Type I is more common, often with limb or joint pain, and usually affecting the shoulder.\textsuperscript{1,2} Classically, this pain is unaffected by range of motion testing, which differentiates it from musculoskeletal trauma or injury. However, one must be cautious because old injuries are predisposed to developing DCS, acting as a nidus for bubble formation and trapping.\textsuperscript{1} In Type II, symptoms are more serious and usually occur with a significant amount of omitted decompression or extreme deep and long dives. Cerebral, cerebellar, spinal, and inner ear DCS comprise the Type II sub-categories, with emergent decompression being necessary to avoid death or long-term neurological defects.\textsuperscript{1}

Whether one is treating decompression sickness or pulmonary overinflation, the mainstay of treatment is to recompress the affected diver in a hyperbaric chamber with 100% oxygen at high partial pressures. This immediately reduces bubble size, and produces an increased diffusion gradient of inert gas (usually nitrogen) out of the bubbles.\textsuperscript{2} This leads to relief of ischemia and hypoxia, and restores normal tissue function. As long as the chambers are operated by well-trained staff and measures are taken to mitigate any adverse effects from decompression therapy, HBO treatment has the potential to reduce mortality and morbidity in most cases of DCS.

**Treatment in Resource Constrained Environments**

In environments which are remote or where resources are limited, divers who succumb to decompression sickness may undergo an increased risk to any of the divers or crew; (3) the diver with DCS is stable, without any altered state and has ability to use his or her extremities without additional risk; and (4) enough air and tanks are available for in-water decompression. One may try to use 100% oxygen underwater, if available, but there MUST be another diver closely monitoring for oxygen toxicity, and the diver should not be at a depth greater than 30 feet of salt water (O2 toxicity risk is greatly increased when submerged to depths greater than this). Another treatment for austere environments includes 100% oxygen on the surface, which may be beneficial as a bridge to more definitive therapy. A final option is air evacuation to the closest treatment facility.

Whenever diving operations are underway there always needs to be a plan for potential emergency situations, and the boat operator needs to have radio and phone numbers available.

**Treatment in Non-austere Environments**

In diving areas where resources are available, treatment for DCS predominantly entails HBO therapy. However, if symptoms are mild, observation only is required. The stricken diver should still have expert consultation either by a diving medical specialist or by the nearest emergency room.

If a diver should surface with symptoms suggestive of severe barotrauma, severe DCS, or arterial gas embolism, all efforts should be undertaken to transport the patient emergently to the nearest hyperbaric chamber.

**Conclusion**

Scuba diving is an extremely popular sport, especially in the Hawai`i. Many experienced divers as well as newly trained divers flock to the region to experience the marine environment. A moderate climate makes the sport popular throughout the year. With the incidence of decompression illness approximating one DCS case per 1000 dives, we would expect at least one event every week. These diving related disorders need to be recognized, and supervisors and the divers should be cognizant of the risks and dangers that can accompany any dive. Divers need to be well-trained, know their limits, follow their planned dive, and always have an emergency plan.

**Disclosures**

The author reported no conflicts of interest.

**Disclaimer**

The views expressed in this manuscript are those of the author and do not reflect the official policy or position of the Department of the Army, Department of Defense, or the US Government.

Author’s Affiliation:
Department of Medicine, Tripler Army Medical Center, Honolulu, HI 96859

Correspondence to:
Jennifer Hall DO; Tripler Army Medical Center, Honolulu, HI 96859; Email: jhalldo@yahoo.com
References

Alternobaric Vertigo (Photo: Russell Gilbert MD)
Hazards of Hawaiʻi Volcanoes National Park

Gregory M. Sprowl MD

Introduction

For the wilderness enthusiast, Hawaiʻi is renowned as a safe tropical destination free from snakes, diseases of the developing world, and dangerous animals. One of the most popular destinations is Hawaiʻi Volcanoes National Park. With over a million visitors per year, the park is one of the top fifteen visited national parks in the United States. It possesses the only active volcanoes, with unique hazards such as the terrain of established lava flows, unstable lava deltas, lava, and volcanic gases. A visitor can sustain trauma, altitude related illness, burns, hyper and hypothermia, and exacerbation of existing lung conditions.

Relevance to Hawaiʻi and Asia-Pacific

Advancing lava has destroyed multiple communities on the island of Hawaiʻi (Big Island) since Kilauea’s latest eruption began in 1983. This has substantially altered the terrain, destroying infrastructure, and making it difficult to traverse by vehicle or foot. Vog, a mixture of sulfur dioxide (SO2) and other gases emitted from a volcano reacting with oxygen in sunlight, exacerbates respiratory and ocular conditions in susceptible individuals. Concentrated levels of SO2 above World Health Organization (WHO) recommended exposure levels exist frequently on Hawaiʻi island communities southwest of Kilauea, downwind of the usual northeasterly trade winds.

Clinical Relevance

Kilauea, the more active of the two active shield volcanoes on Hawaiʻi Island, has historically produced an average of over 100 tons per day of sulfur dioxide, the most abundant gaseous emission. In some of the more active years since 1983, rates have exceeded 1,000 tons per day. In addition to sulfur dioxide, considerable quantities of hydrogen sulfide, carbon dioxide, carbon monoxide, hydrochloric acid, and particulate matter are produced. These gases can produce acute effects on visitors with chronic respiratory and cardiac disease.

Traversing the terrain of the lava fields causes hundreds of mechanical injuries each year, including lacerations, abrasions, fractures, sprains, and strains. Additionally, dozens of thermal burns have been recorded over the last twenty years, including several fatalities.

Sulfur dioxide is a colorless gas; however it is frequently mixed with other gases and particulate matter. It has a strong odor which most people can detect at 0.3 to 1 ppm, and is irritating to the respiratory tract. OSHA limits on sulfur dioxide exposure are an eight hour total weighted average of 2 ppm and a short term (15 minutes) exposure limit of 5 ppm. Asthmatics can experience increased airway resistance with sulfur dioxide concentrations of less than 0.1 ppm when exercising. Healthy adults experience increased airway resistance at 5 ppm, sneezing and coughing at 10 ppm, and bronchospasm at 20 ppm. Respiratory protection is required for exposures at or above 20 ppm. Sulfur dioxide exposure in the National Park varies widely depending on location and wind direction. With normal northeasterly trade winds the most highly trafficked areas such as the Visitor’s Center and Jagger Museum are likely to experience less than one ppm to zero sulfur dioxide exposure. Close to vent sites and several kilometers downwind, visitors can experience levels over 1 ppm and even over 100 ppm in very close proximity. Measurements up to 75 ppm have been recorded at lava coastal entry sites, locations which are particularly sought by visitors.

Exposure to sulfur dioxide in large quantities has been documented to produce reactive airways dysfunction syndrome (RADS). RADS falls within the same spectrum of disease as irritant induced asthma, and can present as acute asthma in individuals with no prior history after just a single high dose exposure. RADS can lead to long-term asthma-like symptoms that persist for years. It is not well characterized histologically, and is not known to be immune mediated. In several studies of RADS, a majority of patients experienced symptoms following methacholine challenge, and spirometry continued to be abnormal over five years later. While most studies show a mild inflammatory response with sparse lymphocytes and granulocytes, there is an absence of eosinophilia, smooth muscle hyperplasia or mucus gland hypertrophy that are characteristic of asthma.

Hydrogen sulfide is also a colorless gas with a distinctive rotten egg odor that is emitted in sizeable quantities from Kilauea. From May to September 1993, the maximum recorded measurement of H2S was 4.23 ppm at Sulphur Banks, but from 1987-1990 measurements only rarely exceeded the Hawai‘i State Department of Health air quality limit of 0.025 ppm at the park monitoring site. OSHA total weighted average exposure limit for H2S is 10 ppm, with a short term exposure limit 15 ppm. At concentrations above 20 ppm, exposure can elicit headaches, dizziness, nausea and eye irritation. Respiratory effects include bronchitis, rhinitis, and pulmonary edema. Studies indicate that hydrogen sulfide inhibits cytochrome oxidase (aa3) resulting in disruption of the electron transport chain and inhibition of oxidative metabolism in the mitochondria. More recent experiments have determined that greater toxicity may result from the production of reactive oxygen species and depletion of glutathione. No recorded deaths from H2S exposure have occurred in Hawai‘i, although numerous deaths have occurred in areas of volcanic activity in Japan and New Zealand. Hydrogen sulfide toxicity is primarily a concern of industrial exposure.
This is a very small primary volcanic HCl gas emission. It is produced in greater quantities when lava exceeding 1,100 degrees C interacts with chlorides in seawater, and is a component of the superheated steam plumes. While 1994 USGS ambient air measurements recorded only one ppm of HCl at a coastal entry point for lava within the park, this was skewed by the fact that the measuring device only recorded gas, not aerosol vapor. The pH of rain water sampled at the same site supports the theory that the HCl measured levels are not an accurate indication of total HCl concentration. OSHA has a permissible exposure limit of 5 ppm established. Exposures to 35 ppm causes throat irritation and, with higher concentrations over time, can result in swelling and spasm of the throat and bronchospasm. The resulting endothelial damage at high exposure levels may lead to pulmonary edema, progression to acute respiratory distress syndrome (ARDS), and may induce RADS.

While exposure to gases poses a risk and has produced injuries and fatalities, most injuries and fatalities are caused by motor vehicles on the roadways or air travel accidents. These caused 178 of 262 park fatalities during 1993 to 2002. Other causes of death from this period during a study of park hazards indicated that thermal burns, heat stroke, cardiac arrest, respiratory distress, and mechanical injury rounded out the remainder.

**Example Cases**

From 1983 to 2003, five deaths of tourists were directly due to SO2 exposure, with few details available. One person had a history of asthma. The other four deaths occurred when these individuals ignored warning signs for off-limits areas marked during periods of elevated volcanic activity. A closer study from 1993 to 2002 indicated that there were 28 asthma and respiratory distress related deaths, but these were not attributed to a single gas at the eruption site of Kilauea. On November 5, 2000, a woman, age 41, of Volcano, Hawai‘i, and a man, 42, of Washington, DC, were found dead near the lava flow front entry into the ocean. They were found with severe burns, cuts and abrasion to their heads, hands, and knees. According to the medical examiner, “the two died as a result of pulmonary edema caused by inhalation of steam sustained when exposed to a steam plume.” While the investigation did not elaborate further regarding the specific etiology of the pulmonary edema, hydrochloric acid and superheated water steam itself are possible causes of such injuries. Of note, the autopsy summary reported advanced decomposition of the exposed skin due to acid rain.

While relatively uncommon, thermal burns have caused high profile injuries and fatalities in the park. In October 2002, a cruise ship passenger, age 45, was found dead just north of the ranger station at the end of Chain of Craters Road. There were no obvious injuries, foul play was ruled out, and she was not found in immediate proximity to active lava flows. However, it was later reported that she was found twenty feet into a cooling lava flow. The official autopsy report declared that she died of natural causes due to exposure. A lawsuit brought against a tour company that transported her to the park mentioned burns to her feet and other skin surfaces. A visitor from Japan died in the late 1980s when he fell on uneven lava while hiking at night from the flow front to his car parked near Kalapana. In March 2003, a man suffered a mix of first, second, and third degree burns over eight percent of his body when he fell into an active flow. In April 1993 a local native of the island of Hawai‘i, a Kona photographer, died at Kilauea’s Eruption Site when a lava bench which appeared to be solid collapsed. He was attempting to photograph the entry site of lava into the ocean. He and several other onlookers had crossed a rope barrier set up by park rangers. When the bench collapsed, the others were able to scramble to safety, but Nagar was swept into the sea. Five years later, in April 1998, a hiker was declared lost at sea after an extensive search when he slid off a coastal cinder cone at the ocean edge. This occurred at approximately 1 a.m. when the group with whom he was hiking crossed a warning barrier.

**Treatment in Resource-Constrained Environments**

Persons entering the backcountry remote areas should ensure avoidance of areas with significant concentrations of toxic gases by using basic information provided by the National Park Service. This is particularly important for individuals with pre-existing asthma, COPD, or interstitial lung disease. Patients with pre-existing conditions should carry an adequate supply of their control medications and avoid high-risk areas. Guides should consider carrying spare inhalers with a quick-acting beta-agonist and possibly an inhaled corticosteroid. Mechanical injury or dehydration are the most likely situations requiring medical care in the park. Individuals should be advised to prepare their skin for sun exposure, travel with water, wear proper footwear, and maintain an ability to contact help should such an injury prevent self-evacuation. Additionally, visitors into the backcountry should register with the park service before venturing into less trafficked areas of the park.

The levels of gas exposure frequently experienced by hikers and outdoor enthusiasts on Kilauea are not likely to harm an able-bodied individual. It is unlikely the victim or first responder will be able to identify the exact gas that is the offending agent in the field without specialized equipment. Therefore, treatment should focus on maintaining the airway and delivering oxygen. The treatment for exposure to sulfur dioxide is similar to an acute asthma attack. Manifestations can include coughing, bronchospasm, upper airway edema, acute lung injury, and death at exposures above 20 ppm. First line treatment includes immediate removal from the exposure, use of an inhaled beta-agonist agent and oxygen support. An emergency room visit is indicated for observation or further treatment. There is minimal risk of secondary exposure to first responders once the patient is out of the contaminated environment.

For HCl exposure, secondary contamination of the rescuer is not a concern if the victim is dry; moist contaminant could expose the rescuer to vapor. Skin or eye irritation should be decontaminated with removal of clothing and thorough washing with soap and water.
Treatment in Non-Austere Environments

There is no direct antidote for sulfur dioxide toxicity. Treatment consists of support of respiratory and cardiovascular functions. Treat bronchospasm with immediate acting aerosolized bronchodilators such as aerosolized beta-2-agonists. Additional treatment includes long acting aerosolized beta-2-agonists, IV and inhaled corticosteroids, oxygen and even intubation as the clinical picture dictates. The use of aerosolized 2% sodium bicarbonate solution has been documented as a successful treatment and is a consideration in more concentrated exposures. In theory, this helps neutralize and prevent the formation of sulfurous acid and breakdown products which are involved in bronchoconstriction.

For eye irritation due to sulfur dioxide exposure, remove any contact lenses and flush the eyes for at least fifteen minutes. For skin exposure, flush skin with water for fifteen minutes, removing from contact any contaminated clothing and shoes. The clothing and shoes may be washed thoroughly and reused. Patients without pulmonary complaints in a 6- to 8-hour observation period are not likely to develop complications. They may be released and advised to rest and to seek medical care promptly if symptoms develop. For symptomatic patients, the injury may evolve over the course of 18 to 24 hours depending on the level of exposure and co-morbidities of the patient. Treatment for acute hydrogen sulfide toxicity begins with supportive care: removal from the offending site, administration of oxygen and suctioning of secretions. Antidotes to consider if hypoxia is not readily corrected with oxygen include those which will induce methemoglobinemia such as high dose inhaled amyl nitrite and IV sodium nitrate. Inhaled amyl nitrite should be avoided if the individual is hypotensive, because a primary mechanism of action is cardiac and peripheral smooth muscle relaxation. Ten mL of 3% IV sodium nitrite over two to four minutes, and IV pyridoxine are more likely to be more effective in inducing the desired methemoglobin state. The methemoglobin production will scavenge the offending sulfide species, forming a transient sulfmethemoglobin species with a half life of two hours at 24 degrees Celsius which will further decay into oxyhemoglobin and an oxidized form of sulfur.

Other treatments to consider include hyperbaric oxygen and therapeutic red cell exchange. The mechanism of hydrochloric acid toxicity is due to corrosive effects and the formation of free radicals causing parenchymal damage to the lung. The care is supportive. Bronchodilators may be required to counteract bronchospasm. Non-invasive ventilation devices may be required to maintain oxygen saturation and intubation may be required. For pulmonary edema, in addition to oxygen support, loop diuretics are the drug of choice, with furosemide having the added benefit of venodilation effects, reducing preload. For furosemide, start with a dose of 0.5mg/kg, up to 1mg/kg for renal insufficiency or chronic diuretic use. Other agents that reduce preload, such as nitrates, are also effective as long as hypotension is not present or induced. Morphine also induces venodilation and relieves dyspnea and anxiety while reducing catecholamine effects that increase afterload. ARDS is a documented consequence of HCl toxicity that requires extensive supportive care time and resources.

Treat carbon dioxide and monoxide poisoning with supplemental oxygen. The half life of COHb at room air is five hours. Normal COHb levels in the blood are 1-3%, and can be increased in smokers up to 5-10%. Levels measured above this on an arterial blood gas require supplemental 100% oxygen therapy, which will reduce the half life of COHb to 74 minutes. According to Center for Disease Control (CDC) guidelines, COHb levels above 25% or the presence of cardiac or neurological impairment are indications for hyperbaric O2 therapy, which is the gold standard for care and reduces COHb half life to 25 minutes. A literature search did not find the levels of COHb or subgroups of victims that would most likely benefit from hyperbaric oxygen therapy.

Conclusion

While volcanic gas and lava flow injuries are important, the vast majority of park injuries are due to motor vehicle accidents and man-made hazards. Most of the over one million tourists to Hawai‘i Volcanoes National Park report no incidents at all. If the visitor is properly equipped, conditioned, realistic about their health, and willing to follow the signs, they will likely avoid becoming a story in a newspaper. In a 2004 survey of 800 hikers leaving the lava fields, which often involves a 10-plus kilometer hike to view active lava, 93% of respondents admitted hiking beyond warning signs posted 500 meters from the eruption site where the lava meets the ocean. Over half were ill prepared, defined as lacking flashlights, sufficient water, first aid kits, and sunscreen. Many had pre-existing health issues, including heart problems and asthma.

It is prudent to remember to use vehicle safety measures, a hydration plan, protection from the sun, and proper footwear when traversing terrain by foot. Individuals with pre-existing respiratory and cardiac conditions should have a supply of medication, minimize physical stress, and consult the air quality guide for the park at http://www.hawaiiso2network.com/ managed by the US Geological Survey. Finally, obey all signs placed by park personnel that warn visitors of hazards such as unstable terrain and high levels of toxic gases.

Disclosure

The author reported no conflicts of interest.

Disclaimer

The views expressed in this publication are those of the author and do not reflect the official policy or position of the Department of the Army, Department of Defense or the US Government.

Author’s Affiliation:
Department of Medicine, Tripler Army Medical Center, Honolulu, HI 96859

Correspondence to:
Gregory M. Sprowl MD, Tripler Army Medical Center, Honolulu, HI 96859; Email: gregory.m.sprowl.mil@mail.mil
References
Leptospirosis: The Microscopic Danger in Paradise

William A. Londeree MD

Abstract
Leptospirosis is a zoonotic infection endemic in Hawai‘i. This review discusses the incidence of documented human cases in Hawai‘i and current recommendations for diagnosis, prevention, and treatment of leptospirosis.

Introduction
While vacationing or living in paradise, the last thought to pass through a person’s mind is the possibility of contracting a serious or life-threatening bacterial illness like leptospirosis after a quick dip at a beautiful waterfall or a hike near a picturesque stream. *Leptospira interrogans* is a freshwater-borne zoonotic spirochete capable of infecting a variety of mammalian hosts to include cattle, swine, goats, and rodents, but rodents and feral swine mainly serve as carriers in Hawai‘i. Once the infection has occurred in these carrier animals the spirochete will be shed intermittently in the urine where it will remain viable for days to months in soil or water with a neutral pH.1,2

The infection in humans can range from a subclinical infection to severe multi-organ failure. Leptospirosis has an incubation period of 2 to 26 days and classically presents with fevers, rashes, myalgias, and headaches in 75 to 100 percent of patients.1,2 Given its variability in clinical presentation, leptospirosis can be a difficult disease to diagnose, so it is usually treated empirically when suspected. Medications and preventive strategies can decrease the likelihood of acquiring the systemic infection.

Relevance to Hawai‘i
Leptospirosis is endemic worldwide, but the majority of infections occur in the tropical regions with Hawai‘i reporting the highest incidence of human cases in the United States. The Hawai‘i Department of Health reported 345 cases to the CDC in Hawai‘i with 198 cases confirmed from 1999-2008.2 Most of the 91% confirmed cases occurred in men, and 71% of these cases occurred in the age range of 20-49 years. Nearly 80% of these cases were due to freshwater exposure from outdoor recreational activities or taro farming. The remaining 11% percent of exposures were from home gardens.3 Since the incidence of infection in Hawai‘i is higher than the rest of the United States, and the main risk factor for leptospirosis is exposure to freshwater and moist soil, infection rates may decrease if people are educated on use of appropriate personal protection equipment, avoidance of freshwater exposure, and pharmacologic prophylaxis.

The most common infecting serovars are from serogroups Icterohemorrhagiae (40%) and Australis (44%), which have been shown to have predominantly rodent and swine carriers, respectively. In the past, serogroup Icterohemorrhagiae was responsible for the majority of infections, but the increasing rate of Australis infections is most likely related to the increasing population and interactions between feral swine and humans.2

Example Case
A 21-year-old man with no prior significant medical history presented to the emergency department with progressive nausea and headache over the past week. Onset occurred one week after swimming at Maunawili Falls. He started having periumbilical pain and became intolerant of oral intake due to persistent nausea. Non-bloody, non-bilious emesis was followed by brown, watery diarrhea and a subjective fever. Initial vital signs demonstrated a temperature of 103.3°F, blood pressure of 110/66 mm Hg, heart rate of 95 beats per minute, respiratory rate of 18 per minute, and oxygen saturation of 100% on room air. Physical exam was only remarkable for mild diffuse abdominal tenderness without hepatosplenomegaly or peritoneal signs. Diagnostic laboratory studies demonstrated elevated C-reactive protein, erythrocyte sedimentation rate, and aspartate aminotransferase. A normochromic normocytic anemia, thrombocytopenia, and leukocytosis were also present. He was admitted to a telemetry ward and treated with ciprofloxacin for a suspected bacterial diarrheal illness and intravenous doxycycline for a possible leptospirosis infection. Four hours after the administration of antibiotics he developed respiratory distress and had to be intubated for hypoxic respiratory failure. A chest X-ray demonstrated pulmonary edema, and shortly after intubation he developed bloody sputum which was suctioned through his endotracheal tube. Diffuse alveolar opacities were seen on CT scan, and bronchoscopy with a bronchoalveolar lavage demonstrated only gross red blood cells consistent with diffuse alveolar hemorrhage. He was extubated after three days of mechanical ventilation and discharged three days later. Microscopic agglutination testing demonstrated a positive, 1:800 IgM antibody titer for *Leptospira interrogans* serovar Icterohemorrhagiae. At follow-up, he had made a complete recovery.

Presentation and Diagnosis
Cases of leptospirosis can range from a mild or subclinical infection to a very severe and potentially fatal one. Most mild cases usually present with fevers, myalgias, and headache similar to an acute viral febrile illness, but severe cases can present with jaundice, renal failure, hemorrhagic diathesis, anemia, hyponatremia, hypokalemia, and thrombocytopenia.2,3 Diagnosis of leptospirosis relies on a detailed history and clinical suspicion, because the current testing modalities have a long turnaround time and are not clinically useful in an acutely ill patient. The gold standard test is the microscopic agglutina-
tion test (MAT), which uses antigens from live spirochetes from multiple serogroups and mixes them with a patient’s serum to detect concentrations of agglutinating antibodies. One downfall of this test is its prolonged turnaround time. Another downfall of the MAT is the requirement for maintenance of live cultures of Leptospira which are endemic to the local area. Also, a single MAT test cannot differentiate between a current or recent prior infection, and there is cross reactivity with other previous spirochete infections. 4 Titer range for test positivity is open to debate, but is typically set at a value > 1:200. Enzyme-linked immunosorbent assay (ELISA) is another test showing promise because it is able to detect an earlier rise in IgM antibodies. The test has variable specificity and sensitivity due to operator expertise, so it is currently not recommended. 2 5 6 Polymerase chain reaction (PCR) is another test undergoing development but it is best when it is utilized in conjunction with ELISA. Combining ELISA for IgM detection, followed by PCR to detect the antigen in the blood or in the urine to confirm an active infection, is currently being evaluated. 7 8

Prevention
Prevention starts with avoiding high risk exposures to infected water sources. There are roles for chemoprophylaxis and vaccination of animals; however, there is no currently approved human vaccine for use in the United States.

Vaccination faces many challenges because leptospirosis has more than 200 pathogenic serovars. China and France are the only countries that have a vaccine available for human use. A difficulty with developing an efficacious vaccine is matching the local serovars to the vaccine. Limited safety data are available on human use. 7 8 Even though there is no vaccination available for residents of Hawai’i, pharmacological prophylaxis strategies are available.

The United States Army assessed 940 US soldiers during deployed jungle training in Panama with doxycycline prophylaxis 200 mg a week orally versus a placebo. The soldiers receiving doxycycline had an infection rate of 0.2% compared to a 4.2% infection rate experienced by the placebo group, yielding an efficacy of 95%. 11 Since the soldiers were likely a naïve population, studies have also been conducted on indigenous populations to assess prophylaxis efficacy. Two studies assessed indigenous populations from Brazil and North Andaman with doxycycline prophylaxis (200mg weekly) versus placebo. The Brazilian study found a protective association with doxycycline and seroconversion, but it was a small pilot study and did not demonstrate statistical significance. 12 The study from North Andaman included 782 persons and it revealed an infection rate of leptospirosis was not statistically different from the placebo group, however, there was a significant protective effect in reducing morbidity and mortality in the doxycycline group. 13 In contrast to these studies, a systematic review conducted in 2000 did not demonstrate certainty for leptospirosis prevention with doxycycline, but it could not make an argument against prophylaxis. 11 14 The World Health Organization recommends physicians consider using prophylactic doxycycline in highly endemic areas and in areas after natural disaster where flooding or contaminated bodies of water are present. 15

Exposure to freshwater or moist soil which has been contaminated with spirochetes from infected mammalian urine is a major risk factor for contracting leptospirosis. Humans can help prevent infection by avoiding exposure to stagnant water, properly draining farm water runoff, and keeping food away from animal waste contamination. Vaccination of domestic animals has been extremely effective; however, some immunized animals still acquire the infection and shed spirochetes in their urine because the infection resides in the renal tubules. These carriers must be treated with antibiotic therapy in order to clear their infection but this is not practical due to the animals continued exposure to leptospirosis. 16 Several control measures are available for humans. Prevention education in high risk areas is important. Controlling the disease in domestic animals by vaccination is useful. Additional measures include limiting exposure to feral swine, rodent control, and protection of food from animal contamination (either domestic or feral). Doxycycline is available as chemoprophylaxis for people at increased risk of exposure. 15

Treatment
The clinical efficacy of antimicrobials in a mild leptospirosis infection is not well established and remains a topic of controversy. A meta-analysis demonstrated that there may be decreased duration for the clinical illness by 2-4 days, but the results were not statistically significant. 17 Given the risks associated with the severe form of infection, a patient should be treated with appropriate antibiotic therapy as soon as possible, preferably within five days of symptom onset. 15

Penicillin is recommended for a severe leptospirosis infection by the World Health Organization (WHO), but usually stronger antibiotics are given in the hospital in order to broaden coverage. Worldwide, leptospirosis has not demonstrated a pattern of resistance, and it is susceptible to a large variety of antibiotics. Antimicrobial activity against leptospirosis has been observed during in vitro studies with penicillins, cephalosporins, tetracyclines, chloramphenicol, fluoroquinolones, macrolides, telithromycin, carbenapens, and aztreonam. In 2008, Ressner, et al, published a comprehensive review of antimicrobial susceptibilities in different geographic regions. Overall, the study analyzed 13 Leptospira isolates from Egypt, Thailand, Nicaragua, and Hawai’i. Among thirteen antimicrobial agents tested, ampicillin, cefpime, azithromycin, and clarithromycin had the lowest MICs (<0.016mcg/ml), with slightly higher MICs recorded for cefotaxime, ceftriaxone, imipenem-clastatin, penicillin G, moxifloxacin, ciprofloxacin, and levofloxacin (0.030- 0.125 mcg/ml). Overall, the highest MICs were for doxycycline and tetracycline at 2.0 and 4.0, respectively, for strains obtained in Egypt. Icterohemorrhagiae was the Hawaiian serovar analyzed in this study and its MICs to doxycycline and tetracycline were the highest when compared to the other antibiotics at 0.50 and 1.00, respectively. 18

Most cases of leptospirosis are subclinical and will never
Introduction

Leptospirosis is a bacterial disease caused by Leptospira interrogans, which can be transmitted to humans through contact with infected animals or water contaminated with their urine. Symptoms can range from mild to severe, and in some cases, patients may develop a Jarisch-Herxheimer reaction (JHR) upon initiation of antibiotic therapy. The JHR is characterized by a variety of symptoms including fever, chills, and other manifestations that may be confused with an acute inflammatory reaction.

Patients at Risk

Individuals at risk for leptospirosis include those with occupational exposure to infected animals or water, as well as those who engage in activities that put them at risk for exposure, such as hiking or swimming in freshwater environments. Pregnant women may also be at risk for severe disease.

Diagnosis

The diagnosis of leptospirosis is typically based on a combination of clinical presentation, laboratory findings, and serology tests. The treatment for leptospirosis should be initiated early in the course of the infection, and often involves the use of antibiotics such as doxycycline or azithromycin.

Conclusions

The best way to avoid a leptospirosis infection is through avoidance of areas of water and moisture which are exposed to feral animals, especially swine and rodents. Patients should be educated by their primary care provider about contact avoidance. Prophylaxis with weekly oral doxycycline should be considered for individuals when exposure is unavoidable. Individuals who elect to forego prophylactic antibiotics should be counselled to seek immediate medical attention if they start feeling ill after an exposure to freshwater. Initiation of antibiotic therapy should be based on history, physical exam findings, and laboratory results that are consistent with possible leptospirosis infection.

Disclosure

The author reported no conflicts of interest.

Acknowledgments

Dr. Marta Guerra and Dr. Sean Shadomy for their guidance on this paper.

References

Ciguatera Fish Poisoning in Hawai‘i and the Pacific

Nathanial K. Copeland MD; Wyatt R. Palmer DO; Paul K. Bienfang PhD

Abstract
Ciguatera fish poisoning (CFP) is a foodborne illness caused by fish containing ciguatoxin (CTX). The toxin is produced by the microalgae Gambierdiscus spp., which are then eaten by reef fish; humans contract the illness when eating either fish that have eaten the algae, or carnivorous fish that have eaten those fish. CTX is an odorless, tasteless, and colorless neurotoxin that blocks voltage-sensitive Na\textsuperscript{+} channels and accumulates in many tissues of the fish, especially the viscera. The illness is typically mild to moderate in severity with gastrointestinal (diarrhea, cramping, nausea, vomiting) and neurological (paraesthesias, cold allodynia, fatigue, pruritis) manifestations. Rarely, the disease can be more severe with significant neuropathic or cardiac effects such as bradycardia and hypotension. Endemic to Hawai‘i and islands throughout the Caribbean and Pacific, CFP incidence rates range from several to thousands of cases per 100,000 per year. Since fishing is important for local food supply, exportation, and recreation throughout the Pacific, CFP is medically and economically significant in these areas. We present a case of CFP from Hawai‘i to illustrate the disease, demonstrating that the diagnosis is primarily clinical, with confirmatory tests from fish samples available in some cases. Treatment is supportive and symptomatic with no disease specific remedy. The prognosis for most cases is good with a short duration of self-limited symptoms, but for some cases neurological sequelae can become chronic. With no effective treatment, education on which species of reef fish and which body parts to avoid eating is essential in the prevention of CFP.

Introduction
Ciguatera fish poisoning (CFP) is a foodborne illness contracted by humans eating reef fish containing ciguatoxin (CTX) and is characterized predominantly by gastrointestinal and neurological manifestations. It is the most common marine poisoning in the world\textsuperscript{1,2} and is a significant public health concern in the Caribbean, Hawai‘i, and the Pacific islands.\textsuperscript{3,4}

The CTX toxin is produced by the microalgae Gambierdiscus spp., which are ingested by herbivorous reef fish that are then eaten by larger carnivorous reef fish and serve as the primary source for human illness.\textsuperscript{1,2} The toxins are lipid soluble and bioaccumulate in greater concentrations as they move up the food chain, making the larger carnivorous fish more likely to cause harm to humans.\textsuperscript{3} However, consumption of any fish in the coral reef food chain can cause disease.\textsuperscript{1} The toxin is colorless, odorless, and tasteless and is not affected by any food storage or preparation techniques.\textsuperscript{6} It is the one of the most potent Na\textsuperscript{+} channel toxins known in mammals, causing activation of voltage-sensitive Na\textsuperscript{+} channels at very small (<1 part per billion) concentrations, leading to its multiple clinical manifestations as discussed below.\textsuperscript{2} Reef fish in particular are associated with CFP, as these are the fish involved in the food chain of the Gambierdiscus spp. Reef fish commonly associated with CTX are many but include: barracudas, groupers, jacks, moray eels, snappers, parrotfish, and surgeonfish.\textsuperscript{2,6,7} Due to its lipid solubility, CTX accumulates more heavily in several places in fish including the brain, liver, and gonads. Anecdotally it has been noted that more severe cases of CFP reported to the Hawai‘i Department of Health involve consumption of the head (brain) or organs of the fish and less severe cases tend to involve consumption of only the muscle.

The manifestations of CFP are predominantly gastrointestinal and neurological, but also include other rare but potentially dangerous effects.\textsuperscript{1,2} Effects typically begin within a window of <1 to 48 hours from ingestion, with gastrointestinal symptoms occurring typically within 12 hours and neurological symptoms developing over the first 24 hours.\textsuperscript{3} Gastrointestinal effects include diarrhea, abdominal pain, nausea, and vomiting. These have the potential for significant dehydration, but are typically short-lived. Neurological effects include perioral and stocking-glove distribution paraesthesias, myalgias, numbness, cold allodynia (burning pain caused by exposure to cold temperature), fatigue, pruritis, and rarely coma.\textsuperscript{1,2} Neurological symptoms can be variable in course, with most resolving in days to weeks, though some can persist for years and are reported to recur with exposure to non-CTX containing triggers (such as consumption of chicken or alcohol).\textsuperscript{2} Rare but significant cardiovascular effects do occur, including bradycardia and hypotension.\textsuperscript{1} Typically moderate, CFP can rarely result in death.

An endemic problem in Hawai‘i and many of the Pacific Islands,\textsuperscript{1,2,4} cases of CFP are reported to the Hawai‘i Department of Health, with a total of 3 to 69 cases per year in the state, averaging 28.5 total cases per year from 2002-2011.\textsuperscript{4} With underreporting of milder cases, it is likely that the actual local incidence is anywhere from 10 to 20 times higher than reported. There have been two reported deaths from CFP in Hawai‘i occurring in 1964 after eating broiled reef fish including their viscera.\textsuperscript{4} There is a broad range of incidence across the Pacific islands, with Samoa at about 1.6 cases per 100,000 per year and the Cook Islands at nearly 1,500 cases per 100,000 per year.\textsuperscript{3} Fish are an important natural resource throughout the Pacific and fishing is a very common means of subsistence, putting many people at risk for CFP. Reef fishing also represents a common sport in Hawai‘i, endangering both visitors and the local population. Based on data from the Hawai‘i Department of Health, the five most common fish associated with cases of CFP in the state from 1963 to 2012 are jack (ulu, kahala, or papio), surgeon fish (kole), groupers (roi or hapu‘u), snappers (lehi, onaga, taaape, or waha-nui), and wrasses (Figure 1; Hawai‘i State Department of Health, unpublished data, Oct 2013). There are many local myths about how to determine which fish are safe, including avoiding fish from the leeward side of the island. From 2008 to 2012 researchers from the University of Hawai‘i cooperated with local fishermen to conduct
surveillance studies of roi caught across the state of Hawai‘i, testing the fish for the presence of CTX. The map in Figure 2 represents the cumulative data from the island of O‘ahu. The study shows that no coast of any Hawaiian Island is free from CTX-positive fish and it is speculated that any relationship to the leeward side of an island is more likely due to which coast is more commonly fished rather than the presence or absence of CTX positive fish.

Example Case
In 2009 the Honolulu Advertiser reported a case of a previously healthy 53-year-old man who developed a severe case of confirmed CFP after consuming a reef fish caught in Hawai‘i. The man and his wife both consumed a knifejaw fish that the man had caught while spear fishing in a reef off Kaua‘i. Both he and his wife had onset of typical ciguatera symptoms, including gastrointestinal upset and paraesthesias, within a few hours of consuming the fish. His wife’s case was relatively mild and short in duration, but he progressed to develop more severe neurological manifestations including severe neuropathy. He was unable to walk, speak, or feed himself and was hospitalized for more than 2 months. Remnants of the fish that the patient ate were tested by the Department of Health and found to be positive for CTX.

Figure 1. Ciguatera incidents by type of fish consumed, Hawai‘i (1963-2012) (image courtesy Hawai‘i State Department of Health)
Discussion
A diagnosis of CFP is clinically based on the presence of typical gastrointestinal and neurological symptoms and history of ingestion of a likely CTX containing fish. There are currently no blood tests available to confirm poisoning with CTX.\(^1\)\(^2\) The current gold standard for confirming the diagnosis is the testing of fish remnants for CTX (when they are available), as illustrated in the case above.\(^1\) Treatment of CFP is supportive, with symptomatic treatment of gastrointestinal manifestations and supportive care in more severe cases with bradycardia and hypotension.\(^2\) There are no proven treatments for neurological manifestations. Mannitol has shown promise in ameliorating the neurological sequelae, but the only double-blind, randomized controlled trial to date showed no difference when compared to normal saline and highlighted concerns for hypotension caused by mannitol.\(^2\)\(^6\) Some limited data suggest that neuromodulating medications such as amitriptyline may be beneficial, but controlled trials are lacking.\(^2\) While no specific remedies are available in austere environments to prevent or treat CFP, there are many local remedies throughout the Pacific Islands such as plant extracts that are unproven by research.\(^1\)\(^7\) One common traditional remedy employed stems from the tree heliotrope, which is found in Hawai‘i and throughout the Pacific Islands; no studies confirming its efficacy have been published.

Most cases do not cause long term effects; however, there are some patients that will have persistent neurological symptoms, including neuropathy, fatigue, or pruritis chronically. There is also evidence of sensitization, with affected individuals having more rapid onset of symptoms upon subsequent exposure to the toxin.\(^2\)

With no specific treatment available for CFP, prevention is a key component to the management of this disease, especially in endemic areas such as Hawai‘i and the Pacific islands. The most effective approach to preventing CFP is to avoid reef fish which are more likely to contain CTX. Fish containing CTX cannot be reliably distinguished from safe fish based on appearance, taste, or smell. Some traditional methods exist in Hawai‘i and the Pacific Islands for determining which fish may be at risk for causing ciguatera, including feeding it first to the family pet or the oldest member of the family, or avoiding fish that flies avoid, but these methods cannot be recommended as they are neither safe nor reliable. There is a common belief that people should avoid larger individual fish of a given species, but any size of fish can potentially harbor sufficient amounts of CTX to elicit symptoms.\(^9\) In the past commercially-produced, portable CTX detection kits were available to test fish prior to eating, but their production has been discontinued due to a significant number of false negative tests.\(^12\) People should avoid consumption of the liver, brain, or gonads of reef fish, as these tissues sequester much higher levels of CTX than the muscle tissue.

The key to prevention is educating the populace about the risks of eating reef fish in endemic areas. In cases where this is not
feasible, such as in areas where reef fish are a primary source of subsistence, education about avoiding consumption of the viscera (which have a higher concentration of the toxin) is also important.

**Conclusion**
Ciguatera fish poisoning is an important fish-borne disease in Hawai‘i and the Pacific islands. Though most cases have mild to moderate gastrointestinal and neurological symptoms, the potential for more severe and even life-threatening illness exists. There is significant variation in the incidence of the disease depending on location and it is important for travelers to these areas to be familiar with the local risk of CFP and to make decisions about eating reef fish accordingly. Diagnosis is clinical, with confirmatory fish tissue sampling. Treatment is supportive and symptomatic with no disease-specific remedy proven. Due to the lack of treatment options, prevention via avoidance of likely CTX containing fish is essential, or at a very minimum avoiding the head and viscera of reef fish.

**Disclosures**
The authors report no financial conflicts of interest.

**Disclaimer**
The views expressed in the manuscript are those of the authors and do not reflect the official policy or position of the Department of the Army, Department of Defense or United States Government.

**Acknowledgment**
The authors would like to thank the Hawai‘i Department of Health’s Disease Investigation Branch for providing data on the incidence of ciguatera by type of fish consumed and the graph used in Figure 1.

**References**
The Potential Danger of Eating Wild Lettuce: A Brief Review of Human Rat Lungworm Infection

Evan C. Ewers MD and Sarah K. Anisowicz MD

Abstract
Angiostrongylus cantonensis, the causative agent of human rat lungworm disease, is the most common cause of eosinophilic meningitis worldwide and is endemic throughout Asia Pacific. It is acquired through the consumption of infected freshwater mollusks or contaminated produce. Human angiostrongyliasis is usually a self-limited disease presenting with headache and various neurologic sequelae varying from cranial nerve palsy to radiculitis and/or paresthesias. Fatal cases are rare, and manifest as fulminant meningomyeloencephalitis. The diagnosis is made through the use of clinical history, exam, and laboratory data including peripheral blood counts, cerebrospinal fluid (CSF) examinations, and serologic or molecular diagnostic techniques. Medical therapy is largely focused on symptomatic relief, and includes analgesics, lumbar puncture, and corticosteroids. In resource-limited settings, prevention is key, and the use of analgesics can provide symptomatic relief after infection. Efforts to increase disease awareness have been made in endemic areas, as evidenced by the recent Rat Lungworm Disease Scientific Workshop which was held in Honolulu in 2011. The proceedings of the workshop were published in a supplement to this journal (Hawaii J Med Public Health. Jun 2013;72(6):Supp 2). However, wilderness medicine and travel medicine specialists must also be aware of the disease, how it is contracted, its presentation, and treatment options should they encounter a patient who is in or has returned from an endemic area. This brief review highlights eosinophilic meningitis caused by A. cantonensis, including an example case, an overview of its clinical presentation, treatment options, and prevention.

Background
The parasitic nematode Angiostrongylus cantonensis is the causative agent of rat lungworm disease, and the most commonly identified cause of eosinophilic meningitis worldwide. Although first isolated from the pulmonary artery of a rat by Chen in 1935, it was not identified as a human pathogen until it was isolated from a case of eosinophilic meningitis in Taiwan ten years later.1 Approximately 2900 cases of eosinophilic meningitis have been reported in the literature since 1944 as a result of rat lungworm infection, although it is believed this number is largely underreported, as many infections may be subclinical or occur in areas where specific diagnostic testing is not routinely performed.2 Today, rat lungworm is considered endemic in most parts of the Asia-Pacific region, and new cases are now routinely being identified in previously non-endemic areas.

Much is unknown about specific pathogenic mechanisms involved in human rat lungworm infection producing clinical disease, but the biology and life cycle of A. cantonensis are well described in the scientific literature.2,3 Angiostrongylus cantonensis is a member of the family Angiostrongylidae in the superfamily Metastrongyloidea, of which only 20 species in the genus Angiostrongylus are known. Of these, two species—A. cantonensis and A. costaricensis—cause disease in humans, although the latter is only associated with a gastrointestinal illness most prevalent in Central and South America. The definitive hosts of A. cantonensis include the rat species of Rattus norvegicus, R. rattus, and R. exulans, which acquire the infection through ingestion of third-stage (L3) larvae by eating a slug or snail. Ingested worms travel from the rat’s gut to the bloodstream, eventually making their way to the central nervous system. In the brain, they develop into the fifth stage (L5) young adults, and travel via the cerebral venous system and make their way to the pulmonary arteries, where the females lay eggs. The eggs make their way to the distal lung, where they hatch into first-stage larvae (L1), extravasate, and migrate up the trachea where they are swallowed and excreted in the rat’s feces. Once in the environment, the L1 larvae mature into L3 larvae, and are ingested by a snail, slug, or other intermediate or paratenic host.3 If the snail or slug is consumed by a rat, the life cycle is repeated. However, the infection of accidental hosts, such as species of crustaceans, frogs, monitor lizards, and planaria results in larvae remaining in the L3 stage inside that animal, which makes them capable of infection once consumed by rats, humans, or other mammals. If consumed by a human, the larvae make their way to the CNS, but are unable to adequately mature to complete their life cycle.

Human infection with rat lungworm results from the ingestion of either infected snails, prawns, crabs or contaminated fruits and vegetables. Since it was first identified as a human pathogen in 1944, a number of outbreaks of eosinophilic meningitis occurring in China, Taiwan, Japan, Southeast Asia, Australia, Hawai‘i, and the South Pacific have been attributed to A. cantonensis, and cases have been identified on the six inhabited continents.2 Although the increasing identification of clinical cases is likely multifactorial, it is widely accepted that international commerce and shipping have contributed to the nematode’s spread through the transportation of either infected rats, snails, slugs, or other hosts. Recently, A. cantonensis has been found to be the etiologic agent of eosinophilic meningitis in a number of travelers returning to northern climates from endemic areas.4 This has highlighted the importance of proper education and increased awareness of both travelers and physicians. Travelers who enjoy camping, consuming wild foodstuffs, or sampling local or cultural cuisine in endemic areas should be aware of the potential risks, and how to mitigate them.

Relevance to Hawai‘i and Asia Pacific
Angiostrongylus cantonensis has been well-established as a causative agent of eosinophilic meningitis in Hawai‘i since 1960, although it did not become a reportable disease in the state of Hawai‘i until 2007.7,8 Hochberg, et al, identified 24 cases of eosinophilic meningitis caused by A. cantonensis in...
Hawai‘i between 2001-2005, with cases reported in the islands of O‘ahu, Maui, Lana‘i, and the island of Hawai‘i, which had the highest incidence. Since 2007, 33 cases of eosinophilic meningitis due to A. cantonensis have been reported in the state. In many of these cases, patients have consumed either contaminated vegetables or snail species acting as intermediate hosts. In Hawai‘i, several species of snails have been identified as carriers of the rat lungworm nematode, including the giant African snail Achatina fulica, and the relatively new, invasive semi-slug Paramarion martensi, which is now abundant throughout the Hawaiian archipelago. Environmental evaluation of mollusk species on the islands of O‘ahu and Hawai‘i have demonstrated infection rates ranging from 24-88% depending on species and location.

A. cantonensis has also been identified as the causative agent of eosinophilic meningitis throughout the South Pacific since the early 1960s, with cases reported in French Polynesia, Samoa, and the Marshall Islands between 1961 and 1964. Numerous outbreaks have occurred in Asia, particularly in the northeastern provinces of Thailand, and throughout inland China and Taiwan, where cases are still routinely documented. Of the nearly 2900 cases that have been reported in the medical literature, approximately 75% have been reported from Thailand and China. Many cases in these regions result from the consumption of raw snails in cultural dishes, or consumption of contaminated raw vegetables or their juices. For visitors, it is important to be mindful that in certain areas eating local cuisine can result in serious illness. This should also serve as a reminder to travel medicine specialists to discuss potential risks with patients traveling to these areas, particularly if they are planning on camping, or immersing themselves in local culture.
### Example Case

A seven-year-old previously healthy boy re-presented to a pediatric clinic with his mother for headache, vomiting, and fatigue. The patient was in his usual state of health until eight days earlier, when he complained of a headache which improved following administration of acetaminophen. The mother noted that the patient seemed more fatigued, preferring to watch television instead of playing with his older siblings. Seven days earlier he began to have intermittent, non-bloody, non-bilious vomiting. Five days prior he was evaluated in the clinic for headaches and vomiting, and diagnosed with gastroenteritis. His mother was instructed to encourage increased fluid intake, and return should his symptoms not resolve. At the time of representation, the mother denied any fevers, rashes, diarrhea, or upper respiratory symptoms.

On exam, the patient was a non-toxic appearing male who was alert, although slightly irritable. Vital signs included temperature of 38.6°C, blood pressure of 104/64, heart rate 98, and respiratory rate of 22. Mucous membranes were dry. The patient was protective of his neck and would not voluntarily move it. Passive range of motion of the neck was made difficult by patient non-compliance with exam. The patient was admitted for mild dehydration and concern for meningitis. Complete blood count showed a leukocytosis of 14,000, hemoglobin of 14.3 g/dL, hematocrit of 41%, and platelets of 420,000. Leukocyte differential was notable for eosinophilia of 18%. Lumbar puncture was unremarkable with protein of 60 mg/dL, glucose of 70 mg/dL, no red blood cells and few eosinophils without organisms on gram stain. Notably, the patient had decreased complaint of headache after the procedure. On day 2 of admission, patient complained of worsening headache, and a repeat lumbar puncture resulted in symptomatic relief of the patient’s headache. Test performed on CSF were negative for enterovirus, herpes simplex virus, syphilis, and bacterial organisms.

Further history taken during admission revealed he ingested a snail from the family garden after being “dared” by his older brothers three days before the onset of symptoms. Based on this history and continued peripheral eosinophilia throughout admission, he was diagnosed with eosinophilic meningitis. At the time of discharge, the patient continued to complain of headache which was controlled with oral ibuprofen and acetaminophen. He was followed closely by his outpatient provider and did not develop any further neurologic symptoms with resolution of headache by one month after admission.

### Clinical Discussion

As in this example case above, ingestion of larvae occurs either by direct consumption of contaminated mollusk species, or through the ingestion of contaminated produce. In Hawaii, both kinds of exposures have been reported in the clinical literature, and similar exposures resulting in outbreaks in Thailand, China, and the South Pacific. Incubation periods typically last from 5-40 days post exposure. In a study of eosinophilic meningitis in Thailand, 83% of patients reported symptom onset by 20 days following ingestion of a raw freshwater mollusk, with the majority of patients developing symptoms by two weeks following the inciting event. However, incubation periods as short as one day have been reported.

The manifestations of the infection are primarily localized to the central nervous system, where it is known to cause mild, subacute, or fulminant eosinophilic meningitis. Rarely, ocular manifestations can occur with nematode invasion in the 3 major chambers of the eye. The most common presenting symptoms of human angiostrongyliasis and various clinical manifestations have been well-described in case reports, series, and epidemiologic studies on the disease. In cases, the presenting complaint, typically occurring in greater than 90% of those afflicted. Headache, as well as nausea and vomiting, is attributed primarily to increased intracranial pressure, or direct neuronal damage caused by both dying larvae and the host immune response. In some cases, serial lumbar punctures have resulted in symptomatic decrease of a patient’s headache, although these have not been associated with curing infection. Subjective neck pain and stiffness, fatigue, fever, visual complaints, and paresthesias develop between 30%-50% of patients, occurring in the early course of the disease. The paresthesias experienced are often profound, and range from burning and tingling, to symptoms reflective of an acute painful radiculitis with hyperesthesia. Neurologic symptoms and signs can also result in cranial nerve palsies, and rarely, altered sensorium. Upper respiratory symptoms including sinus congestion, rhinorrhea, sore throat, and dry cough can be seen early in the disease course. In ocular cases, the presenting complaint is usually blurred vision developing over several weeks. Overall, the clinical presentation of A. cantonensis varies broadly, and can include mild self-resolving symptoms to fulminant meningomyeloencephalitis resulting in coma and permanent neurologic deficits. Death has been reported in only a few cases. These catastrophic cases are associated with heavy infections found to have large nematode burden and advanced inflammatory reaction involving macrophages, T-cells, eosinophils, and neutrophils on pathologic review.

Physical findings are related to the onset of neurologic sequelae and can include decreased reflexes, bowel or bladder dysfunction, muscle weakness, cranial nerve deficits, and evidence of increased intracranial pressure. Non-suppurative pharyngitis and cervical adenopathy have been reported. Meningeal signs are inconsistently reported in association with eosinophilic meningitis. In cases of ocular angiostrongyliasis, living larvae can be observed in the anterior, vitreal, or retinal chambers, with accompanied findings of epithelial alteration and subretinal tracking.

Diagnosis of rat lungworm infection is often difficult to establish. Typically, a combination of patient history, symptoms, clinical findings, and laboratory tests are required to make the diagnosis. The “gold” standard is isolation of the nematode from the CSF, occurring in only 2-12% of cases. However, it should be noted that nematode isolation from CSF is more common in pediatric patients than in adults. Absolute peripheral eosinophilia (> 450/µL) as seen on CBC, particularly in the...
setting of leukocytosis, and the presence of eosinophils in CSF are frequently seen in human infections. Chemical examination of CSF also demonstrates elevated protein, and normal to slightly decreased glucose. \(^{18-20,22-26}\) Serologic studies can also be employed in the diagnosis. Tsai, et al, reported the presence of anti-Angiostrongylus antibodies in the serum in 100% of patients as well as in the CSF of 80% of patients, indicating that even during acute infection serologic testing plays an important role in assisting with diagnosis. \(^{19}\) Recently, gene amplification using polymerase chain reaction has been employed to identify the presence of nematodes in both serum and CSF. \(^{24}\) As molecular and point-of-care diagnostic techniques for parasitic diseases improve in the coming years, rapid definitive diagnosis using CSF samples might become possible, and have great utilization in endemic areas.

Since the 1990s, advances in neuroradiology has also played a role in assisting with diagnosis of eosinophilic meningitis. Hyperintense areas in white matter seen on T2-weighted images have been observed in patients with human angiostrongylisiasis, as well as post-gadolinium enhancement of subcortical white matter and affected cranial nerves on T1-weighted images. Leptomeningeal enhancement is also frequently seen.\(^{22,24,27}\) Interestingly, Tsai and colleagues observed a significant correlation (\(P<.05\)) between T1 signaling on MRI and clinically more severe headache, as well as CSF and peripheral eosinophilia. To date, there has been no associated pathognomonic radiographic finding in patients with eosinophilic meningitis. However, neuroradiology can be used to support the diagnosis and rule out other potential causes—amebiasis, neurocysticercosis, gnathostomiasis, and paragonamiasis, among others.

**Treatment in Resource-Constrained Environments**

Campers, hikers, farmers, and adventurous eaters are among those individuals who should be educated regarding the risks of human angiostrongylisiasis, including its mechanism of infection and the disease sequelae. Luckily, the vast majority of reported cases have occurred within some proximity to medical attention. The best way to manage eosinophilic meningitis is to limit exposure. This is accomplished by avoiding the consumption of raw freshwater mollusks including snails, slugs, prawns, crab, and even raw freshwater fish. This includes washing fresh fruits and vegetables, particularly lettuces, and ensuring that roadside juice stands are serving products made from properly washed and peeled produce.

Since snails and their slime can be found in and on fresh fruits and vegetables, washing the produce prior to consumption is an important step in the food preparation process. A comparison of salt, bleach, and vinegar solutions demonstrated no significant difference compared with tap water alone when used to remove snails/slugs from produce, although multiple washing/rinsing cycles was found to decrease snail burden.\(^{28}\) However, the use of different household chemicals and treatments has been shown to decrease larval infectivity in rats. Eamsobhana, et al, reported that mixing raw snails in the traditional Thai “Koi-Hoi” marinade consisting of spices and citrus juice resulted in decreased larval infectivity, particularly when a local 80-proof spirit was added.\(^{17}\) In their study, an 80% decrease in motile larvae was seen after 60 minutes in the marinade alone, or 5 minutes in the marinade followed by 30 minutes in the alcohol drink. A 100% non-motile rate was seen when the snails were allowed to marinate for 25 minutes followed by soaking in alcohol for 30 minutes. This serves to demonstrate that household products, or those available to campers and hikers can be used to reduce risk. The use of vinegar, salt solution, and 1.5% bleach solution have also been shown to decrease larval infectivity in A. costaricensis after soaking contaminated produce in the solutions for 15 minutes.\(^{29}\) To our knowledge, this study has not been replicated for A. cantonenensis, and might be a direction for future research as cases of the disease are spreading globally and awareness is increasing.

**Treatment in Non-Austere Environments**

Clinical variability is broad and symptoms often guide medical intervention. Mild cases resolve spontaneously, requiring only symptomatic therapy. For highly symptomatic cases, serial lumbar punctures have been demonstrated to provide symptomatic relief for headache, nausea, and vomiting caused by increased intracranial pressure.\(^{18,22,24-26}\) Antihelminthic therapy is controversial due to the theoretical risk that immune reaction caused by rapidly dying worms can exacerbate patient’s neurologic symptoms and lead to potentially catastrophic results. However, case-series and epidemiologic studies published in which antihelminthic therapy was used have not reported significant adverse reactions directly related to their use, and most of the treated patients have made full recovery.\(^{15,19,20}\) The role of corticosteroids has also been evaluated in the treatment of eosinophilic meningitis, particularly with the goal of reducing cerebral inflammation caused by the immune response. In most situations, corticosteroids have not been shown to shorten disease course.\(^{2,18,19}\) However, Chotmongkol, et al, reported in a randomized, controlled trial that two weeks of corticosteroids decreased headache duration when compared with placebo, as well as decreasing repeat lumbar punctures in the experimental group.\(^{20}\) Additionally, the same author demonstrated their safety for use in patients with eosinophilic meningitis.\(^{31}\) Corticosteroids have also been evaluated when used concurrently with antihelmintics such as albendazole. In their study, Chotmongkol, et al, found that adding albendazole to the corticosteroid regimen did not result in shortened disease course or improved symptomatic relief when compared with steroids alone.\(^{32}\) Importantly, no adverse drug outcomes were reported in their study for either the control or treatment groups.

Based on the available evidence, there are no current official recommendations regarding treatment of eosinophilic meningitis caused by A. cantonenensis. Generally, it is accepted that the use of corticosteroids (prednisolone at 20mg three times daily for 14 days) might provide some benefit in symptomatic reduction, with minimal adverse effects. The use of albendazole or another antihelminctic agent remains controversial due to theoretical
risks, although these have not been demonstrated in limited clinical studies. However, their use is usually not required and should be reserved for cases non-responsive to symptomatic management, or those with severe systemic effects.

**Conclusion**

*Angiostrongylus cantonensis* is the most common cause of eosinophilic meningitis worldwide, and is endemic throughout the Asia-Pacific region. It has been shown to cause disease in returning travelers. Travelers to endemic areas, particularly those who camp, eat local cultural cuisine, consume raw freshwater mollusks, eat raw fruits and vegetables are at risk of acquiring the nematode infection. Human angiostrongyliaisis has a variable clinical presentation that can range from a short, self-limited illness, to fulminant meningoencephalomyelitis, or encephalitis, with permanent neurologic sequelae and death. The mainstay of treatment includes management of headache through LP, analgesics, and potentially steroids or antihelminthic agents, although the role of the latter is controversial.

**Disclosures**

The authors reported no conflicts of interest.

**Disclaimer**

The views expressed in this abstract/manuscript are those of the author(s) and do not reflect the official policy or position of the Department of the Army, Department of Defense, or the US Government.

**Acknowledgements**

The authors would like to thank the Editors of the Wilderness Medicine Supplement for considering our article. We also wish to thank the library staff at Tripler Army Medical Center for assisting with the literature review, as well as our reviewers for providing insightful comments and suggestions.

Authors’ Affiliation: Department of Medicine, Tripler Army Medical Center, Honolulu, HI 96859

Correspondence to: Evan C. Ewers MD, Department of Medicine, Tripler Army Medical Center, Honolulu, HI 96859; Ph: (808) 433-9772; Email: evan.c.ewers.ml@mail.mil

**References**

Heat Illness in Hawai‘i

Sarah Gordon MD

Abstract
Heat illness is a commonly encountered health problem in the Hawaiian Islands. Year round warm temperatures, proximity to the equator, and high humidity combined with a plethora of opportunities for outdoor activities put many individuals at risk. This paper will focus on the physiology, identification, and treatment of varying forms of heat illness. Severe heat illness can be life threatening. All outdoor enthusiasts should have a basic understanding of how to recognize this potentially life-threatening condition and employ preventive measures. We will discuss appropriate management in pre-hospital and hospital settings. Early recognition and cooling are the most crucial aspects of the management of heat illness.

Background
The human body’s temperature is regulated in the pre-optic nucleus of the anterior hypothalamus, which in normal physiology is set at a core temperature of 98.6° F ± 2 degrees (37 C ± 1 degree). Outside of this range, the human body has the ability to tolerate significantly colder temperatures, but at warmer temperatures, particularly above 105° F, physiologic dysfunction occurs.

There are a variety of changes that occur on a cellular level in extreme heat. Elevated temperatures affect the exchange of ions across transport membranes in all cells. This process, coupled with alterations in renal blood flow, and changes in plasma volume, can lead to a variety of electrolyte derangements. Hyperthermia causes changes in the structure of cellular organelles including disruption of microfilaments and swelling of the mitochondria and the endoplasmic reticulum, leading to collapse of the cytoskeleton and deformation of the plasma membrane. Reaction kinetics dictate that rate increases with heat, however, above a certain temperature reactions are unable to occur since the responsible enzyme begins to denature.

To prevent heat illness, the body has cooling mechanisms to remove heat. Evaporation, radiation, convection, and conduction are the four main processes which reduce heat energy. Evaporation occurs when water vaporizes from the skin and respiratory tract. This is the body’s most effective mechanism for getting rid of heat (an example would be an athlete sweating in the hot sun). Radiation occurs when heat is directly emitted into the environment. Convection is the transfer of heat to a liquid overlying the body (eg, a swimmer cooling off in the water). Conduction is direct transfer of heat to a cooler object (eg, application of an ice pack).

These processes all require a cardiovascular system that is able to increase blood flow to the distal vasculature to facilitate transfer of heat from the body core to the skin, where the four mechanisms for dissipating heat can take effect. During high heat loads, blood flow to the skin increases drastically to enable these processes. However, when the ambient temperature is higher than the body’s core temperature, convection, conduction, and radiation are no longer effective. Environmental conditions also affect evaporative cooling. A water vapor pressure gradient must exist for sweat to evaporate and release energy into the environment. In high humidity evaporation becomes ineffective for transferring heat (typically around relative humidity >75%). Intrinsic factors that decrease ability to compensate for generation of heat energy include age (both very young and very old individuals), stimulants, poor cardiovascular fitness, and possibly the most modifiable risk factor, dehydration. Studies suggest that during intense exercise in the heat for every one percent of body mass lost from dehydration there is a concomitant increase in core body temperature of 0.22° C.

The human body also has significant adaptive cellular mechanisms in place to counteract heat illness. A highly conserved response to physiologic stress including hyperthermia and hypoxia exists in nearly all prokaryotic and eukaryotic cells. This process is largely mediated by heat shock proteins. These are transcriptionally inducible molecular chaperones. They work to prevent the formation of damaged protein aggregates and assist proteins in the acquisition of their native structures. It is theorized that they are a physiologic response to cellular stress, rather than a prophylactic process already in place. Studies have shown those with high basal levels of heat shock proteins are more likely to experience acute heat induced illness. These proteins may represent a biomarker to assess susceptibility to heat stress. When these defenses are overwhelmed by either passive heat accumulation (classic heat stroke) or by generation of excessive heat energy via activity (exertional heat stroke), heat illness occurs.

Relevance to Hawai‘i and Asia-Pacific
Heat illness on a wide spectrum is a common threat to people who spend time outdoors in Hawai‘i. On all the islands, May through October is considered summer and winter the rest of the year. Honolulu is a well studied example of a sea level climate, where summer highs average 85° F while winter highs average 78° F. Due to proximity to the equator the UV index is almost always high, with an average index of 6-7 in the winter and 11-12 for summer months. Recent research suggests that temperatures will be increasing for the Hawaiian Islands in coming years. In Honolulu, Hawai‘i, the average temperature has increased 4.4° F over the last century and research suggests it will continue to increase, while precipitation has decreased approximately 20% over the last 90 years.

Due to year round warm temperatures and sunshine, Hawai‘i is a popular tourist destination. There are many outdoor activities which require exertion, including hiking and water sports, both of which result in prolonged sun exposure. Non-acclimatized or
poorly conditioned individuals, as well as young children and the elderly are particularly at risk of experiencing heat illness under these conditions.

In addition, there are numerous organized sporting events. A glance at a calendar for one of Honolulu’s sporting events organizations shows an average of eight organized races of various distances per month. The Big Island of Hawai’i is home to the world championship ironman triathlon, the ultimate test of endurance. Participants in these events are typically well conditioned and acclimatized, yet a determined athlete may ignore warning signs.

Finally, Hawai’i has a large military population. The military has classically been a leading source of literature on the prevention and management of heat illnesses. A soldier’s duties often require engaging in vigorous activity while wearing heavy gear, often under conditions which make adequate nutritional and hydration status difficult to maintain. While training exercises are meant to simulate the stress of combat, not all soldiers are able to stand these conditions for prolonged periods of time.

Clinical Description
Heat illness occurs on a spectrum. The ICD 9 contains ten different diagnoses which categorize the physiologic manifestations of excess heat stress. The umbrella term for these illnesses is heat injury (evidence of both hyperthermia and end organ damage). We will discuss a few specific terms. The main distinction to note is the difference between intermediate and severe forms of heat illness. Intermediate illnesses have no neurologic impairment, while severe heat illness is a form of distributive shock in which patients have altered or depressed mentation as a consequence.

Heat rash, characterized by pruritic papules due to occluded sweat glands, and heat cramps, painful muscle spasms attributed to electrolyte abnormalities and dehydration, are both mild forms of heat illness. Heat exhaustion presents with malaise, nausea and vomiting, headaches, and is classified as an intermediate severity form of heat illness. A patient’s vital signs may demonstrate tachycardia and hypotension; however there is normal mentation and no central nervous system involvement. Heat syncope, also called exercise associated collapse, falls within the intermediate range of illness as well. Both syndromes are associated with slightly elevated core temperature.

Heat shock is a multisystem illness, and is the most severe form of heat illness. The hallmark features are core temperature of 104°F or greater, encephalopathy, and additional evidence of end organ damage. Complications of heat shock are numerous and usually attributable to ischemia and oxidative damage. A systemic inflammatory response occurs as in other forms of distributive shock. Clinical findings include tachycardia, tachypnea, and hypotension, skin is warm and may be hyperidrotic or dry, pupils are often dilated from activation of the sympathetic nervous system, patients may have altered sensorium or even be comatose, and seizures can occur. Heat shock can be a cause of non-cardiogenic pulmonary edema and crackles may be auscultated. From a cardiac standpoint, myocardial infarction is a potential concern, as are arrhythmias due to electrolyte derangements including massive potassium shifts. Other physiologic complications include rhabdomyolysis, acute kidney injury from either decreased renal perfusion in the setting of massive hemodynamic compromise and/or pigment nephropathy, gastrointestinal bleeding as a consequence of ischemic colitis, and ischemic hepatic injury. Interestingly heat shock can lead to sepsis; when the combination of high temperature and reduced intestinal blood flow injures the intestinal wall, translocation of lipopolysaccharide and other bacteria, toxins occurs through the portal vein and peritoneal space.

Example Cases
Case 1
A four-year-old child on vacation from Michigan spends the day on the beach. His parents remind him to drink water and he finishes one water bottle over the course of the day. As the family packs up to leave, the boy’s parents notice he is less interactive than usual. He sits still and appears flushed. Thirty minutes later he is unresponsive and an ambulance is called. His vital signs are as follows: blood pressure, 80/50; heart rate, 130; respiratory rate, 24; oxygen saturation, 98% on room air; and a rectal temperature is measured at 105°F. Before the initial assessment is complete, the patient suffers a five minute generalized tonic clonic seizure which spontaneously abates. His pupils are reactive but dilated. This patient has classic (non-exertional) heat stroke, in which the environment overwhelms the patient’s ability to dissipate heat.

Case 2
A thirty-year-old schoolteacher who lives in Kaneohe has recently decided to get in shape. At the urging of a few of her friends, she signs up for a fifteen mile trail run. She trains for only a week beforehand by running a few miles after work in the evenings. The race starts at 1000. It is a hot and humid day. Two miles from the finish line, she collapses. On assessment by the medical staff present, her rectal temperature is 104°F, she is profusely sweating and disoriented when questioned. This patient has exertional heat stroke, in which intrinsic heat production is the primary cause for hyperthermia.

Case 3
A 19-year-old military recruit is hospitalized after competing in the expert field medical badge. Following completion of the course, he collapses and is brought to the emergency room. He recovers consciousness and on initial assessment there is notable hypotension, temperature of 105°F, diffuse myalgias, elevated CK, acute renal failure with a Cr of 2.5, potassium of 6.0, electrocardiogram showed peaked T waves, and elevated liver associated enzymes. He is immediately given fluids for his rhabdomyolysis, calcium chloride, albuterol, and insulin with dextrose for his hyperkalemia. A few hours after presentation, he goes into hypoxic respiratory failure and is emergently intubated. The patient spends the next three days in the intensive
case for a patient with heat stroke involves three main concepts: stabilization of airway and circulation, rapid cooling, and finally transport to advanced care with monitoring for potential complications of heat illness. Sources agree that if the patient is otherwise stable, cooling should be addressed first and transport second. Morbidity and mortality are directly related to the duration of core temperature elevation, and therefore rapid cooling is strongly stressed.3

In an austere environment, materials to initiate cooling may not be readily available. All equipment and excess clothing should be immediately removed. The patient should be moved to a shaded area. Immersion in ice water is highly effective for rapid cooling. The water should be stirred frequently during cooling to ensure continual contact with cool fluid. If ice water is not available, any cool water source will suffice. A nearby stream or pool of water can be used if no other options exist. If ice is available but no tub, the patient can be placed in a tarp or sheet, covered with a large amount of ice, and then the tarp or sheet can be wrapped around them. Alternatively, if immersion in water is not possible, application of cool wet cloths can be used, with frequent re-application as soon as the material warms. As much body surface area as possible should be covered when using this method. Spraying cool water over the patient is also an alternative method if available. Fanning may be helpful, however never allow a conscious patient to fan themselves as this will only generate more heat via skeletal muscle contraction.3

Cooling via ice water immersion is estimated to occur at a rate of approximately 1°C for every five minutes (1°F every three minutes).3 If temperature monitoring is not possible, we suggest cooling the patient for fifteen to twenty minutes, or until the patient begins to shiver. This allows for a reduction to a safe temperature without risking over cooling. Other general interventions which should be performed in the field include intravenous access if possible followed by fluid administration, or oral rehydration if the patient is able to tolerate oral intake.

**Treatment in Non-Austere Environments**

Research has shown mortality correlates with the degree of temperature elevation, time to initiation of cooling, and number of organ systems involved. Management in the hospital setting should focus on modifying these factors. Continuous temperature monitoring should be initiated. External temperature is not reliable. Instead a bladder catheter with temperature probe, flexible rectal thermistor, or esophageal temperature monitoring should be used.10

A detailed history regarding precipitation of the event, medications, drugs of abuse, and medical history looking in particular for predisposing processes like sickle cell disease and heart disease, should be obtained.

If the cooling process initiated in the field has not lowered temperature to normal limits, it should be continued with ice water immersion or cutaneous application of ice packs in the hospital setting. Some studies show systemic cooling with a central venous catheter is also effective; however the potential complications from the procedure outweigh any benefit over using conventional methods of cooling. Multiple studies have shown pharmacologic treatment, including non-steroidal anti-inflammatories, acetaminophen, and dantrolene, have no outcome improvement, but may worsen complications. Therefore pharmacologic adjuncts to cooling are not generally recommended.10

Laboratory studies should be obtained including a complete blood count, electrolyte panel, creatine kinase level, urinalysis, lactate, blood gas, hepatic function tests, and coagulation studies. An electrocardiogram and chest X-ray should also be obtained. These studies are adequate to assess for the potential complications of heat stroke discussed above. During assessment, continuous vital signs monitoring and telemetry should be initiated.

Resuscitation with crystalloids should be aggressive and titrated to improvement in vital signs and laboratory parameters. Delirium and seizures are both treated with intravenous benzodiazepines. Electrolyte abnormalities should be corrected appropriately with removal or repletion. Many affected patients may need to be hospitalized for a period of observation and monitoring for complications. Hypotension after fluid resuscitation, seizure, encephalopathy that is not rapidly resolving, persistent oliguria, rhabdomyolysis, and evidence of gastrointestinal bleeding, are all examples. Patients with signs of multi organ dysfunction or evidence of disseminated intravascular coagulation are admitted to an intensive care setting.5

The importance of preventive measures should be stressed. Proper education regarding hydration, warning signs, and risk factors is crucial in preventing heat illness. Acclimatization by gradually increasing exertion in heat and maintenance of cardiovascular fitness are also important. The physiologic adjustments that occur are expansion of the plasma volume, earlier and increased sweating, lower salt concentration in sweat, and lower skin and core temperatures for a given amount of exercise. Wet bulb globe temperature (WBGT) should be used to predict environmental risk instead of ambient temperature alone. WBGT is a composite temperature used to estimate the effect of temperature, humidity, and wind speed on overall heat burden.7 Patients with risk factors for heat related illness should be aware of their predilection. A variety of medications increase risk, including diuretics, antihistamines, and sympathomimetics.1

Though an individual presenting with heat illness may appear to have a straightforward etiology of their illness, a differential diagnosis should still be considered. Clinical entities that could present with decreased consciousness, autonomic dysfunction, and hyperthermia include pontine or midbrain infarct, meningitis, drug induced toxidrome, and parasitic infections such as malaria.
Conclusion
Heat stroke continues to be a common and potentially lethal event. The method used for cooling is not crucial, instead the speed of cooling and transport to advanced medical care is more important prognostically. The recognized complications of heat stroke are numerous and in the worst case scenario it can result in multiorgan failure. Thorough laboratory assessment as well as electrocardiogram and chest X-ray should be performed upon arrival to a higher level of care to avoid missing complications. Medical care is largely aimed at supportive therapy with cooling and intravenous crystalloids, with the goal of maintaining normal vital signs and preventing end organ damage.

Disclosures
The author reported no conflicts of interest.

Disclaimer
The views expressed in this manuscript are those of the author and do not reflect the official policy or position of the Department of the Army, Department of Defense, or the US Government.

References
Foreign Body Synovitis in the Pacific

Caleb Anderson MD; Rodger Stitt MD; and Jefferson Roberts MD

Abstract

Foreign body synovitis in the Pacific region typically involves a penetrating injury to a joint. The introduced biomaterial produces an inflammatory reaction or inoculates the tissue with bacteria, creating an infection. Each year millions of people visit the Hawaiian Islands to hike, surf, snorkel, and participate in other outdoor activities, creating an array of interactions between people and nature. The two most commonly reported penetrating foreign body joint injuries are sea urchin synovitis and synovitis due to implantation of organic material such as wood splinters or plant thorns. In this article we describe the presentation, infectious profile, and treatment of these joint injuries.

Introduction

Foreign body synovitis is a form of arthropathy, primarily monoarticular arthritis, resulting from an inflammatory reaction of the synovium due to a foreign object. The foreign fragments are characterized as either inorganic or organic. In this article, we will focus on foreign body synovitis from organic fragments. Typical materials that may cause organic foreign body synovitis include rose bush thorns, wood splinters, starfish, coral, sea urchin spines, and shell fragments. A common scenario is a young person who suffers a penetrating injury while working, gardening, hiking in the wilderness, or swimming. The presence of foreign objects lodged in the joint can lead to an inflammatory reaction. Foreign objects can also seed the joint with bacterial or fungal elements producing a septic arthritis. It is important to rule out septic arthritis when evaluating possible foreign body synovitis. The synovitis may be acute (hours to days after the injury), or chronic (months to years after the injury). This paper will describe the presentation, progression, diagnosis, and treatment of the two most common causes of foreign body synovitis in the Pacific Region, namely sea urchin synovitis and synovitis secondary to implantation of organic plant material.

Example Case One: Retained Sea Urchin Spines

A 55-year-old woman vacationing in Hawai‘i was snorkeling when she was pushed against a rock by a large wave. Her hand landed on a sea urchin while bracing against the rock. She felt sudden pain and noted several purple puncture wounds on the palmar surface at the base of her right ring finger. Her wound was dressed at a local urgent care clinic but she noticed worsening stiffness and paresthesias in the ulnar distribution of her hand over the next few months. Ten weeks after the event an MRI demonstrated extensive flexor tenosynovitis as well as a possible foreign body. She was taken for surgical exploration which revealed multiple sea urchin spines embedded in the subcutaneous tissue, flexor digitorum superficialis tendon, and flexor tendon sheath. The fragments were removed and extensive flexor tenosynovectomy was performed.

Example Case Two: Sea Urchin Retained Foreign Body

A 33-year-old woman suffered a puncture from sea urchin spines in the interphalangeal joint of her left hallux while surfing in Sukuoka, Japan. The sea urchin spines were removed ten days later and she was started on broad spectrum antibiotics. Radiographs taken seven weeks post injury showed periarticular osteopenia, sub-articular cyst-like lucencies at the base of the distal phalanx, and subtle marginal erosion at the medial head of the proximal phalanx. An MRI done 8 weeks post injury showed a possible retained foreign body, although surgical exploration one week later yielded no foreign bodies and negative cultures. At four months following the injury the patient experienced worsening swelling and pain. Radiographs showed new extensive peri-articular erosion as well as worsening of the subchondral cyst-like pathology of the distal phalanx. The patient was restarted on antimicrobial therapy including mycobacterial and fungal coverage. The patient experienced worsening pain and range of motion and at nine months post injury was noted to have more extensive peri-articular erosions. At ten months post injury, a synovectomy, arthrodesis, and debridement of the interphalangeal joint were performed. No retained spine was recovered, and histopathology was notable for granulomatous synovitis with central fibrinoid necrosis as well as metaplastic bone formation.

Discussion

Foreign body synovitis secondary to implanted organic material is a common cause of monoarticular and tendon synovial tissue inflammation and should always be on the list of differential diagnoses in a patient presenting with joint swelling and erythema, even if a history of penetrating injury is not reported. If not removed and treated appropriately, acute synovitis, chronic monoarticular synovitis, chronic bursitis, chronic tenosynovitis, or a soft-tissue foreign body cyst may result. Common causes of foreign body synovitis in the wilderness environment include sea shell fragments, plant thorns, and sea urchin spines, with synovitis secondary to sea urchin spike implantation and implantation of organic plant material being the most commonly reported in literature.

Sea Urchin Synovitis

Sea urchin injuries are a very common cause of foreign body synovitis in the Asia Pacific region. Sea urchins, also known as *wana* in Hawaiian, are of the class Echinoidea of the echinoderm phylum, with some 940 species. *Wana* are covered in sharp spines attached via muscle and ball and socket joints. Hawai‘i has at least three known species of sharp spined sea
splinters are considered highly inflammatory as compared to imbedded in or near a joint space. Both plant thorns and wood splinters can include infection, inflammation, toxic reactions, and granuloma formation, which can ultimately lead to synovitis if imbedded in or near a joint space. Both plant thorns and wood splinters are considered highly inflammatory as compared to other inorganic foreign bodies such as metal and plastic, mostly due to inherent alkaloids (blackthorn) and oils and resins (many types of wood). Progression to synovitis with wood splinters and plant thorns is generally delayed, with one study finding a median interval of 68.5 days between injury and onset of joint pain/stiffness.

Most literature on plant thorn synovitis from the Pacific region involves the blackthorn shrub (Prunus spinous), date palm, or cactus. However, a case report of plant thorn synovitis due to Zanthoxylum ailanthoides, an aromatic plant used as spice and found throughout East Asia, was reported in 2007. This specific case was the first instance of plant thorn synovitis reported in Asia and was diagnosed by high-resolution ultrasound. Synovectomy was the definitive treatment.

Historically, plant thorn synovitis was considered an aseptic inflammatory reaction since most of the early literature failed to grow a causative organism on culture, although joint aspiration yielded a turbid fluid containing pus much like septic arthritis. In the case of published literature on the blackthorn shrub, or Prunus spinous, it was hypothesized that the joint inflammation was secondary to alkaloids present in the thorn, although it was noted that antibiotics provided temporary symptomatic relief. It was not until literature published in 1977 that positive cultures were noted when three out of five patients were found to have infectious aspirate growing Staphylococcus albus, gram negative rods, and alpha hemolytic strep. According to one literature review covering 1977 to 2008, of the 56 reported cases of plant thorn synovitis, 27% were found to have cultures positive for infectious organisms. Of these infectious cases, 73% were found to be due to Pantoea agglomerans (formerly referred to as Enterobacter agglomerans), a facultative anaerobic gram negative rod found commonly in soil and on vegetation. Other implicated agents include Staphylococcus aureus, Enterobacter sp, and Nocardia asteroides. It is unclear whether this represents a superinfection from a primarily sterile inflammatory process or whether these are the early culprits of the inflammatory reaction.

Patients with both septic and noninfectious plant thorn arthritis (ie, foreign body synovitis) often present afebrile with a warm, swollen, and tender joint weeks to months after the initial injury, and may not report a history of implantation. A delay of up to nine months in diagnosis has been reported. Joint aspiration generally yields a turbid fluid with high leukocyte count and polymorphonuclear leukocyte predominance. Both non-infectious plant thorn synovitis and septic arthritis can be quite dormant in presentation, especially considering that the majority of infections are due to Pantoea agglomerans, a quite indolent pathogen. This is why it is important to rule out septic arthritis before making the diagnosis of foreign body synovitis.

**Plant Thorn and Wood Splinter Synovitis**

Wood splinter and plant thorn implantation are very common in both children and adults, and while most superficial splinters can be extracted by the patients themselves, deeper splinters and those that break upon removal often require removal by a trained health care provider. Complications from retained splinters can include infection, inflammation, toxic reactions, and granuloma formation, which can ultimately lead to synovitis if imbedded in or near a joint space. Both plant thorns and wood splinters are considered highly inflammatory as compared to...
be made to remove the spine since the spine is very brittle and can fragment. It has also been suggested to strike the affected area with a stone in an attempt to crush the spine and make it more easily resorbed.15 Home remedies have included using vinegar and urine to attempt to dissolve the spine, although there is no good evidence to support these claims. Application of a salicylic-acid paste has also been used to attempt to dissolve the spines. Immersion of the affected area in hot water (110-115°F) has been found to provide pain relief initially, likely through inactivation of toxins, though this does nothing to prevent long-term complications.16 Most interventions in the austere environment are anecdotal and have little if any supporting evidence. Generally, all that can be done is to remove small superficial splinters in the field before infection or inflammation occurs, and to keep the injury site clean.17

**Treatment in Non-Austere Environment**

In a non-austere environment, most patients with foreign body synovitis present to the emergency department or their primary care physician with symptoms consistent with an acute, subacute, or chronic inflammatory monoarticular arthritis. The first step in a diagnostic evaluation is generally a plain radiograph, which may detect a foreign object depending on its density compared to soft tissue. Imaging with a plain radiograph generally can only detect radiodense material such as fish bone, sea urchin spines, or metal, and will usually miss wood or plant thorns, with one study noting only 5% of wood splinters being seen on radiography.18 High resolution ultrasound, CT, and MRI imaging are quite efficacious at detecting wood splinters and plant thorns. Even with advanced imaging, small foreign objects may remain unidentified.9

If a foreign body is found, the patient should be evaluated by an orthopedic surgeon for possible arthroscopic surgery with debridement and irrigation. This is the recommended treatment of choice since it is less invasive than synovectomy. Antibiotics may be indicated if there are signs of infection or if there is an isolated organism from synovial culture. Joint infection is much more likely with a retained wooden body than with a sea urchin spine. Reactive arthritis due to retained material should be considered in cases with continued swelling and pain without positive cultures following initial arthroscopic surgery. Arthroscopy is the preferred treatment for such refractory cases because it will provide better visualization and more definitive therapy.10 It has been suggested that sea urchin synovitis should be treated initially with synovectomy given the high rate of progression to arthritis and joint destruction. In one case series of twelve patients with chronic sea urchin arthritis of the hand, one patient required amputation of the affected finger and six patients required surgical debridement, with a trend towards better outcome with earlier synovectomy. Ensuring up to date tetanus vaccination status is very important in all cases of foreign body synovitis or penetrating trauma.

**Conclusion**

Foreign body synovitis is common in the Pacific region and can range from a chronic inflammatory state to a serious secondary acute bacterial infection. It is important for the patient and physician to suspect joint involvement after a penetrating injury near the intra-articular joint space even if the injury appears superficial. It is also important for the physician to realize that each individual cause of foreign body synovitis has a different presentation, progression of disease, microbial profile, and risk of infection, and thus, different treatment. Patients presenting with a vegetative foreign body have a much greater risk of secondary infection due to the organic nature and porous structure of the body as compared to sea urchin spine, which is very seldom found to be infectious.19

A radiograph or other advanced imaging of the joint may aid in detection of a foreign body, although it is often negative and the disease process can progress even without concomitant radiographic evidence of retained foreign body. If an inflammatory process is suspected, the joint should be aspirated and sent for cell count and culture. If clinical suspicion is high enough, arthroscopic exploration and debridement should be considered. Untreated foreign body synovitis can cause serious destruction of the joint and surrounding tissue if not treated early and appropriately.

**Disclosures**

The authors reported no conflicts of interest.

**Disclaimer**

The views expressed in this manuscript are those of the authors and do not reflect the official policy or position of the Department of the Army, Department of Defense, or the US Government.

Authors’ Affiliation:
Department of Medicine, Tripler Army Medical Center, Honolulu, HI 96859

Correspondence to:
Caleb Anderson MD; 1 Jarrett White Rd, Honolulu, HI 96859; Ph: (808) 433-4266; Email: caleb.w.anderson8.mil@mail.mil

**References**

Centipede Envenomation: Bringing the Pain to Hawai‘i and Pacific Islands

Joshua L. Fenderson MD

Abstract
Scolopendra subspinipes is the only clinically significant centipede found in Hawai‘i. Envenomation typically leads to extreme localized pain, erythema, induration, and tissue necrosis and possible lymphedema or lymphangitis. Mortality is uncommon and results from secondary infection or anaphylaxis. Management is supportive and includes wound care, pain control, and treatment with topical or oral antihistamines and anti-inflammatory medications.

Introduction
Centipede envenomations are frequent occurrences among hikers, campers and other visitors to Hawai‘i. It is not uncommon to find recipients of these bites in clinics or emergency rooms throughout the state. Bites from centipedes can cause extreme pain, edema, erythema and other localized symptoms as well as anxiety and panic. Rarely, anaphylaxis and death have occurred after a centipede bite. In a remote setting where resources are limited, importance should be placed on identification of the source of the bite and recognition of common symptoms of centipede bites to distinguish them from more serious and potentially fatal arthropod bites.

Relevance To Hawai‘i And Asian Pacific
Hawai‘i is home to three species of centipede Lithobius sp., Mecistocephalus maxillaries, and Scolopendra subspinipes. S. subspinipes is the only Hawaiian centipede with clinical significance and has a number of aliases, including Giant Centipede, Jungle Centipede, Vietnamese Centipede, and Chinese Red Head. As is implied by these aliases, these centipedes are large and have a broad habitat that includes all of the Pacific islands.

Many residents and tourists engage in camping, hiking, and other outdoor activities that bring human and centipede into close proximity, setting the stage for bite occurrences. The actual incidence is difficult to determine since not all bite victims seek care, but a significant number of cases do get evaluated in emergency rooms. The Hawai‘i Department of Health’s most recent injury prevention plan reports that, of the unintentional injuries presenting to emergency departments with a “natural or environmental” cause from 2007-2011, 11% were due to bites by centipedes. For perspective, 36% were due to canine bites and 11% were due to stings from bees and wasps.

Example Cases
Many human encounters with S. subspinipes and other centipedes end in a startle at the expense of either or both parties. Some may be unfortunate enough to experience the grasp of a centipede’s forcipules and the nagging pain and inflammation induced by envenomation. Very few, though, experience symptoms of such severity that their lives are at risk.

A 22-year-old gardener, with no risk factors for coronary artery disease, developed ST elevation myocardial infarction after being bitten on his finger while working. His initial presentation was typical. He had severe pain and edema which subsided over the next hour with application of cold and pressure to the area. Approximately two hours after the bite, he began to have intense, retrosternal chest pain with nausea, diaphoresis, and radiation down his left arm. He presented to the emergency department at fourteen hours after the bite with persistent chest pain, but vital signs and examination were unremarkable. His electrocardiograph showed ST segment elevations in the anterior leads and his cardiac enzymes were significantly elevated. Echocardiography showed a hypokinetic anterior wall and a left ventricular ejection fraction of 35%; coronary angiography was unremarkable. Three days after the bite, his chest pain had resolved and his repeat electrocardiograph and echocardiography were without abnormality.

A 44-year-old woman developed rhabdomyolysis and acute kidney injury after a centipede bite to her right foot in her eastern Arizona home. Her immediate symptoms of localized pain, swelling, numbness and discoloration spread to involve her entire right leg over the following three days which drove her to seek medical attention. She was referred to the University of Arizona Health Sciences Center at five days after the bite due to worsening of her condition despite therapy.

She was found to be afebrile, but hypertensive and tachycardic with a marked non-erythematous swelling, warmth, and tenderness of her entire right lower extremity. She also had impaired motor function and abnormal sensation of her ankle and toes. She had 2+ blood on urine dipstick with two erythrocytes per high power field on urine microscopy. Her serum creatinine was elevated to 11.4 mg/dl and hemodialysis was initiated within 24 hours of admission. She underwent three sessions of hemodialysis with her renal function returning to near her baseline; however, she was found to have neurovascular compromise in her right lower extremity due to compartment syndrome. Her motor function and sensation improved with fasciotomy, but her neurologic function was not fully recovered at the time the report was written.

The number of fatalities documented after centipede envenomation are few, and there is just one report that is well authenticated. A publication from 1932 reports the case of a seven year old female in the Phillipines who became severely ill and died shortly after she was bitten on the head by a S. subspinipes centipede. Other fatalities have been reported but not substantiated.
Discussion

S. subspinipes is among the largest species of centipede measuring up to 20 cm in length. These arthropods have a brown or redish head and dark green body with 21 total segments. From each segment extends one pair of yellow or yellow-orange legs. The legs attached to the head are modified into sharp claws, called forcipules, which are hollow and connected to venom glands; and the legs attached to the final segment are long and prominent, extending behind the body. These centipede are easily distinguished from other centipedes in Hawai‘i by their significant size since both Lithobius sp. and M. maxillaries reach just 5 cm in length.

S. subspinipes are fast and aggressive centipedes which strike instinctively with little provocation. S. subspinipes is nocturnally active, generally preying on other insects and arthropods; however, these centipedes will feed on anything they can overpower to include mice and small reptiles. During the day, they hide in moist and darkened areas, under logs, leaves, and rocks. Shoes, clothing, bedding and sleeping bags are adequate substitutes and common meeting places for human stings.2 S. subspinipes grasp victims with their forcipules and inject a venom comprised of a complex mix of chemicals, including phospholipase A2, serotonin, and acidic proteins, from their venom glands via hollow openings in the forcipules.3,4

In humans, this leads to immediate and often excruciating burning pain followed by local edema and erythema. Lymphadenopathy and lymphangitis are also common. Tissue necrosis may be seen at the forcipule puncture sites.3,4 The degree of symptoms varies from person to person and bite to bite. Pain and edema generally resolve spontaneously over a few days to one week, but can persist for up to three weeks.4 Cellulitis and secondary infection occur, but are uncommon complications of S. subspinipes bites. Systemic reactions and death from centipede envenomation rarely occur, however, acute myocardial ischemia in an adult male as well as death in a 7-year-old girl after a bite to the head have been reported.3,9 Urticaria, anaphylaxis and rhabdomyolysis are also very rare complications which have been attributed to envenomation by S. subspinipes.3,4

Treatment In Resource Constrained Environment

Data on treatment is limited and there is no standard or protocol that exists for management of these wounds. Treatment for centipede bites is primarily supportive.3,4 In a resource constrained environment, it is helpful if the centipede is positively identified because it can be difficult to distinguish a centipede bite from bites of other poisonous arthropods which require more resource intensive management. Anesthetic, anti-inflammatory and antihistamine medications are suggested for control of pain and inflammation.4 Hot water or other mode of heat application soon after the bite occurs is thought to denature proteins in venom, reducing degree and duration of symptoms.3,4,8 After the initial heat application, icing the bite area assists with reduction in swelling and inflammation.2 For subjects with more severe symptoms, a short course of corticosteroids may decrease pain intensity and edema. The wound should be kept clean and should be covered to prevent contamination or secondary infection, and a tetanus immunization should be administered.4

Treatment In Non-Austere Environments

Many victims of centipede envenomation do not seek medical attention and most symptoms will resolve spontaneously. Treatment of uncomplicated wounds does not greatly differ when limited resources are not a concern. Management should be supportive with wound care and control of pain and inflammation being the mainstay of treatment. Initial heating and later ice application is again suggested.3,4,9 Prophylactic antibiotics are generally unnecessary; however, if evidence of secondary infection is present, the wound should be cultured and a course of antibiotics which cover gram positive organisms should be initiated. Benzodiazepines may be helpful in the centipede victim with symptoms of anxiety.3 Cardiac ischemia and anaphylaxis are very rare complications and managing these conditions with standard protocols takes priority over treating localized symptoms. All patients presenting with centipede bites should be monitored in the emergency room for at least 4 hours for evidence of toxic shock.2 In addition, bite victims should receive an immunization for tetanus.3,4,9

Conclusion

Scolopendra subspinipes is the vector of all medically significant centipede envenomations in Hawai‘i. Though their bites can be very painful, serious morbidity is very uncommon. Treatment is primarily supportive in both the resource constrained and hospital settings, and therapy is directed at reducing pain, swelling, and anxiety. There is anecdotal evidence supporting the use of heat soon after the bite has occurred with the goal of denaturing the venomous proteins. Anaphalaxis, cardiac ischemia, or other serious complications are rare and should be managed according to existing treatment protocols. Victims of centipede bites should be monitored for four hours for development of symptoms of these complications, and all should receive a tetanus vaccination.

Disclosure

The author reported no conflicts of interest.

Disclaimer

The views expressed in this manuscript are those of the author and do not reflect the official policy or position of the Department of the Army, Department of Defense, or the US Government.

Acknowledgements

The author thanks the library staff at Tripler Army Medical Center for assisting with the literature review and the reviewers of this paper for their insightful comments and suggestions.

Author’s Affiliation:
Department of Medicine, Tripler Army Medical Center, Honolulu, HI 96859

Correspondence to:
Joshua L. Fenderson MD; Tripler Army Medical Center, Honolulu, HI 96859; Email: joshua.l.fenderson.mil@mail.mil
References


Ua mau ke ea o ka ‘aina i ka pono.
The life of the land is preserved in righteousness.
Tropic Lightning: Myth or Menace?

John McCarthy MD

Abstract

Lightning is one of the leading causes of death related to environmental disaster. Of all lightning fatalities documented between 2006 and 2012, leisure activities contributed the largest proportion of deaths, with water-associated, sports, and camping being the most common. Despite the prevalence of these activities throughout the islands, Hawai‘i has had zero documented lightning fatalities since weather data tracking was initiated in 1959. There is a common misconception that lightning does not strike the ground in Hawai‘i. This myth may contribute to a potentially dangerous false sense of security, and recognition of warning signs and risk factor modification remain the most important prevention strategies. Lightning damage occurs on a spectrum, from minor burns to multi-organ dysfunction. After injury, initial treatment should focus on "reverse triage" and immediate cardiopulmonary resuscitation when indicated, followed by transfer to a healthcare facility. Definitive treatment entails monitoring and management of potential sequelae, to include cardiovascular, neurologic, dermatologic, ophthalmologic, audiovestibular, and psychiatric complications.

Background

Lightning physics is complex, but all lightning begins with a thunderstorm. A thunderstorm develops when the sun heats pockets of air that cause those pockets to rise in columns which eventually form clouds. Within these clouds, layers of precipitation develop (ice, hail, rain), which based on their weights, stratify to different layers in the cloud. Rising warm air with falling ice particles results in collisions between the different particles. This creates charge, and eventually the top of the cloud becomes predominantly positive and the bottom of the cloud negative. The earth is generally negatively charged, but when storm clouds roll over, the negatively charged bottom of the cloud induces the earth to take on a positive charge. When the separation of charge becomes too much (a difference in voltage between the cloud and the ground or object exceeds 2 million volts per minute), the result is lightning.

Lightning strikes the earth approximately 8 million times a day or 100 times per second, making it one of the leading causes of weather related death in the world. In the United States, according to the National Oceanic and Atmospheric Administration, there were 4002 fatalities from 1959 to 2012. Climate, region, and season play a role in risk of lightning injury, as shown by the differences in incidence of lightning injury across the United States. Florida and Texas account for the most lightning fatalities (415 and 215 respectively), while Alaska and Hawai‘i (tied for 0) account for the least. Compared to last century, despite increasing population density, there are far fewer annual lightning fatalities. In the 1920s-1940s, the United States averaged around 400 lightning deaths per year. In the last 50 years there was an average of 40 deaths annually, and in the last three years, there was an average of 25 deaths annually. It is unclear why this decline is occurring, but some proposed explanations include increased urbanization, modernization of rural infrastructure and farm equipment, and increased awareness and education regarding lightning safety.

The most common lightning victim appears to be a male who is engaging in a leisure activity, particularly a water-related activity such as fishing or boating. Based on data gathered from 238 lightning fatalities occurring from 2006 to 2012, males are more likely to be victims than females (82% vs 18%); leisure associated activities are the most common (64%); water-related (36%), sports (19%), and camping (10%) made up the top three most common activities; and of water-related lightning deaths, the distribution included fishing (46%), boating (25%), beach (20%), and swimming (9%).

Mechanisms of lightning injury take five different forms. A direct strike generally occurs when a person is in the open, and is the most likely to result in instantaneous death. Splash injury occurs when lightning strikes a nearby object and current jumps or "splashes" via the path of least resistance (eg, nearby victim). Contact injury occurs when a victim is touching an object that is hit by lightning. Ground current occurs when lightning strikes the ground near a victim and passes from the strike point into the victim. A fifth mechanism involves passage of lightning from the victim upward. There are also instances of blunt injury due to the sheer concussive force of the associated thunder.

Lightning is unidirectional and produces a massive current impulse of very short duration. The impulse can result in vaporization of skin moisture and blasting apart of clothes, rendering the victim naked. Unlike electrical injuries sustained from alternating current, the current of a lightning strike only remains internally for a very short time period, making deep tissue burns and myoglobinuric renal failure uncommon. Rather, that brief intense current may result in cardiac arrest (asystole more than ventricular fibrillation); damage to the central, peripheral, and/or autonomic nervous systems; superficial burns, ocular injuries (cataracts, corneal lesions, retinal detachment); ear injuries (tympanic membrane rupture, ossicle disruption, tinnitus); and psychological injury (fatigue, sleep disturbance, depression, memory problems).

Relevance to Hawai‘i and Asia Pacific

It is a common misconception that lightning does not strike the ground (or at all) in Hawai‘i. While it is true that lightning in Hawai‘i is far less common than on the mainland (the exact quantification remains elusive because the National Lightning Detection Network does not include Hawai‘i), there are many examples of significant lightning events on the islands. In May 2011, O‘ahu experienced a particularly impressive storm with
an estimated forty thousand cloud to ground lightning strikes over a 30 hour period. In March 2012, lightning struck the hull of a 36 foot catamaran in O‘ahu, and the boat sank to the bottom of Keehi Lagoon Boat Harbor. A storm in Dec 2013 on the Big Island of Hawai‘i yielded an estimated ten to twenty thousand lightning strikes. No injuries were documented during any of these incidents.

Lightning in the Hawaiian Islands is referenced in Hawaiian mythology. Pele is not only the goddess of fire, wind, and volcanoes, but also lightning. In one story, Pele disguises herself and enters a store. She approaches a Chinese proprietor and asks for a cup of coffee. When the proprietor refuses, Pele unleashes lightning, causing the store to burn down and killing the proprietor inside.

Aside from stories of Pele’s vengeance, Hawai‘i has had no official documented lightning fatalities since storm data tracking was initiated in 1959. Hawai‘i is ranked last (tied with Alaska) in the least lightning fatalities per year (zero), and an estimated 0.1 injuries from lightning per 1 million people per year.

The scarcity of lightning injuries may engender a false sense of security. Hawai‘i is home to recreational activities (e.g., water sports, camping, hiking, sporting events) at the highest risk for lightning injury. Therefore, individuals and providers should remain knowledgeable regarding recognition, prevention, and treatment strategies.

**Example Case(s)**

In July 2013, a 47-year-old man was inside his home in Maui during Hurricane Flossie. He was washing dishes, and when he reached to turn on the faucet, he saw a blue streak connecting the faucet to his hand. The shock sent him to his knees, but shortly after, he was able to walk to the living room to tell his wife what had happened. He reported hand numbness for five minutes. His wife took his pulse, estimated at 180, and subsequently called 911.

**Treatment in Resource-Constrained Environments**

**Recognition:** The “30-30 Rule” is still often utilized, but may not be the current best practice. This rule suggests that if less than thirty seconds passes between seeing lightning and hearing corresponding thunder, then an individual should seek a safer location. Although easy to remember, practice guidelines now express concern that this rule may instill a false sense of security. A safer approach suggests that if lightning is seen or thunder is heard, an individual is already at greater risk for lightning injury and precautions should be initiated regardless of time separation. Lightning is always associated with a thundercloud, but has been known to strike as much as 10 miles from the periphery of a storm. Therefore, lightning injury can still be sustained after a storm has passed or even in sunny conditions, and individuals should wait at least 30 minutes from the most recent thunder clap or lightning sighting before resuming activities. Signs of a more imminent strike include static electricity, nearby crackling, funny smells, or even “St. Elmo’s fire” (i.e., a blue haze around a person or object).

**Prevention:** No location is absolutely safe from lightning. Large structures and fully enclosed metal vehicles offer more protection than small open structures. Even while inside a structure, injury can occur with contact to conducting materials, as in the case example above (the victim touched his faucet). Individuals should avoid touching metal during thunderstorms, to include plumbing, telephones, and electronics. Areas to avoid would include open fields, tall structures (e.g., flagpoles, trees), or being near or in water (e.g., oceans, beaches, pools). Efforts should focus on fleeing these areas, however if this is not possible and signs of imminent strike are present (as explained above), individuals should take the lightning position as last resort.

**Treatment:** Most victims survive lightning injury if they receive timely medical treatment. The most likely cause of death from a lightning strike is cardiac arrest, and therefore if the victim is not breathing or without a pulse, advanced cardiac life support (ACLS) should be initiated as soon as possible with the following considerations: (1) patients struck by lightning do not carry electrical charge, and it is therefore safe to touch them, (2) caution must be exercised when running to save a lightning victim, as the rescuer is often putting themselves at risk (there is no explicit evidence that suggests lightning does not hit the same place twice, and in fact, it often does), (3) ventilation must often be supported, as respiratory arrest due to suppression of the medullary respiratory center as well as thoracic muscle spasm may persist despite return of spontaneous circulation, and (4) even if appearing dead (fixed and dilated pupils), or the interval between injury and resuscitation has been prolonged, lightning patients have been known to respond to resuscitation. For this reason, if multiple victims are present, “reverse triage” should be initiated, where those patients who are not breathing or are without a pulse should actually be treated first.

**Treatment in Non-Austere Environments**

In absence of cardiopulmonary arrest from the initial lightning strike, dying from lightning is a rare occurrence. Unfortunately, of the 70%-90% of people who survive lightning injury, many may still have permanent disability afterward. Getting struck by lightning can cause derangement in multiple organ systems, particularly related to cardiovascular, neurologic, dermatologic, eye, ear, and psychiatric manifestations. Recognition of high risk is therefore important when determining need for closer monitoring. These factors include suspected direct strike, any loss of consciousness, focal neurologic complaints, chest pain, dyspnea, major trauma, cranial burns, leg burns or burns greater than 10% of total surface body area, or pregnancy.

Lightning causes massive simultaneous depolarization of the myocardium as well as possible muscle paralysis of chest wall muscles and suppression of medullary respiratory center.
Additional cardiopulmonary manifestations from lightning strikes may include atrial fibrillation, elevated cardiac enzymes, cardiomyopathy, and ECG changes (ST elevation and QT prolongation). As discussed above, this emphasizes the importance of initial implementation of ACLS. Once resuscitation is achieved, high-risk patients should be admitted to the hospital for cardiac monitoring (at least 24 hours), screening ECG, and echocardiography.\(^1,5\)

Neurologic injury due to lightning strike varies widely in severity. Most sustained neurologic injuries are transient, and include loss of consciousness, headaches, paresthesias, confusion, seizures, and memory loss. Another phenomenon of neurologic lightning injury is known as keraunoparalysis which is a transient paralysis of limbs thought secondary to massive overstimulation of the autonomic nervous system. Subsequent vascular spasms may be accompanied by pulselessness, cyanosis, and motor/sensory deficits. Close monitoring for compartment syndrome should occur, but most instances of keraunoparalysis resolve spontaneously after several hours. Direct lightning strikes are associated with intracranial hemorrhage, cerebral infarctions, and hypoxic encephalopathy (often secondary to cardiopulmonary arrest). Delayed neurologic symptoms following lightning strike (eg, progressive myelopathy) are also possible, which would warrant close follow up with a neurologist to establish a long term treatment plan.\(^1,3,5\)

Whereas the American Burn Association recommends referral to a burn center for affected patients, generally, burns sustained after lightning injury are more superficial, less severe, and quickly healing when compared to high-voltage electrical burns. In order to avoid contact burns after initial strike, metal objects (eg, jewelry) should be promptly removed. Internal burns resulting in underlying muscle damage, compartment syndrome, and rhabdomyolysis are less common, however, suspicion should remain, particularly in a hypotensive patient. Hypotensive patients require fluid resuscitation as well as thorough evaluation for potential internal trauma caused by the concussive force of the lightning injury. Full thickness burns are generally seen in areas of skin that had contact to metal objects or synthetic melted materials. Full thickness burns requiring grafting are generally uncommon in lightning injury, and patients with superficial burns that involve less than 20% of total body surface area have been shown to heal relatively quickly.\(^1,3,5\)

Otologic and ophthalmologic findings after lightning injury are also well documented. Blast overpressures from lightning may result in tympanic membrane rupture and temporary deafness. Small perforations can be managed conservatively with bed rest and head elevation to prevent perilymph leakage. More severe injuries such as sensorineural deafness, vestibular injury, or complex perforations will likely require specialized care. The most common eye injury from lightning is cataracts which may occur soon after injury, or sometimes as late as four years after initial insult. Prompt referral to an ophthalmologist is always indicated in survivors of lighting injury, both for potential intervention (eg, steroids), as well as for closer evaluation of other potential injuries (eg, corneal burns, intraocular hemorrhage, uveitis, orbital fractures).\(^1,3,5\)

Psychological and neurocognitive sequelae of lightning injury are often delayed. Depression, changes in behavior, impaired memory, and difficulty with assimilation of new information have all been described in post-lightning strike syndrome. The highest recovery rates are seen within the first year after injury, with mild improvement after three years. Antidepressants have been found to be useful, but any dysfunction after three years is thought to be chronic, requiring long-term management.\(^1\)

**Conclusion**

In answering the question, “Tropic lightning: myth or menace?” the answer is neither. Lightning in Hawai‘i, while less common compared to the mainland, does occur and has the potential to be just as dangerous. The lack of documented lightning fatalities in Hawai‘i should not be construed to mean that lightning does not strike the ground, and the population should be educated regarding lightning safety.

**Disclosure**

The author reported no conflicts of interest.

**Disclaimer**

The views expressed in this abstract/manuscript are those of the author and do not reflect the official policy or position of the Department of the Army, Department of Defense, or the US Government.

Author's Affiliation: Department of Medicine, Tripler Army Medical Center, Honolulu, HI 96859

Correspondence to: John G. McCarthy MD; Tripler Army Medical Center, Honolulu, HI 96859; Email: johnnymccarthy@gmail.com

**References**


