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CURRICULUM

Emergency Medicine Curriculum Utilizing the Flipped Classroom Method: Environmental Emergencies

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ABSTRACT:

Audience: This curriculum created and implemented at The Ohio State University Emergency Medicine Residency was designed to educate our emergency medicine (EM) residents, PGY-1 to PGY-3, as well as medical students and attending physicians.

Introduction: Environmental complaints are commonly seen in the emergency department (ED). In 2015, injuries from natural and environmental factors represented about 2 million ED visits in the United States, representing 4.9% of all ED visits.[1] Environmental illness includes injuries from cold, heat, barotrauma, altitude, electricity, radiation, submersion, and animal bites, stings, and envenomation. Emergency physicians must be proficient in the differential diagnosis and management of the wide variety of environmental emergencies.

The flipped classroom curricular model emphasizes self-directed learning activities completed by learners, followed by small group discussions pertaining to the topic reviewed. The active learning fostered by this curriculum increases faculty and learner engagement and interaction time typically absent in traditional lecture-based formats.[2-6] Studies have revealed that the application of knowledge through case studies, personal interaction with content experts, and integrated questions are effective learning strategies for emergency medicine residents.[6-8] The Ohio State University Emergency Medicine Residency didactic curriculum recently transitioned to a "flipped classroom" approach.[9-12] We created this innovative curriculum aimed to improve our residency education program and to share educational resources with other EM residency programs. Our curriculum utilizes an 18-month curricular cycle to cover the defined emergency medicine content based on the American Board of Emergency Medicine (ABEM) model curriculum. The flipped classroom curriculum maximizes didactic time and resident engagement, fosters intellectual curiosity and active learning, and meets the needs of today's learners. [6,9,13]

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Objectives: Through a flipped classroom design, we aim to teach the presentation and management of environmental emergencies, specifically cold related illness, heat related illness, undersea medicine, high altitude medicine, submersion, electrocution, radiation injury, and envenomation. This unique, innovative curriculum utilizes resources chosen by education faculty and resident learners, study questions, real-life experiences, and small group discussions in place of traditional lectures. In doing so, a goal of the curriculum is to encourage self-directed learning, improve understanding and knowledge retention, and improve the educational experience of our residents.

Methods: The educational strategies used in this curriculum include small group modules authored by education faculty and content experts based on the core emergency medicine content. Faculty were trained to facilitate sessions using the Socratic Method question and answer format, with a focus on fostering an open learning environment. Small groups also focus on the synthesis and application of knowledge through the discussion of real-life experiences. Resources were provided for residents as pre-reading prior to in-person, case-based discussion. Students were also encouraged to find their own references including use of free open access medical education (FOAM) resources.

Topics: Emergency medicine, flipped classroom, medical education, environmental emergencies, pedagogy, teaching.

USER GUIDE

List of Resources:

Learner Audience:
Medical students, interns, junior residents, senior residents

Length of Curriculum:
The entire didactic curriculum was developed to utilize an 18-month curricular cycle; therefore, resident learners experience each curricular topic twice in the course of their residency training. The genitourinary emergencies module consists of seven 45-60-minute small group sessions.

Topics:
Emergency medicine, flipped classroom, medical education, environmental emergencies, pedagogy, teaching.

Objectives:
Each chapter within our curriculum has individual objectives; however, educational objectives for the curriculum and more specifically, the environmental emergencies module include:

1. Cold and Injury Illness
    a. List and define stages of hypothermia.
    b. Review indications and methods of re-warming for hypothermic patients.
    c. Discuss pathophysiology of core temperature afterdrop.
    d. Review management of a hypothermic patient in cardiac arrest.
    e. Discuss differences between cold injuries of the soft tissue (trench foot, chilblains, frostbite) and their management.
2. Heat-Related Illness
    a. Discuss the body's methods of heat loss that allows temperature regulation.
    b. Differentiate between patients presenting with heat exhaustion versus heat stroke.
    c. Describe the risk factors for classic heat stroke.
    d. Describe the various methods of cooling for the hyperthermic patient.
3. Barotrauma, Hyperbaric Oxygen, and Altitude.
    a. Compare/contrast the different types of diving-related illness including: decompression sickness, barotrauma, and gas toxicity.
    b. Describe the treatment options for diving-related illness.
    c. Discuss the spectrum of altitude illness and list the preventative and acute treatment approaches.
4. Electrical/Radiation/Submersion Injuries
    a. Review the key pathophysiology related to drowning and the major organ systems involved.
    b. Discuss management of drowning.
    c. List risk factors for drowning
    d. Discuss the characteristics of different electrical injuries: high voltage versus low voltage.
    e. Explore the initial management of electrical injuries and how triage of a mass casualty lightning strike differs from standard mass casualty triage.
    f. Define the difference between radiation exposure and radiologic contamination.
    g. Discuss the concept of time/distance/shielding related to radiation exposure.
    h. List the expected manifestations of acute radiation sickness based on the dose absorbed.
5. Bites, Stings and Envenomation
    a. Identify and characterize common bite/envenomation patterns of poisonous fauna found in the United States (US).
    b. Describe the management and treatment for common reptile and arthropod envenomation.
    c. Understand and perform initial management of these injuries including venom specific antidotes.
    d. Describe the presenting symptoms of rabies.
    e. Compare the difference in the natural reservoirs for rabies in the US and internationally.
    f. Review the post-exposure prophylaxis for rabies exposure.

USER GUIDE

Brief introduction:

In 2015, injuries from natural and environmental factors represented about two million ED visits in the United States, representing 4.9% of all ED visits.[1] While this topic represents a relatively minor percent of questions on the ABEM qualifying exam,[13] this content is not well covered in undergraduate medical education or other specialties post-graduate education. Thus, many emergency medicine physicians are the de facto experts for these somewhat infrequent presentations. Also, there are many recognized environmental medicine-related fellowships that emergency medicine residency graduates can pursue. Because of these, when we developed our 18-month flipped classroom residency curriculum, we decided to include a block on environmental emergencies.

The flipped classroom learning approach is becoming more commonly recognized as a preferred curricular model for mature learners, specifically those in medical education. This particular model is a natural fit for the hands-on, experiential emergency medicine learner.[7] The active learning fostered by this curriculum increases faculty and learner engagement and interaction time, which is typically absent in traditional lecture-based formats.[8,12] Education literature shows that resident learners prefer learning activities that involve small group discussion, are case/skill based, and emphasize the application of newly obtained knowledge.[3,4] This educational model also provides a clear channel for the incorporation of evidence-based medicine and increases opportunities for educator-learner conversations. A successful flipped classroom curriculum fosters learner accountability and provides robust opportunities for formal assessment in various emergency medicine milestones.[7,11,13] For these reasons, we developed a flipped classroom curriculum at The Ohio State University Emergency Medicine Residency. This environmental emergencies curriculum is one of several topics in our overall 18-month didactic curriculum.

Problem identification, general and targeted needs assessment:

Traditional lecture-based didactics may not be the most effective or preferred method for emergency medicine resident education.[9] Previously, we used a traditional lecture format in our residency curriculum despite overwhelming evidence favoring a more hands-on, "flipped classroom" approach.[11,12] From the perspective of resident learners, the chance to remain fully engaged through the asking of questions developed from personal experiences, in addition to learning from the experiences of others, provides a technique of learning that makes a topic more difficult to forget.[8]

The ABEM model curriculum lists environmental disorders as an important core content area for training emergency medicine residents.[14] We used this model curriculum to guide creation of the block content. Environmental emergencies are not emphasized in undergraduate medical education, and many times emergency medicine providers are the most likely to encounter environmental-related disorders. Also, wilderness medicine, hyperbaric and undersea medicine are recognized fellowships that emergency medicine residency graduates can pursue.

As current literature reveals, both educators and learners benefit from an interactive and collaborative classroom, leading to the creation and implementation of this proposed curricular model at our emergency medicine residency program. Learners divide into small groups of at most 20 participants, but the curriculum could be effectively run with much smaller groups. A faculty facilitator leads the discussion through the cases and question prompts. Since implementation, residents and educators are engaging in new, valuable flipped classroom learning communities at The Ohio State University Emergency Medicine Residency. Through the curriculum, we continually seek to foster self-directed learning and increased collaboration between resident learners and education faculty members. This ensures that resident time will be maximized and learning will be more efficient and effective, therefore providing a potential positive impact on patient care and physician wellness. Currently, minimal flipped classroom curricular materials dedicated to the core content of emergency medicine exist.

Goals of the curriculum:

We aim to teach the presentation and management of environmental emergencies in an emergency department through the creation of a flipped classroom design. The topics specifically include cold-related illness, heat-related illness, undersea medicine, high altitude medicine, submersion, electrocution, radiation injury, and envenomation. Resident learners will learn the core content of emergency medicine in an 18-month curriculum utilizing self-directed learning and small group discussions based on the flipped classroom model.

This unique, innovative curriculum utilizes resources chosen by education faculty and resident learners, study questions, real-life experiences, and small group discussions in place of traditional lectures. In doing so, a goal of the curriculum is to encourage self-directed learning, improve understanding and knowledge retention, and improve the educational experience of our residents. Of note, this curriculum does not include specific pre-hospital management of environmental emergencies.

Objectives of the curriculum:

Each chapter within our curriculum has individual objectives; however, educational objectives for the curriculum and more specifically, the environmental emergencies module include:

1. Cold and Injury Illness
    a. List and define stages of hypothermia.
    b. Review indications and methods of re-warming for hypothermic patients.
    c. Discuss pathophysiology of core temperature afterdrop.
    d. Review management of a hypothermic patient in cardiac arrest.
    e. Discuss differences between cold injuries of the soft tissue (trench foot, chilblains, frostbite) and their management.
2. Heat-Related Illness
    a. Discuss the body's methods of heat loss that allows temperature regulation.
    b. Differentiate between patients presenting with heat exhaustion versus heat stroke.
    c. Describe the risk factors for classic heat stroke.
    d. Describe the various methods of cooling for the hyperthermic patient.
3. Barotrauma, Hyperbaric Oxygen, and Altitude.
    a. Compare/contrast the different types of diving-related illness including: decompression sickness, barotrauma, and gas toxicity.
    b. Describe the treatment options for diving-related illness.
    c. Discuss the spectrum of altitude illness and list the preventative and acute treatment approaches.
4. Electrical/Radiation/Submersion Injuries
    a. Review the key pathophysiology related to drowning and the major organ systems involved.
    b. Discuss management of drowning.
    c. List risk factors for drowning
    d. Discuss the characteristics of different electrical injuries: high voltage versus low voltage.
    e. Explore the initial management of electrical injuries and how triage of a mass casualty lightning strike differs from standard mass casualty triage.
    f. Define the difference between radiation exposure and radiologic contamination.
    g. Discuss the concept of time/distance/shielding related to radiation exposure.
    h. List the expected manifestations of acute radiation sickness based on the dose absorbed.
5. Bites, Stings and Envenomation
    a. Identify and characterize common bite/envenomation patterns of poisonous fauna found in the United States (US).
    b. Describe the management and treatment for common reptile and arthropod envenomation.
    c. Understand and perform initial management of these injuries including venom specific antidotes.
    d. Describe the presenting symptoms of rabies.
    e. Compare the difference in the natural reservoirs for rabies in the US and internationally.
    f. Review the post-exposure prophylaxis for rabies exposure.

Educational Strategies:
(See curriculum chart) Please refer to the curriculum chart of linked objectives and educational strategies.

Evaluation and Feedback:
This innovative curriculum was literature-based and specifically designed to maximize active learning using the flipped classroom learning model. We overcame initial challenges and skepticism from both educators and learners to execute a successful, novel curricular model. Both resident learners and faculty educators have provided positive feedback. Additionally, a survey was administered to each resident prior to initiation of the curricular innovation and repeated at the conclusion of the first 18-month cycle. Learners and educators were enthusiastic about the conference structure and expressed a preference for it rather than the previous, lecture-based didactics.

More recently during the second 18-month cycle of the flipped classroom curriculum, students were surveyed on their perceived quality of instruction of the various program components. A majority of residents (60.9%) reported that the small group discussions were good or excellent, compared to only 26% of residents that felt that our grand rounds sessions during the same time were good or excellent. This curriculum has been delivered to two cohorts of learners, who have received the content twice in three years, with about 50 residents per cycle. On the most recent iteration, residents evaluated the teaching methods as effective, with an average rating of more than 4.5 out of 5 (4 being agree, 5 being strongly agree). The curriculum is critically evaluated and updated by education faculty members in order to ensure educational material remains current and consistent with the emergency

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Educational resources are available within each individual chapter of this environmental emergencies curricular module; however, a complete list of resources and educational materials are listed here.

Appendix A: Cold Injury and Illness

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DIDACTICS AND HANDS-ON CURRICULUM

Topic	Recommended Educational Strategy	Educational Content	Objectives	Learners	Timing, Resources Needed (Space, Instructors, Equipment, Citations of JETem pubs or other literature)	Recommended Assessment, Milestones Addressed
Cold Injury and Illness	-"Flipped" classroom discussion of pre-reading material, case discussions, and discussion questions. -Encourage participants to share clinical experiences to enhance discussion. -30-45 minutes for case and content discussion.	-Indications for and rates of rewarming in different stages of hypothermia. -Resuscitation in hypothermia and concept of core temperature afterdrop. -Characteristic findings and management for soft tissue injuries including chilblains, trench foot and frostbite.	By the end of this session, learners will: -List and define stages of hypothermia; -Review indications and methods of re-warming for hypothermic patients. -Discuss pathophysiology of core temperature afterdrop. -Review management of a hypothermic patient in cardiac arrest. -Discuss differences between cold injuries of the soft tissue (trench foot, chilblains, frostbite) and their management.	PGY-1 PGY-2 PGY-3 Medical Students	Equipment: Projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion. Instructors: 1-2 faculty members or content experts. Recommended senior resident discussion leader. Timing: Small group discussions involve no more than 20 learners and last 45-60 minutes.	Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), pharmacology (PC5), wound management (PC13), medical knowledge (MK). Assessment: Faculty evaluation of resident participation during small group activities. Evaluation: Resident evaluation of small group session content and facilitators. Yearly program evaluation of overall small group component.

DIDACTICS AND HANDS-ON CURRICULUM

Topic	Recommended Educational Strategy	Educational Content	Objectives	Learners	Timing, Resources Needed (Space, Instructors, Equipment, Citations of JETem pubs or other literature)	Recommended Assessment, Milestones Addressed
Heat-Related Illness	-"Flipped" classroom discussion of pre-reading material, case discussions, and discussion questions. -Encourage participants to share clinical experiences to enhance discussion. -30-45 minutes for case and content discussion.	-Pathophysiology and methods of temperature regulation. -Risk factors, clinical differences, and complications of heat exhaustion and heat stroke. -Methods and considerations of rapid cooling.	By the end of this session, learners will: -Discuss the body's methods of heat loss that allow temperature regulation. -Differentiate between patients presenting with heat exhaustion versus heat stroke. -Describe the risk factors for classic heat stroke. -Describe the various methods of cooling for the hyperthermic patient.	PGY-1 PGY-2 PGY-3 Medical Students	Equipment: Projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion. Instructors: 1-2 faculty members or content experts. Recommended senior resident discussion leader. Timing: Small group discussions involve no more than 20 learners and last 45-60 minutes.	Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), pharmacology (PC5), medical knowledge (MK). Assessment: Faculty evaluation of resident participation during small group activities. Evaluation: Resident evaluation of small group session content and facilitators. Yearly program evaluation of overall small group component.

DIDACTICS AND HANDS-ON CURRICULUM

Topic	Recommended Educational Strategy	Educational Content	Objectives	Learners	Timing, Resources Needed (Space, Instructors, Equipment, Citations of JETem pubs or other literature)	Recommended Assessment, Milestones Addressed
Barotrauma, Hyperbaric Oxygen, and Altitude	-"Flipped" classroom discussion of pre-reading material, case discussions, and discussion questions. -Encourage participants to share clinical experiences to enhance discussion. -30-45 minutes for case and content discussion.	-Pathophysiology, diagnosis, and management of common diving-related illnesses including decompression sickness, barotrauma, and gas toxicity. -Pathophysiology, diagnosis, and management of common altitude-related illnesses including acute mountain sickness (AMS), high altitude pulmonary edema (HAPE), and high-altitude cerebral edema (HACE).	By the end of this session, learners will: -Compare/ contrast the different types of diving-related illness including decompression sickness, barotrauma, and gas toxicity. -Describe the treatment options for diving-related illness and list some other clinical indications that hyperbaric oxygen can be used. -Discuss the spectrum of altitude illness and list the preventative and acute treatment approaches.	PGY-1 PGY-2 PGY-3 Medical Students	Equipment: Projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion. Instructors: 1-2 faculty members or content experts. Recommended senior resident discussion leader. Timing: Small group discussions involve no more than 20 learners and last 45-60 minutes.	Milestone: Emergency stabilization (PC1), differential diagnosis (PC4), pharmacology (PC5), medical knowledge (MK). Assessment: Faculty evaluation of resident participation during small group activities. Evaluation: Resident evaluation of small group session content and facilitators. Yearly program evaluation of overall small group component.

Electrical, Radiation, and Submersion Injuries	-"Flipped" classroom discussion of pre-reading material, case discussions, and discussion questions. -Encourage participants to share clinical experiences to enhance discussion. -30-45 minutes for case and content discussion.	-Pathophysiology, diagnosis, and management of drowning. -Diagnostic evaluation, complications of high- and low-voltage electrical injuries. -Decontamination procedures for a radiologically contaminated patient. -Definition and management of Acute Radiation Syndrome (ARS).	By the end of this session, learners will: -Review the key pathophysiology related to drowning and the major organ systems involved. -Discuss management of drowning. -List risk factors for drowning. -Discuss the characteristics of different electrical injuries: high voltage versus low voltage. -Explore the initial management of electrical injuries and how triage of a mass casualty lightning strike differs from standard mass casualty triage. -Define the difference between radiation exposure and radiologic contamination. -Discuss the concept of time/distance/shielding related to radiation exposure. -List the expected manifestations of acute radiation sickness based on the dose absorbed.	PGY-1 PGY-2 PGY-3 Medical Students	Equipment: projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion. Instructors: 1-2 faculty members or content experts. Recommended senior resident discussion leader. Timing: small group discussions involve no more than 20 learners and last 45-60 minutes.	Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), pharmacology (PC5), wound management (PC13), medical knowledge (MK). Assessment: Faculty evaluation of resident participation during small group activities. Evaluation: Resident evaluation of small group session content and facilitators. Yearly program evaluation of overall small group component.

DIDACTICS AND HANDS-ON CURRICULUM

Topic	Recommended Educational Strategy	Educational Content	Objectives	Learners	Timing, Resources Needed (Space, Instructors, Equipment, Citations of JETem pubs or other literature)	Recommended Assessment, Milestones Addressed
Bites, Stings, and Envenomation	"Flipped" classroom discussion of pre-reading material, case discussions, and discussion questions. Encourage participants to share clinical experiences to enhance discussion. 30-45 minutes for case and content discussion.	Pathophysiology, clinical manifestations, and management of reptilian envenomation. Pathophysiology, clinical manifestations, and management of black widow spider envenomation. Pathophysiology, clinical manifestations, and management of rabies infection.	By the end of this session, learners will: -Identify and characterize common bite/envenomation patterns of poisonous fauna found in the United States (US). -Describe the management and treatment for common reptile and arthropod envenomation. -Understand and perform initial management of these injuries including venom specific antidotes. -Describe the presenting symptoms of rabies. -Compare the difference in the natural reservoirs for rabies in the US and internationally. -Review the post-exposure prophylaxis for rabies exposure.	PGY-1 PGY-2 PGY-3 Medical Students	Equipment: Projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion. Instructors: 1-2 faculty members or content experts. Recommended senior resident discussion leader. Timing: Small group discussions involve no more than 20 learners and last 45-60 minutes.	Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), pharmacology (PC5), wound management (PC13), medical knowledge (MK). Assessment: Faculty evaluation of resident participation during small group activities. Evaluation: Resident evaluation of small group session content and facilitators. Yearly program evaluation of overall small group component.

Appendix A:
Cold Injury & Illness

Objectives

By the end of this small group session, learners will:

1. List and define stages of hypothermia;
2. Review indications and methods of re-warming for hypothermic patients;
3. Discuss pathophysiology of core temperature afterdrop;
4. Review management of a hypothermic patient in cardiac arrest;
5. Discuss differences between cold injuries of the soft tissue (trench foot, chilblains, frostbite) and their management.

Case Studies

Case 1: An approximately 30-year-old female is brought into the emergency department (ED) at 4 AM by a man who found her lying at the side of the road. It is 2°F outside and she has no coat or shoes. The man does not know her and is unable to provide any additional history except that she was blue and having trouble breathing when he found her. She is noted to have a decreased level of consciousness and labored breathing though no obvious signs of trauma are present.

Question Prompts:

1. Define the different stages of hypothermia (temperature and symptoms).
    a. Characterization of hypothermia depends on an accurate ***core*** temperature. The best core temperature measurement is made with an esophageal probe. Rectal and bladder temperatures are not as accurate because they tend to lag behind true core temperature. Oral temperatures are typically colder than core temperature and less reliable.
    b. The stage of hypothermia is determined by the patient's symptoms, with typical temperature ranges listed below. The Swiss staging system includes I-IV (with V being death), and is based on symptoms of the patient. The patient who is shivering and completely normal from a mental status standpoint is considered to NOT be hypothermic but simply cold-stressed (>35°C). The patient who is shivering and not quite completely normal is MILDLY hypothermic (32-35°C). The patient who is altered and may or may not be shivering is MODERATELY hypothermic (28-32°C). The patient who is unconscious is SEVERELY hypothermic (<28°C). The shivering response usually ceases below 30-31°C. These temperature ranges are generally accepted ranges where symptoms develop, however this relies on an accurate core temperature which may be difficult to obtain especially in the field.
    c. Hypothermia stages:

Staging of hypothermia		Symptoms
I: Mild	32-35°C (**89.6-95°F**)	Conscious, but shivering
II: Moderate	28-32°C (**82.4-89.6°F**)	Altered mental status, shivering stops
III: Severe	24-28°C (**75.2-82.4°F**)	Unconscious, no shivering
IV: Look dead	<24°C (**<75.2°F**)	No vital signs
V: Actually dead	<9°C **(<48.2°F) –** unlikely to survive	Death due to hypothermia

2. Discuss indications for rewarming methods (external vs internal, active vs passive). What are the rates of re-warming based on method?
    a. Passive rewarming takes advantage of the heat that is already being produced by the body. This is useful for the mildly hypothermic patient who is shivering. The shivering response can increase temperature by 1-3°C/hour. Passive rewarming techniques include removing wet clothes and replacing with dry clothes, seeking shelter, wrapping with a barrier layer to hold in the heat produced by radiation, and providing caloric intake to support metabolism.
    b. Active external rewarming is indicated for the patient who is moderately hypothermic. Depending on the environment, this may include chemical heating pads, warm water bottles, or warmed air (Bair Hugger). Once the patient loses consciousness (severely hypothermic), active internal rewarming is indicated; these techniques include: use of warmed humidified air on the ventilation circuit, use of warmed intravenous (IV) fluids (when fluids are needed for resuscitation; warmed IV fluid will not actively rewarm but prevents cooling from room temperature saline), body cavity lavage (thorax, peritoneal cavity, bladder), and intravascular devices (for example: Zoll catheter). Extracorporeal membrane oxygenation (ECMO) is the ultimate internal rewarming method and is indicated for loss of vital signs (stage IV hypothermia).
    c. Rewarming techniques and associated rate of rewarming:

Rewarming Method	Rates
Warmed IV fluids	Non-significant, does not cool either
Warm O_2	1°C/hour with mask; 1.5°C/hour with endotracheal tube
Shivering	1.5°C/hour
Warming blanket	2°C/hour
Peritoneal lavage	3°C/hour
Thoracic lavage with chest tubes	3-6°C/hour
ECMO	9-18°C/hour

3. This patient has an initial core temperature of 81°F. Thirty minutes after resuscitation and warming was initiated, the core temperature is now 75°F and the patient loses pulses. Telemetry shows ventricular fibrillation. Why did the temperature decrease despite warming measures? What is the role for advanced cardiac life support (ACLS) drugs and defibrillation in severe hypothermia?
    a. Core temperature afterdrop is the result of cooled blood in the periphery being mobilized back to the core as rewarming measures are conducted. Increased peripheral blood flow and decreased vasoconstriction increase the cool blood moving back to the warm core. This

afterdrop can be significant and produce instability in a severely hypothermic patient. For this reason, distal limb rewarming is generally not advised in hypothermic patients. Instead, core rewarming measures should be initiated such as thoracic lavage with chest tubes or ECMO. Distal limb rewarming in the mildly hypothermic, alert patient is acceptable.[3] This can be accomplished by placing the patients arms and legs in warm water. Again, this is not recommended in moderately or severely hypothermic patients.

b. Management of the hypothermic unresponsive or arrest patient can be challenging. These are some of the principles to consider:
    i. Patients are bradycardic and thus pulse checks should be done for up to one minute or assisted by use of Doppler.
    ii. Minimize patient movement to avoid cardiac dysrhythmias.
    iii. Airway management and chest compressions are unchanged in hypothermic patients compared with normothermic patients.
    iv. If defibrillation is indicated, then shock up to three times. Hold further shocks until core temperature has increased by 1-2°C. Normal defibrillation protocol is resumed once temperature is >30°C.
    v. Vasoactive ACLS drugs have not been well-studied in this population, though it is reasonable to withhold use until temperature is >30°C. Double the dosing time interval to every 6-10 minutes for ACLS drugs when temperature is 30-35°C.
c. A useful algorithm for management of the cold patient is outlined by Zafren, et al, in Wilderness Medical Society practice guidelines for the out-of-hospital evaluation and treatment of accidental hypothermia.[3]

Case 2: You are hiking in Denali National Park with friends when you come upon a man in his twenties who seems distraught and anxious. He states that he was hiking by himself and is lost. Yesterday he fell near a crevasse. He was able to self-arrest before going over the edge, but he lost his pack, trekking poles and ones of his gloves into the crevasse. Without his map, GPS, or any means of communication, he was stranded overnight outside and without shelter. He is ambulatory, not shivering, and able to answer questions appropriately. His left hand is exposed and cold to the touch. The skin is white with blue mottling and his distal fingers are insensate. You are at approximately 13,500 ft elevation with an ambient air temperature 30-35°F (wind chill temperature is 20-25°F). Overnight temperatures were 10-15°F.

Question Prompts:

1. Describe the difference between different cold injuries of the soft tissue (trench foot, chilblains, frostbite). What are the characteristic findings for each degree of frostbite (first to fourth)? Discuss the management of soft tissue cold injuries.
    a. “Trench foot” is a non-freezing, immersion injury with initial vasoconstriction followed by an hyperemic vasodilated phase. This can be seen in environments with ambient temperatures above freezing. This is almost always associated with wet skin. This is treated with analgesic medications, keeping the foot dry, and elevation of the foot to avoid excessive edema.

Chilblains (pernio) is an acrally located cutaneous eruption resulting from exposure to cold. This is self-limited and also requires only supportive care.

b. Frostbite is soft tissue injury occurring through freeze-warming cycles, which is more complex than just freezing of tissue itself. This injury is classified similar to burns. The characteristic findings include:[4]

First degree	white-yellowish, edema
Second degree	erythema with clear blisters
Third degree	deep blisters with hemorrhagic fluid
Fourth degree	injury thru dermis into the deep tissue (muscle and bone)

c. For deep frostbite (third and fourth degree), bone scanning or magnetic resonance imaging (MRI) is often needed to define the extent of injury. Time is also needed for the distribution of injury to fully demarcate.

2. What are the pre-hospital and initial hospital management principles for treatment of frostbite?
   a. The most important principle of frostbite treatment is to avoid any further freeze-thaw cycles because this is the most destructive pattern of the injury. This could possibly mean intentionally NOT rewarming tissue in the field until sustained resources are available. Dressings that will accommodate for swelling may be loosely applied but care should be taken not to rub or compress the skin. Once rewarming is available, the tissue can be placed in a 37-39°C water bath for 20-30 minutes. Analgesic medication is a must; a non-steroidal anti-inflammatory drug (NSAID) may be sufficient for mild cases, though opiate analgesia may also be considered.
   b. In the hospital setting, severe frostbite is often managed by burn specialists. Novel approaches to treatment are focused on the microvascular thrombosis that occurs with reperfusion. Current protocols include use of iloprost (a prostacyclin vasodilator) for third degree frostbite and also with tissue plasminogen activator (tPA) for fourth degree injuries. Long-term care may include amputation for more severe cases; however, this is not something that can be predicted at initial injury.

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Brown DJ, Brugger H, Boyd J, Paal P. Accidental hypothermia. *N Engl J Med*. 2012;367(20):1930. doi: 10.1056/NEJMra1114208

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Appendix B:
Heat-Related Illnesses

Objectives

By the end of this small group session, learners will:

1. Discuss physiological compensatory mechanisms for acute acid / base disorders
2. Discuss the body's methods of heat loss that allows temperature regulation.
3. Differentiate between patients presenting with heat exhaustion versus heat stroke.
4. Describe the risk factors for classic heat stroke.
5. Describe the various methods of cooling for the hyperthermic patient.

Case Studies

Case 1: A 55-year-old female is brought to the medical aid tent during an outdoor concert. The patient is complaining of dizziness and nausea. She started tailgating around 9am and has been outside for the past seven hours. The ambient air temperature is 92°F with 90% relative humidity and there is no cloud-cover or shade where she has been standing. She reports some alcohol consumption but can't quantify how much or whether she had any non-alcoholic hydration. She felt like she might pass out at one point but did not actually do so. Vital signs show a heart rate of 100/minute, blood pressure of 118/84 mmHg, pulse oximetry of 98%, and oral temperature of 100.5°F. Examination is notable for clammy skin and diaphoresis.

Question Prompts:

1. What are the body's methods of heat loss that allow temperature regulation?
    a. There are four methods of transferring heat out of the body:
        i. Radiation - direct heat loss
            1. Minimal heat loss in a hot environment.
        ii. Conduction - heat loss due to contact with colder surface
            1. Minimal heat loss in a hot environment, though can be significant in a cold environment when the body is trying to retain heat.
        iii. Convection - heat loss due to air movement
            1. An effective mechanism but it requires active circulation of the air or fluid surrounding the body.
        iv. Evaporation - heat loss due to warmed fluid loss, like sweat
            1. The body's primary mechanism for heat dissipation, but the efficacy of this is reduced as ambient humidity increases.
    b. Higher mechanisms of cooling also contribute to temperature regulation. This includes the behavioral actions of humans in hot environments. Examples include: decreasing physical activity, increasing hydration, seeking shade or rest, and limiting heat exposure.

2. What are the symptoms of heat edema, heat cramps, and heat syncope? What is the underlying pathophysiology?
    a. Heat edema is **lower extremity swelling that occurs secondary to compensatory vasodilation resulting in elevated hydrostatic pressures and vascular leak.**
    b. Heat cramps are **involuntary skeletal muscle contractions usually occurring in the calves** and associated with electrolyte-poor hydration.
    c. Heat syncope typically occurs due to peripheral vasodilation, orthostatic blood pooling, and low blood volume secondary to dehydration. Healthcare providers should be vigilant and comprehensive in their approach to these patients to address other causes of syncope.
3. Compare and contrast the symptoms and findings related to heat exhaustion versus heat stroke.
    a. This patient has a presentation consistent with heat exhaustion. Heat exhaustion shares many similar features with heat stroke including: elevated temperature, tachycardia, and diaphoresis (though not universally present in heat stroke). The temperature is usually higher with heat stroke (>40C), though this can be inconsistent. The main differentiating factor for heat stroke is the presence of end-organ dysfunction. The central nervous system (CNS) is the most sensitive organ system to hyperthermia. The liver is also sensitive to elevated temperatures. Thus, altered mental status, neurological deficits, or hepatic dysfunction are keystones of the diagnosis of heat stroke. CNS dysfunction is most commonly considered with heat stroke because it is manifest without need for labs and because the hepatic dysfunction may lag up to 12 hours after heat exposure.
    b. Here is a table comparing heat exhaustion to heat stroke. The most important feature that distinguishes heat stroke from heat exhaustion is the presence of neurologic deficits.

	Heat Exhaustion	**Heat Stroke**
Temperature	<40C (104F)	≥40C (104F)
Mental Status	Normal mental status Brief period of mild confusion Brief syncope	Altered mental status – hallmark of heat stroke (confusion, poor attention, poor memory, agitation, delirium, confusion, hallucinations, coma, seizures)
Airway & Breathing	Clear airway May be tachypneic	Airway may be compromised Tachypneic
Circulation	Tachycardic Normal BP Mild-moderate dehydration	Tachycardic Hypotension/ wide pulse pressure Moderate-severe dehydration
Skin	Sweating	Dry skin (classic heat stroke) Sweating (exertional heat stroke)
Other	Nausea/vomiting Headache Generalized fatigue Weakness Hypo/hypernatremia	Nausea/vomiting Diarrhea Disseminated intravascular coagulation (DIC) Rhabdomyolysis Renal failure

		Cardiogenic shock Liver failure

4. Discuss the management plan for this patient.
    a. The management for this patient's heat exhaustion starts with removing the patient from the hot environment if possible.
    b. Most patients with heat-related illness have some degree of hypovolemia so fluid replacement is usually warranted. Oral rehydration with slightly hypotonic solution is adequate for heat exhaustion, though anti-emetic medication may be required.
    c. Clothes should be removed to assist convective heat loss with fans if available. If the patient does not improve with 30 minutes of rest and hydration, she may require transfer to a higher level of care. Close monitoring should be made for progression to heat stroke.

Case 2: A 19-year-old male student athlete is brought to the emergency department (ED) by paramedics after he collapsed during pre-season team practice. He takes methylphenidate (Ritalin) for attention deficit hyperactivity disorder (ADHD) but is otherwise healthy. He does not have any complaints, but he is only oriented to name and he is confused when asking him questions. Vital signs show a pulse 110/min, blood pressure of 100/62 mmHg, pulse oximetry of 98%, and oral temperature of 104.2°F. Examination is notable for warm skin and diaphoresis.

Question Prompts:

1. What are the typical circumstances for classic heat stroke versus exertional heat stroke? What are some risk factors for these to occur?
    a. Heat stroke is sub-classified based on the context of the heat exposure. There are two types of heat stroke, classic and exertional heat stroke.
        1. Classic heat stroke results from exposure to high environmental temperatures, usually in elderly individuals or those who do not have control over their ability to moderate their presence in a hot climate. These individuals typically have hot dry skin.
        2. Exertional heat stroke is a result of elevated core body temperatures due to strenuous activity. This is more likely to present as a young person who may still be sweating. This is often seen in athletes and military individuals who over-exert in a hot climate especially with high humidity.
    b. **Risk factors for classic heat stroke include:**
        1. Alcohol
        2. Recreational drugs
        3. Anticholinergics
        4. Anhidrosis - an inability to sweat, which can be congenital, caused by other medical causes, or by medication side effects, among other causes
        5. Obesity
        6. Coma
        7. Psychosis

8. Physical or mental disability
9. Extremes of age
10. Diuretics
11. Poverty in an inescapably hot climate

2. What are methods for rapid cooling and how does their relative efficacy compare?
    a. The primary treatment for heat stroke (classic or exertional) starts with considerations of airway, breathing and circulation (ABCs) and basic resuscitation. These patients may present with a spectrum of manifestations which could be relatively mild or include cardiac arrest. Airway should be secured if warranted and intravenous (IV) access established. All of these patients need comprehensive care and should be transported to an ED as soon as possible.
    b. Once hemodynamic stability is achieved, the focus should turn to rapid cooling of the patient. Rapid cooling is the most effective method to prevent heat-related mortality. The recommended target temp range is 38-39°C to prevent overshooting normothermia. Cold water immersion is the most rapid form of cooling and is especially successful with exertional heat stroke. Shivering does not counteract this method of cooling. The potential drawbacks of ice water immersion include limited ability for monitoring and lack of adequate vessel large enough for an ice bath in an ED. Innovative attempts have been performed using a large plastic sealable bag (i.e. a "body bag") filled with ice and water and partially closed around the patient to maintain the fluid contact. Evaporative cooling with mist and fan is also effective. This method is much easier to perform in most EDs. Other cooling methods include ice pack cooling to groin and axilla. Cooling blankets are generally not effective. Body cavity lavage with cold fluid is not well studied and should be avoided except in severe cases.[6] In these cases, cardio-pulmonary bypass may be more effective and more practical than body cavity lavage.
3. What are other considerations in the acute treatment of heat stroke?
    a. Medications have little role in the treatment of hyperthermia, and antipyretics and dantrolene should be avoided. Complications such as seizures and severe shivering can be managed with short acting benzodiazepines. These patients are often hypovolemic, and empiric IV isotonic fluid hydration is warranted.
    b. Consideration of end-organ failure aside from neurologic effects is important. Rhabdomyolysis is common and monitoring of creatine kinase (CK) levels is advised. Other screening labs should include: compete blood count (CBC), electrolytes, arterial or venous blood gas, glucose, blood urea nitrogen (BUN)/creatinine, liver enzymes, coagulation studies, urinalysis, urine myoglobin, and electrocardiogram (ECG).
4. List some of the common complications of heat related illness.
    a. The complications of heat stroke are generally the direct or indirect manifestations of various end-organ dysfunction. These may include: pulmonary edema, hemorrhage and acute respiratory distress syndrome (ARDS), cardiac failure and arrhythmias (relatively unusual in heatstroke, and resolve as the temperature comes down),[4] hypotension (due to hypovolemia, vasodilatation and cardiac dysfunction), seizures, rhabdomyolysis, renal failure, hepatic injury, and coagulopathy such as DIC.

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Appendix C:
Barotrauma, Hyperbaric Oxygen, and Altitude

Objectives

By the end of this small group session, learners will:

1. Compare/contrast the different types of diving-related illness including: decompression sickness, barotrauma, and gas toxicity.
2. Describe the treatment options for diving-related illness.
3. Discuss the spectrum of altitude illness and list the preventive and acute treatment approaches.

Case Studies

Case 1: A 20-year-old male diver presents to emergency department (ED) with right elbow pain. He completed a scuba dive at a maximum depth of 100 feet sea water for 40 minutes. He was in cold water, spearfishing, and the pain began approximately two hours after the dive. He describes the pain as throbbing, 6/10 severity. Review of systems are otherwise negative. He has no past medical history and no pertinent social history. Physical exam shows right elbow with normal range of motion but pain with movement. There is no edema or ecchymosis, and sensory and motor functions are intact.

Question Prompts:

1. What is the differential diagnosis for this patient? What diving related illnesses are not likely in this patient and why?
    a. The differential includes decompression sickness (DCS) or musculoskeletal injury. Specifically, the patient appears to have DCS type 1 because there are only pain symptoms associated with his complaint. DCS type 1 could also include skin findings with a characteristic rash called cutis marmorata.

Cutis marmorata in DCS1:

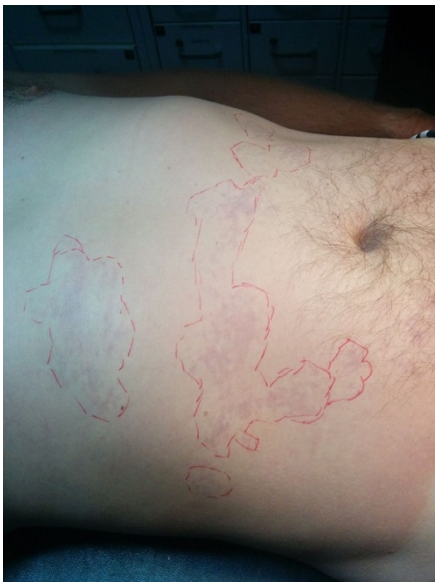

Image source: Huckfinne. Cutis marmorata DCS I. In: Wikimedia Commons. https://commons.wikimedia.org/wiki/File:Cutis_marmorata_DCS_I.jpg. Published July 25, 2014. Accessed January 20, 2019. Public domain.

b. DCS type 2 requires the additional findings of neurologic deficits (usually sensory or motor changes but can also include extreme fatigue).
c. DCS type 3 is rare but more profound including pulmonary symptoms (cough, dyspnea), cardiovascular collapse, or cerebellar symptoms related to inner ear bubble formation (vertigo, nystagmus, and vomiting).

Decompression sickness type 1	Only pain symptoms, may include rash (cutis marmorata)
Decompression sickness type 2	Neurologic deficits – including extreme fatigue
Decompression sickness type 3	Pulmonary symptoms (cough, shortness of breath), cardiovascular collapse, cerebellar symptoms (vertigo, nystagmus, vomiting)

d. The features of the dive itself that favor DCS are greater depth and more prolonged duration. Both of these factors increase the nitrogen that dissolves in the blood at depth and then eventually returns to bubble form (think of opening a can of Guinness with nitrogen in it; bubbles that form are due to the nitrogen going from solute to gas as the can is rapidly depressurized). These bubbles get trapped in the joints and vascular system (primarily the venous side) when decompression back to the surface is not slow enough to allow the bubbles to be flushed and filtered in the lungs. Bubbles at the air-blood & air-endothelial interfaces are highly reactive, initiating thrombotic and inflammatory processes.
e. Barotrauma is unlikely to be at play here. Barotrauma occurs due to rapid changes in pressure that result in corresponding changes in the volume of air in a fixed space (recall Boyle's Law: $P_1V_1 = P_2V_2$; or the volume of a gas will vary inversely with pressure).

Barotrauma can affect any air-filled part of the body, though the most common site is the middle ear due to inability of the eustachian tube to equilibrate the pressure changes in that small cavity. Other sites of non-pulmonary barotrauma include: the sinuses, teeth, the face (covered by the dive mask), and rarely the gastrointestinal (GI) tract. Pulmonary barotrauma is less common but can be more severe. This occurs when a diver ascends rapidly while breath holding. The manifestations include: pneumomediastinum, pneumothorax, and arterial gas embolism (AGE). The damaged lung parenchyma from over inflation allows large air bubbles to escape into the pulmonary circulation. These bubbles can traverse the left heart and embolize to the cerebral circulation. Thus, AGE presents with stroke-like symptoms rapidly after surfacing (usually within 10 minutes of surfacing). This type of diving related illness does not require prolonged duration or deep depth, only rapid ascent to the surface.

f. Gas toxicity primarily relates to a phenomenon called nitrogen narcosis. This refers to the intoxicating effect of breathing inert gas (nitrogen) at high partial pressures. It affects individuals differently, but usually is not seen at depths less than 100 feet of sea water. Symptoms include: loss of judgment, skill, or concern, euphoria (similar to alcohol intoxication), and inappropriate laughter. Oxygen can also cause side effects at high partial pressures, but this is only noted with prolonged exposures. This patient's symptoms are not consistent with gas toxicity.

g. While musculoskeletal injury is always a possible etiology for acute joint pain, the context of this dive requires serious consideration for DCS and empiric treatment for it.

2. What is the best treatment modality for each diving related diagnosis?
    a. The treatment for any DCS is compression in a hyperbaric oxygen (HBO) chamber. The basis for this treatment is recompression followed by slow decompression to allow the nitrogen to be washed out without trapping bubbles in the joints and microvasculature. Additionally, 100% oxygen is breathed during the treatment to avoid additional nitrogen loading. A qualified hyperbaric physician should be consulted for specific HBO protocols. The Divers Alert Network (DAN) is a good resource for community physicians in the United States (DAN Hotline: 919-684-9111).
    b. Emergent hyperbaric oxygen treatment is also the answer for AGE, though the mechanism is somewhat different. In this case, the rapid recompression eliminates the bubble in the cerebral circulation that is causing ischemia.
    c. Treatment for the other types of barotrauma is primarily supportive, though close observation for worsening is often warranted. Gas toxicity is self-limited and resolves with return to normal partial pressures of nitrogen or oxygen.

Case 2: A 38-year-old female from Miami, Florida is mountain climbing in Colorado. She is just starting her second day of climbing and is at 13,800 feet. She starts to get short of breath and develops a dry cough. She is now experiencing fatigue with minimal effort. The previous day she noted some mild headache and nausea but dismissed the symptoms. Her pulse is fast but other vital signs are unknown because she is not currently in a healthcare location. She reports compliance with recommended prophylactic medications.

Question Prompts:

1. What is the differential diagnosis for this patient? What other conditions should be considered and monitored for?
    a. The differential includes the spectrum of altitude-related illness. These conditions are all related to rapid ascent to high altitudes and the body's response to the resultant hypobaric, hypoxic environment.
    b. The most common and benign type of altitude illness is called acute mountain sickness (AMS). This is a syndrome of nonspecific symptoms usually seen above 10,000 feet.
    c. Simple diagnostic criteria for AMS include *headache* plus one of the following:
        1. Gastrointestinal upset, such as anorexia, nausea, vomiting
        2. General weakness or fatigue
        3. Dizziness or lightheadedness
        4. Difficulty sleeping.
    d. The risk factors for AMS include: Prior altitude illness, residence less than 2500 feet, exertion, and rapid ascent.
    e. The most likely etiology on the differential is a more severe form of altitude illness called high altitude pulmonary edema (HAPE). This is the most common fatal manifestation of altitude illness. The pathophysiology relates to hypoxia causing pulmonary vasoconstriction and subsequently edema.
    f. Clinical presentation of HAPE includes:
        1. Dyspnea on exertion or resting dyspnea
        2. Fatigue with minimal effort
        3. Dry cough
        4. Progressive tachypnea and tachycardia
        5. Fever (common)
        6. Pink or bloody sputum (late sign)
        7. Rales in bilateral lung fields.
        8. Acute mountain sickness is seen concomitantly with HAPE in 50% of patients afflicted by HAPE.
    g. Also on the differential, but less concerning for this patient is high altitude cerebral edema (HACE). This can also be severe and is thought to represent progression from mild AMS. The symptoms include those of AMS plus neurologic deficits (ataxia, seizures, speech changes, or altered mental status). Ataxia is the most common symptom. All patients with AMS must be monitored closely for progression to HACE because *mortality exceeds 60%* once coma is present.
2. What is the best treatment modality for each possible diagnosis?
    a. Acute mountain sickness usually peaks at 24-48 hours of altitude and resolves by 96 hours as acclimatization is achieved. Slow ascent with sleep at lower altitudes is the best prevention. Medications can be used prophylactically with acetazolamide 125mg twice daily being used most commonly. Acute treatment of AMS includes no further ascent until

symptoms have resolved, monitoring for progression to HACE, and medications (acetazolamide, dexamethasone, analgesics and anti-emetics).

b. High altitude cerebral edema can be treated prophylactically for individuals with prior HAPE. The primary goal of acute treatment is rapid descent with minimal exertion. If available, moderate flow supplemental oxygen is useful (4-6L/minute nasal cannula). If rapid descent is not possible, a portable hyperbaric chamber (Gamow bag) can be used for temporizing descent. Nifedipine is the only proven pharmacologic option. Recommended dosing is Nifedipine ER 30mg every 12 hours.
c. Treatment for HACE is similar to HAPE with rapid descent being the priority. Supplemental oxygen and a Gamow bag may also be useful temporizing measures. High dose dexamethasone can also be used (8mg loading followed by 4mg every 6hrs).

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Appendix D:
Environmental Potpourri—Electrical, Radiation, and Submersion Injuries

Objectives

By the end of this small group session, learners will be able to:

1. Review the key pathophysiology related to drowning and the major organ systems involved.
2. Discuss management of drowning.
3. List risk factors for drowning.
4. Discuss the characteristics of different electrical injuries: high voltage versus low voltage.
5. Explore the initial management of electrical injuries and how triage of a mass casualty lightning strike differs from standard mass casualty triage.
6. Define the difference between radiation exposure and radiologic contamination.
7. Discuss the concept of time/distance/shielding related to radiation exposure.
8. List the expected manifestations of acute radiation sickness based on the dose absorbed.

Case Studies

Case 1: A 10-month-old male presents to the emergency department (ED) via ambulance. The patient was placed in a bathtub by his parent who subsequently left to get her cell phone for one minute. When she returned the infant was face down in the water, apneic and cyanotic. She called 911 and proceeded to give rescue breaths. When paramedics arrived, the patient was awake and coughing.

Question Prompts:

1. What is the pathophysiology of drowning and how does it lead to mortality?
    a. Drowning is the process of experiencing respiratory impairment from submersion/immersion in liquid. Drowning can be due to: submersion (the airway goes below the level of the surface of the liquid) or immersion (a liquid is splashed across a person's face, eg waterboarding). Respiratory impairment must be present for drowning to have occurred.
    b. Terms such as "near drowning," "dry or wet drowning," "secondary drowning," "active and passive drowning," and "delayed onset of respiratory distress" are no longer used.[4]
    c. Preferred terminology is non-fatal or fatal. Non-fatal drowning is where the drowning process is interrupted and the person survives. Fatal drowning is an event when the person dies during the drowning process at any stage.[4]
    d. The primary cause of mortality is related to pulmonary dysfunction. Aspirated water causes surfactant dysfunction and washout. An osmotic gradient is formed which damages the

alveolar–capillary membrane. This disrupts the integrity of the membrane, increases its permeability, and exacerbates fluid, plasma, and electrolyte shifts. Often massive pulmonary edema ensues. This results in decreased lung compliance, ventilation-perfusion mismatch, atelectasis and bronchospasm.

e. The secondary organ systems involved are a cascade from the primary hypoxemia resulting from the lung dysfunction. The secondary effects include cardiac, neurological, renal, and hematologic.

2. What are some known risk factors for drowning?
    a. The major risk factor for pediatric drowning is inadequate supervision of small children. This includes lack of mitigating measures for residential areas such as a fence with locking gate around a pool, a pool cover, or buckets and tubs that are not emptied when not in use. In-ground swimming pools without complete 4-sided isolation fencing are 60% more likely to be involved in drownings than those with 4-sided isolation fencing.
    b. Other more generalized risk factors include: neurological events (eg, epilepsy, stroke), cardiac events (eg, myocardial infarction, hypertrophic cardiomyopathy, dysrhythmia, long QT, and short QT), impaired judgement due to intoxication, trauma, or overdose.
    c. Mitigation measures can significantly decrease many of these risk factors.
    d. Hypothermia associated with drowning can provide a protective mechanism that allows persons to survive prolonged submersion episodes. Rate of cerebral oxygen consumption is reduced by ~ 5% for each reduction of 1°C in temperature within the range of 37°C to 20°C. For hypothermia to be protective, the body temperature must drop rapidly to prevent significant cerebral hypoxia before the metabolism slows.
3. What would be the appropriate diagnostic evaluation and management for this patient? What should the disposition be for this patient?
    a. The diagnostic tests to consider include: arterial blood gas (ABG), chemistry panel, electrocardiogram (ECG), chest X-ray and pulse oximetry monitoring. Secondary trauma should also be considered and may require more comprehensive imaging.
    b. Treatment begins with addressing airway, breathing and circulation (ABCs). Re-warming should be initiated if hypothermic. Treatment should be focused on the organ systems involved based on physical findings, vital signs, labs, and imaging.
        i. Neurologic measures include: Keeping the head of bed elevated, maintaining a low normal CO_2 and mean arterial pressure of 80mmHg (no need for intracranial pressure monitoring), benzodiazepines if needed for seizures, and consideration of therapeutic hypothermia (by actively re-warming only to 34°C) to prevent secondary brain injury.[4]
        ii. From a respiratory standpoint, the patients are at risk for acute lung injury (ALI) and acute respiratory distress syndrome (ARDS). Protective lung ventilation settings should be used for intubated patients. For less severe patients (like this 10-month-old), beta-agonist bronchodilators can be used with or without non-invasive positive pressure ventilation.
        iii. Cardiac management includes telemetry monitoring, intravenous (IV) fluid support for hypotension, and stabilization of cardiac dysrhythmias. The cardiac treatment

should be adjusted based on body temperature (including the use of defibrillation and advanced cardiac life support [ACLS] medication usage for severe hypothermia).

c. Disposition for this patient should be observation for at least four to six hours. Despite improvement with rescue breathing, the patient does have risk for worsening depending on degree of aspiration and amount of hypoxia experienced. However, if the patient is back to a normal neuro-behavioral baseline, has no significant abnormalities on screening tests, and shows no decline in cardiopulmonary function over this observation period, then discharge with precautions for return is warranted. If the Glascow coma score (GCS) is depressed or the patient does not have rapid improvement in symptoms, then admission is needed.
d. Management of adult patients after submersion injury is similar. While the mechanism of submersion injuries may be different, such as adults are more likely to be involved in open water drowning versus bathtub drowning, the management principles remain the same.

Case 2: A 4-year-old male presents to the ED after getting "shocked" by placing a key into an electrical outlet at home. His father reports that he was in the other room when he heard the patient cry out. He found his son holding his hand and crying. On exam there are deep burns to the distal pads of his right thumb and index finger but no other wounds.

Question Prompts:

1. What diagnostic evaluation would be most appropriate? How does the work-up and management differ for high vs low-voltage electrical injuries?
    a. The mechanisms of injury related to electrical exposure fall into three categories:
        1. Direct effect of the electrical current on the organs and tissue.
        2. Thermal burns due to conversion of the electrical current into heat.
        3. Trauma secondary to being thrown or falling.
    b. The level of energy is classified into low-voltage (less than 1000V), high-voltage (greater than 1000V), lightning, or electrical arc.
    c. The extent of injury depends on these factors:
        1. Strength of the current
        2. Alternating current (AC) vs direct current (DC)
        3. Pathway of the current
        4. Voltage
        5. Resistance of the skin (ie, wet vs dry)
        6. Duration of contact
    d. The mechanism of injury and level of energy dictates the diagnostic evaluation for electrical injuries.
    e. This patient has a low-voltage injury with evidence of severe burn. Most low-voltage injuries in asymptomatic patients without evidence of severe burns can be managed with no diagnostic testing and early discharge from the ED.
    f. Patients with high-voltage injuries, lightning strikes, and low-voltage injuries with severe burns should have comprehensive screening tests to include: Complete blood count (CBC),

basic metabolic panel (BMP), creatine kinase (CK), coagulation studies, and urine myoglobin. ECG should be obtained in lightning injuries, though in lower voltage injuries ECG is unlikely to affect management.[5] The clinical importance of elevated troponin levels after electrical injury is unclear.

2. What delayed complication can occur due to an oral commissure burn from biting into an electrical cord?
    a. Burns to the oral commissure can be seen in toddlers who chew an electrical cord. Close follow-up is warranted due to risk of severe hemorrhage as the eschar comes off allowing bleeding from the labial artery. Fortunately, this is a rare complication (only 2 out of 48 in one case series) and generally will occur in a delayed fashion seven to ten days after initial injury. Other complications include contraction of the mouth leading to facial deformities.
3. Consider a different scenario: How is mass causality triage different in lightning strikes?
    a. Typical triage protocol would label an adult patient who is not breathing as deceased (black tag). However, in lightning strikes it is recommended to "reverse triage," where those in cardiopulmonary arrest are managed first.
    b. Despite these patients lacking vital signs and appearing deceased with fixed and dilated pupils, many have the potential for full recovery.
    c. Management of these patients follows standard Advanced Cardiac Life Support (ACLS) with high quality chest compressions, manual respirations, and defibrillation if in a shockable rhythm.

Case 3: A 12-year-old male and his 38-year-old mother are brought in by paramedics after an explosion at a nearby park. The explosive consisted of shrapnel and some sort of "radioactive material." The male patient is awake and alert with some open wounds noted to both lower extremities. The female has no obvious traumatic injuries, but she is very distressed and agitated. She has been following Twitter and is very concerned about their exposure to this "dirty bomb."

Question Prompts:

1. What decontamination procedures should be done prior to medical evaluation? What is the priority for care of a contaminated patient?
    a. The most effective decontamination method is simply removing the clothing of a patient. This may reduce contamination by 90-95%. The next step is simply washing with copious soap and water. Decontaminate wounds first and then intact skin. Start with areas of the body with the highest measured levels. If open wounds are involved, scrubbing the wound with gauze and soap/water is advised. If there is significant contamination of hair, shaving can be performed.
    b. The medical triage and treatment of the radiologically contaminated patient is always the priority. If a patient is in extremis, then decontamination efforts can be delayed until medical stability is achieved.[1] This does not mean that screening for level of contamination cannot be done while medical resuscitation is being provided. It also does not mean that healthcare providers should not do as much as they can to protect themselves from

exposure (for example: use of proper personal protective equipment and limited time in close proximity to the patient prior to decontamination). Unlike other types of exposures (like chemical weapons) that may pose an ongoing threat to caretakers, radiation contamination is dramatically reduced by removing the patient's clothes and extricating them from the scene of exposure.

c. Decontamination is a cyclic process of measuring levels of radioactivity on the patient, applying a decontamination measure, and then repeating until the radiation level is less than twice background or multiple decontamination attempts yield no further reduction in radiation level.

2. What defines acute radiation syndrome (ARS) and how would you evaluate and treat it?
    a. The four stages of ARS are:
        1. **Prodromal stage (nausea, vomiting, diarrhea stage):** The classic symptoms for this stage are nausea, vomiting, as well as anorexia and possibly diarrhea (depending on dose), which occur from minutes to days following exposure. The symptoms may last (episodically) for minutes up to several days.
        2. **Latent stage:** In this stage, the patient looks and feels generally healthy for a few hours or even up to a few weeks.
        3. **Manifest illness stage:** In this stage the symptoms very based on the specific syndrome (see below) and last from hours up to several months.
        4. **Recovery or death:** Most patients who do not recover will die within several months of exposure. The recovery process lasts from several weeks up to two years.
    b. The three classic acute radiation syndromes are bone marrow syndrome, gastrointestinal syndrome, and cardiovascular/central nervous system syndrome. Patients will manifest one or multiple of these specific syndromes during the "manifest illness" stage of ARS.
        1. **Bone marrow syndrome**: Sometimes referred to as hematopoietic syndrome, the full syndrome will usually occur with a dose between 0.7 and 10 Gy (70 – 1000 rads) though mild symptoms may occur as low as 0.3 Gy or 30 rads. The survival rate of patients with this syndrome decreases with increasing dose. The primary cause of death is the destruction of the bone marrow, resulting in infection and hemorrhage.
        2. **Gastrointestinal (GI) syndrome:** The full syndrome will usually occur with a dose greater than approximately 10 Gy (1000 rads) although some symptoms may occur as low as 6 Gy or 600 rads. Survival is extremely unlikely with this syndrome. Destructive and irreparable changes in the GI tract and bone marrow usually cause infection, dehydration, and electrolyte imbalance. Death usually occurs within two weeks.
        3. **Cardiovascular (CV)/ central nervous system (CNS) syndrome:** The full syndrome will usually occur with a dose greater than approximately 50 Gy (5000 rads) although some symptoms may occur as low as 20 Gy or 2000 rads. Patients with this syndrome typically die within 3 days. Death is likely due to collapse of the circulatory system as well as increased pressure in the confining cranial vault as a result of increased fluid content caused by edema, vasculitis, and meningitis secondary to direct radiation injury.

c. If a patient is suspected or known to have been exposed to a large radiation dose, draw blood for CBC analysis with special attention to the decreasing lymphocyte count, every 2 to 3 hours during the first 8 hours after exposure (and every 4 to 6 hours for the next 2 days).[6] Observe the patient during this time for symptoms and consult with radiation experts before ruling out ARS.
d. If no radiation exposure is initially suspected, you may consider ARS in the differential diagnosis if a history exists of nausea and vomiting that is unexplained by other causes. Other indications are bleeding, hair loss, or white blood count (WBC) and platelet counts abnormally low a few days or weeks after unexplained nausea and vomiting. Again, consider CBC and chromosome analysis and consultation with radiation experts (such as poison control) to confirm diagnosis. After initial screening labs and estimation of exposure, begin the following more long-term measures (as indicated):
    1. Supportive care in a clean environment (if available, the use of a burn unit may be effective).
    2. Prevention and treatment of infections.
    3. Stimulation of hematopoiesis by use of growth factors.
    4. Stem cell transfusions or platelet transfusions (if platelet count is too low).
    5. Psychological support.
    6. Careful observation for erythema (document locations), hair loss, skin injury, mucositis, parotitis, weight loss, or fever.
    7. Confirmation of initial dose estimate using chromosome aberration cytogenetic bioassay when possible. Although resource intensive, this is the best method of dose assessment following acute exposures.
    8. Consultation with experts in radiation accident management.

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Appendix E:
Bites, Stings, and Envenomation

Objectives

By the end of this small group session, learners will:

1. Identify and characterize common bite/envenomation patterns of poisonous fauna found in the United States (US).
2. Describe the management and treatment for common reptile and arthropod envenomation.
3. Understand and perform initial management of these injuries including venom specific antidotes.
4. Describe the presenting symptoms of rabies.
5. Compare the difference in the natural reservoirs for rabies in the US and internationally.
6. Review the post-exposure prophylaxis for rabies exposure.

Case Studies

Case 1: An 8-year-old boy was playing in the grass when he felt a bite on his right thumb. He did not see an animal or an insect and he went inside to tell his mother. Bleeding, swelling, and bruising of the boy's thumb began immediately following the bite. After giving him a dose of diphenhydramine, the boy's mother used a stick to look into the bushes, where she found a snake. A family member killed the snake, and they brought it with them to the hospital for identification. It was later identified as *Crotalus adamanteus* or an Eastern diamondback rattlesnake. The boy is complaining of increasing pain with progressing edema.

Question Prompts:

1. What type of snake is the diamondback (family and genus)? Describe the mechanism of action of their venom and clinical manifestations of their bite.
    a. Diamondbacks are a type of pit viper, a family of venomous snakes found throughout the US. The pit viper family includes genus *Crotalus* (rattlesnakes) and genus *Agkistrodon* (copperheads and cottonmouths). Pit vipers are given this name for a pair of forward-located heat-sensing pits below the nostrils used for prey location, aiming strikes, and metering venom dose based on prey size.[1] Up to 25% of snake bites from pit vipers are dry bites.
    b. The *Elapidae* snakes are the other family indigenous to the US and comprise three different species of coral snake. These snake bites are much less common and only account for 2% of all US snake bites.
    c. Pit viper venom is a cytotoxic and hemolytic toxin but less common neurologic effects such as numbness, tingling, and weakness have been observed. Primarily these toxins bring about coagulation of the blood and clotting of the vasculature. The pain of the wound is severe and is rapidly followed by swelling and discoloration. Ecchymosis and blister

formation can also develop, with necrosis seen in severe envenomation only. The action on the nervous system is not significant. There can occasionally be systemic manifestations with more severe bites. These may include nausea, vomiting, diaphoresis, hypotension, respiratory distress, and cardiovascular collapse. Fortunately, these severe presentations are rare, representing approximately 1% of envenomations.[1]

d. The other type of venomous snake in North America is the Coral snake. Its bites result in little to no local damage. Instead, venom causes neuromuscular blockade that manifests as weakness, ptosis, cranial nerve palsies, dysarthria, and dysphagia. This can progress to full respiratory paralysis and death without treatment.
e. Since the development of anti-venom, bites are rarely fatal. No deaths were reported from 1983 through 2006, and published case reports are in those that did not seek treatment.[4]

2. What is the initial management for reptilian envenomation in the emergency department (ED) setting and what definitive care options exist?
    a. Envenomation care includes: Rapid transport to an ED, stabilization of airway, breathing and circulation (ABCs), antivenom therapy, and intensive care unit admission for those with severe systemic symptoms. Attempts to kill the snake are NOT recommended because this could result in additional bites. Sites of progressing erythema should be marked every fifteen minutes.
    b. Keep in mind that 25% of bites are dry bites, so patients with no symptoms or signs of actual envenomation after 6-8 hours of observation can safely be discharged home. Note this observation period is longer for coral snakes due to delayed manifestations; patients should be observed 24 hours before safe discharge after coral snake exposure.
    c. Local wound care should be provided in similar fashion to other traumatic wounds with cleansing of the area, update of tetanus immunization as indicated, and consideration of antibiotics (but only if there is concern for active infection and not recommended prophylactically).
    d. Lab workup includes: complete blood count (CBC) with manual differential and peripheral blood smear, prothrombin time (PT), activated partial thromboplastin time (PTT), international normalized ratio (INR), fibrinogen and split products, blood type and cross-match, basic metabolic panel (BMP) and a urinalysis for myoglobinuria, arterial blood gas (ABG) and lactate level for patients with systemic symptoms.
    e. Antivenom is a polyvalent immune Fab product called CroFab. Pediatric CroFab dosages are the same as adult dosages because dosage of antivenin reflects the venom load, not a patient's size. Treatment is indicated in any patient with progressive local tissue effects, hematologic venom effects, and systemic signs attributable to venom.[5] It is suggested that administration of CroFab occur within 6 hours of envenomation. The usual initial dose is between 4 and 6 vials. After initial control of the envenomation is established, additional 2 or 4 or 6-vial doses may be administered every 6 hours for up to 18 hours (3 doses) until swelling halts or labs stabilize.[5]
    f. Treatments that are generally **NOT** recommended include: Use of tourniquets, excision of the bite site or fasciotomy, and use of a venom extractor.

g. In confirmed copperhead envenomation, a randomized controlled trial showed that long term outcomes were similar regardless of treatment with the antidote. However, it did show more rapid resolution of symptoms.[6]
h. Please note, these recommendations only apply to these specific snakes found in North America. Bites from snakes that live outside of North America can have different clinical presentations and management.

Case 2: An 8-year-old boy is brought to the ED by his mother after sustaining a spider bite to his left foot. The spider was in a shoe that the boy put on. The patient complains of severe pain that began in his foot and since has progressed to his leg, low back, abdomen, and chest. At presentation, his vital signs are blood pressure 134/92 mmHg, respiratory rate 26/min; heart rate 96/min, temperature 97.8°F and pulse oxygen saturation 96% on room air. He appears to be in distress and is diaphoretic. His abdomen is tight, and he exhibits diffuse guarding. His lungs are clear, and findings on his cardiac examination are normal. There is mild erythema of his left foot, but there are no identifiable puncture marks. While in the ED, the patient begins to complain of difficulty breathing. He is treated with morphine and diazepam, which do not provide relief.

Question Prompts:

1. What type of bite are these symptoms pathognomonic for? What is the term for the toxidrome associated with this envenomation?
    a. This is consistent with a black widow spider bite. Although the most common species in the US are jet black with a red-orange hourglass spot on the ventral abdomen, the genus is usually described as widow spiders because not all species are black. It is found throughout the US except for Alaska. The widow spider is non-aggressive except when disturbed and tends to live in dark, protected areas such as corners of garages, woodpiles, and outhouses. Only females envenomate due to their larger size when compared to males.
    b. Latrodectism is the clinical syndrome following a black widow bite. Typically, the bite is felt as a pinprick sensation and the victim will usually recognize the injury. Onset of symptoms is usually within one hour (range: immediate to 12 hours) and can persist for a prolonged period (average: 22 hours +/- 25 hours, with symptoms persisting longer than 72 hours not unusual), often with a waxing and waning pattern.
    c. Grading of envenomation:
        1. Grade 1 - Mild envenomation: Localized pain at bite, normal vital signs.
        2. Grade 2 - Moderate envenomation: Muscular pain in envenomated extremity, extension of muscular pain to chest or abdomen, local diaphoresis at bite, normal vital signs.
        3. Grade 3 - Severe envenomation: Generalized muscular pain in back, abdomen and chest, diffuse diaphoresis, latrodectus facies (facial muscle spasm, edematous eyelids and lacrimation), abnormal vital signs, nausea and vomiting, headache.
    d. Brown recluse spider bites can cause local tissue damage and necrosis, but rarely can cause systemic symptoms such as malaise, fever, myalgias, nausea, or vomiting.

2. Describe the initial management of these bites in the ED and the expected disposition for this patient.
    a. The traditional therapies for black widow spider envenomation are aimed at providing symptomatic relief while venom effects resolve. These include primarily opioid analgesics and muscle relaxants.
    b. In addition, calcium therapy was once considered to be an antidote for black widow envenomation. Calcium was thought to stabilize nerve membrane permeability, resulting in decreased neurotransmitter release. Although this effect was demonstrated in vitro and reported in some early clinical series, subsequent experience has not shown effectiveness. Therefore, *calcium therapy has lost favor in the medical toxicology community.*[7]
    c. Dantrolene is another medication that has NOT been shown to have clinical efficacy.[7]
    d. The patient will need admission for monitoring of systemic symptoms and supportive care.
    e. Latrodectus or black widow spider antivenom should only be considered for severe cases. The active component of the venom known as alpha-latrotoxin acts as a cation pore inducing release of neurotransmitters from presynaptic neurons.

Case 3: A 9-year-old previously healthy boy complains of pain over the upper third of his left arm, associated with a fever. His pain is associated with numbness and has progressed over the previous three days, radiating to the left wrist, left shoulder and lateral aspect of his neck. The pain is worse on palpation, with active movement and at night, causing insomnia. Mom states that eight weeks ago they stayed at a cottage in a wildlife sanctuary in western Quebec. The patient is admitted for further management. The following day the patient develops severe tremor and myoclonus of the face and all extremities, priapism, drooling, pharyngeal spasm, and a feeling of suffocation. He is intubated, heavily sedated and transferred to a tertiary care facility.

Question Prompts:

1. What is the patient suffering from and what is the natural reservoir or vector for transmission of this disease?
    a. The patient is suffering from rabies.
    b. In the past, human rabies cases in the US usually resulted from a dog bite. Recently, more cases of human rabies have been linked to bats and raccoons. Although dog bites are a common cause of rabies in developing countries, there have been **no reports** of human rabies caused by a domestic dog bite in the US since 1979 due to widespread animal vaccination. There are rare reports of dogs contracting rabies, specifically unvaccinated animals exposed to wildlife. Domestic dogs are no longer considered a reservoir for rabies virus in the United States. However, guidelines still recommend observation of animals after a bite.[8] In other countries without vaccination programs, domestic dogs remain the most common vector of disease transmission.
    c. Rabies is an infectious viral disease that is almost always fatal following the onset of clinical signs.
    d. Rabies affects domestic and wild animals, and is spread to people through bites or scratches, usually via saliva. Very rarely, rabies has been transmitted without an actual bite.

This type of infection is believed to be caused by infected saliva that has gotten into the air, usually in bat caves. The average incubation period is 2-3 months but reported potential range is highly variable from 14 days to 6 years.[9]

e. Small rodents such as squirrels, hamsters, guinea pigs, gerbils, chipmunks, rats, and mice are not known to transmit rabies to humans.

2. What are the clinical symptoms of this disease?
    a. The initial symptoms of rabies are fever and pain or an unusual or unexplained tingling, pricking or burning sensation (paresthesia) at the wound site. Two forms of the disease can follow.
        1. People with **furious rabies** exhibit signs of hyperactivity, excited behavior, hydrophobia (fear of water) and sometimes aerophobia (fear of flying). After a few days, death occurs by cardiorespiratory arrest.
        2. **Paralytic rabies** accounts for about 30% of the total number of human cases. This form of rabies runs a less dramatic and usually longer course than the furious form. The muscles gradually become paralyzed, starting at the site of the bite or scratch. A coma slowly develops, and eventually death occurs. The paralytic form of rabies is often misdiagnosed, contributing to the under-reporting of the disease.
    b. No tests are available to diagnose rabies infection in humans before the onset of clinical disease.
3. Describe the management of rabies exposures.
    a. Post-exposure prophylaxis (PEP) means the treatment of a bite victim that is started immediately after exposure to animal bite that could potentially transmit rabies in order to prevent rabies from entering the central nervous system. This consists of:
        1. Local treatment of the wound, initiated as soon as possible after exposure;
        2. A course of rabies vaccine that meets WHO standards; and
        3. The administration of rabies immunoglobulin (RIG), if indicated.
    b. Effective treatment soon after exposure to rabies can prevent the onset of symptoms and death. It is reported as 100% effective if given early.[10]
    c. **Local treatment of the wound** involves first-aid of the wound that includes immediate and thorough flushing and washing of the wound for a minimum of fifteen minutes with soap and water, detergent, povidone iodine or other substances that kill the rabies virus.
    d. Depending on the severity of the contact with an animal that could harbor rabies, administration of PEP is recommended as follows:

Categories of contact with suspect rabid animal	Post-exposure prophylaxis measures
Category I – Touching or feeding animals, licks on intact skin	None
Category II – Nibbling of uncovered skin, minor scratches or abrasions without bleeding	Immediate vaccination and local treatment of the wound
Category III – Single or multiple transdermal bites or scratches, licks on broken skin; contamination of	Immediate vaccination and administration of rabies

mucous membrane with saliva from licks; or **contacts with bats**.	immunoglobulin; local treatment of the wound

e. All category II and III exposures assessed as carrying a risk of developing rabies require PEP. This risk is increased if:
    1. The biting mammal is a known rabies reservoir or vector species
    2. The animal looks sick or displays an abnormal behavior (healthy animals should be observed for 10 days)
    3. A wound or mucous membrane was contaminated by the animal's saliva
    4. The bite was unprovoked
    5. The animal has not been vaccinated
f. The **post-exposure** schedule for rabies vaccination is four doses, given at the following times:
    1. A person who is exposed and has never been vaccinated against rabies should get **four doses** of rabies vaccine - one dose right away, and additional doses on the 3rd, 7th, and 14th days. They should also get another shot called rabies immune globulin at the same time as the first dose.
    2. Most persons in the US are unvaccinated, but some populations notably veterinarians may have previously been vaccinated for rabies. A person who has been previously vaccinated should get **two doses** of rabies vaccine - one right away and another on the 3rd day. Rabies immune globulin is **not** needed.

Case 4: A 20-year-old female with no significant past medical history presents to the emergency department one hour after she was stung by a flying insect. She believes it was a yellow jacket or a wasp. She has no prior history of bee stings. Currently she is complaining of redness, swelling, itching, and pain at the sting site on her left upper arm. She denies dyspnea, oral/throat swelling, wheezing, abdominal upset, nausea, or dizziness. Her vital signs are as following: heart rate 90/min, respiratory rate 14/minute, blood pressure 136/80mmHg, pulse oximetry 97% on room air, and oral temperature of 98.8°F. Exam revels a non-distressed female with a 6cm diameter area of swelling and erythema on the left upper arm. There is no stinger present. Distal neurovascular exam of the left arm is unremarkable.

Question Prompts:

1. Describe the clinical manifestations of a bee sting.
    a. Bees are part of the venomous order of insects known as *Hymenoptera.* This order includes honeybees, wasps, yellow jackets, hornets, bumblebees and even fire ants. Stinging insects from this order are found on every continent except Antarctica.
    b. Hymenoptera venom is a mixture of toxins, enzymes, and neurotransmitters. One of the major mechanisms of injury related to bee stings is the release of histamine. In a single sting to a non-sensitized victim—such as this one—the symptoms are typically limited to localized wheal and flare reaction preceded by intense pain. Multiple stings can produce more severe and widespread manifestations such as vomiting, diarrhea, hypotension,

generalized edema and hemodynamic instability. The number of stings required for this more severe reaction varies depending on the size of the victim and the type of insect.[11]

c. The most severe response to Hymenoptera stings is related to anaphylactic reaction and can be elicited with even a single envenomation. An estimated 0.4% of the U.S. population shows some degree of clinical allergy to insect venoms, and 40-50 deaths are reported annually.[12] The manifestations are typical of other signs and symptoms seen in an anaphylactic reaction: flushing, pruritus, urticaria, angioedema, nausea, respiratory distress, hypotension and cardiovascular collapse.

2. What does the hospital management of a bee sting include?
    a. The stinger with venom sac should be removed if still present at the sting location. This may be done by scraping the area with a firm edge, such as the edge of a plastic credit card or similar item.
    b. For mild reactions, topical or oral antihistamine administration is effective. Ice administration may also provide comfort if available.
    c. For more severe reactions—including anaphylaxis—the care should begin with basic assessment and resuscitation of the airway, breathing and circulation. If available, use of intramuscular epinephrine should be provided (0.3ml to 0.5ml of 1:1000 aqueous epinephrine). Intravenous fluids may be required for hypotension and shock. Corticosteroids are also of benefit, though their effect is delayed. These patients should also be rapidly moved to a hospital for ongoing and escalating care.
3. Describe 3 different types of marine envenomation and treatments considerations.
    a. Coelenterates, which include jellyfish, anemone, and coral, cause stinging through release of nematocysts upon direct contact. This results in a painful and often pruritic popular eruption lasting hours to days. The treatment consists of nematocyst removal using a razor or sharp edge and nematocyst inactivation using vinegar, ammonia, or saline. Antihistamines may also be used for symptom relief.
    b. Echinoderms, such as sea urchins and starfish, have spines which contain their venom. Manifestations are usually localized to intense pain with erythema and edema, though systemic symptoms of nausea and paresthesias are occasionally seen. After removal of the spine(s), treatment consists of hot water immersion for 30-90 minutes and general wound care.
    c. Venomous fish are also abundant throughout the oceans. Stingrays and scorpionfish are two common groups of venomous marine vertebrates. The heat labile venom is inflicted upon victims with spines that are covered with integument. Local effects include immediate pain and necrosis. Systemic effects include vomiting, muscle cramps, seizures, paralysis, and cardiac dysrhythmias. Treatment is similar to that of the echinoderms with spine removal, hot water immersion, and wound management. Antivenin is available for certain species of stonefish. Delayed effects of these wounds include infection.

Suggested Readings:

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Small Group Evaluation

The moderator demonstrated adequate knowledge of subject.

5) Strongly Agree 4) Agree 3) Slightly Agree 2) Disagree 1) Strongly Disagree

The moderator's facilitation of the conference facilitated my learning.

5) Strongly Agree 4) Agree 3) Slightly Agree 2) Disagree 1) Strongly Disagree

The overall discussion was relevant to the stated topic(s).

5) Strongly Agree 4) Agree 3) Slightly Agree 2) Disagree 1) Strongly Disagree

The faculty/resident's teaching methods (slides, handouts, videos, etc.) were effective.

5) Strongly Agree 4) Agree 3) Slightly Agree 2) Disagree 1) Strongly Disagree

Faculty Facilitator Evaluation

1. **Preparation – was faculty well prepared?**

Needs Improvement Effective Exemplary

2. **Engaged residents - Encouraged discussion and actively participated, demonstrated enthusiasm?**

Needs Improvement Effective Exemplary

Strengths:

Areas for Improvement:

Reviewer Recommendations:

Resident Facilitator Evaluation

1. **Preparation – was the resident facilitator well prepared?**

Needs Improvement Effective Exemplary

2. **Engaged residents – Controlled and led the session and encouraged discussion, active involvement, and demonstrated enthusiasm?**

Needs Improvement Effective Exemplary

Strengths:

Areas for Improvement:

Reviewer Recommendations:

Evaluation of the Teaching materials

1. **Were the objectives appropriate for the topic?**

Needs Improvement Effective Exemplary

2. **Was the amount of material appropriate?**

Too Short Appropriate Too Long

Strengths:

Areas for Improvement:

Reviewer Recommendations:

Small Group Resident Assessment

Session:		
Facilitator (s):		
DATE:		
Small Group 3	Contributes to group discussion	
	BE/ME/EE	Comments
Resident 1		
Resident 2		
Resident 3		
Resident 4		
Resident 5		
Resident 6		
Resident 7		
Resident 8		
Resident 9		
Resident 10		
Resident 11		
Resident 12		

BE—Below Expectations
Minimal discussion during the session
No discussion on the site discussion board
Comments not contributory to discussion or distracting to discussion
Has minimal knowledge of topic as expected of PGY year

ME—Meets Expectations
Contributes to group discussion in a meaningful way
Incorporate textbook/website/podcast reading into discussion
Has knowledge of topic appropriate to level of training

EE—Exceeds Expectations
Contributes to group discussion in a meaningful way
Incorporate literature into discussion
Has advanced knowledge of topic