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EDITOR'S NOTE

Prioritizing Reviewer Training

Peer review plays a critical role in much of the best scientific publishing. The best peer reviewers are both subject matter experts and masters of the rules and rhythms of scientific writing. True subject matter expertise can take years to develop, as can true mastery over scientific writing, but competent reviewers can emerge with more modest capabilities in both areas with appropriate training. It is common, however, for reviewer skills to be learned informally over time rather than through a formal training curriculum.

Journals rely on peer reviewers, but often with little knowledge of the capabilities of reviewers and almost always with no real control over their process. Although the value of peer review and editing ability may be recognized, there is no mandate on when or how the relevant skills should be acquired. Individuals actively engaged in research are more likely to refine their skills as they practice scientific writing, but those with a non-research focus or those who do not lead in scientific writing efforts may never get the grounding that would best serve them in this area.

One of the economical ways to help reviewers to develop their reviewing skills is to share the reviews and comments of all reviewers and editors who look at a given manuscript. The motivated reviewer will look to see if their recommendations matched the editorial decision and then look through the comments of others to try to understand any disagreements and/or to try to refine their insight and evaluation skills.

The sharing of review content can provide substantial learning potential with little extra work for journal editors or administrative staff, but it is a passive effort, relying on reviewers to self-assess their performance and hone their skills. Direct engagement with reviewers is often limited to egregious issues, possibly associated with decisions to remove individuals from reviewer rosters.

Regular, direct engagement with reviewers can amplify the lessons learned from shared reviews. The quality of an individual's contribution can be frankly assessed, both to directly inform and to promote greater attention to the package. Candid comments can reassure or sting, with a common goal of motivating reviewers to strengthen their reviewing efforts.

Wilderness & Environmental Medicine implemented a reviewer-in-training (RIT) program in 2018 with the goal of complementing reviewer training workshops held at society conferences. The program was voluntary, with participants being promised a direct evaluation of their reviews by the editor-in-chief, and sometimes by associate or section editors. Comments were always intended to be concise to minimize the time required to produce them, with a focus on perceived shortcomings that could foreseeably be overcome. RIT reviews follow the standard 2 reviews of a manuscript, ultimately providing more feedback to authors.

Participation in the RIT program has increased steadily, from 24 individuals in 2018 to 77 in 2021. The greatest amount of feedback is usually provided in the first reviews of a manuscript, with subsequent comments variable as commensurate with the review content. In retrospect, a notable deficiency of the initial program was the undefined endpoint for participation. It was expected that individuals would remove themselves as they wished, and there was a vague plan that when an individual's reviews were largely seen to be without important fault (a clearly subjective state), they would be "graduated." These endpoints were not well communicated, though, and the reality was that determination of a call for graduation was rarely made, given the variable nature of every review and a reticence to arbitrarily push anyone out.

The problem with the lack of clarity regarding endpoints came out in 2021 when a participant expressed marked dissatisfaction regarding the value of the feedback. The participant's choice to stay in the program to that point was probably due to an expectation that "graduation" was the only successful way to move forward. This prompted review and revision of the program description, with a new simple guideline stating that participation is voluntary and should only be continued as long as the feedback is thought to be beneficial. The goal is to help reviewers to develop their skill, but there is no passing metric, so control over the RIT designation is in the hands of the participant.

The response to the RIT program has been overwhelmingly positive, allowing individuals to gain

confidence through the extra support. RIT reviews receive closer editorial scrutiny to avoid minefields, and authors may be a bit more forgiving of imperfections in reviewer tone or focus when seeing the trainee designation.

The RIT program will continue, along with scientific writing and reviewing workshops, as an investment in our community of reviewers and authors. Feedback will continue to be concise, with an intent to highlight areas where reviewer focus can enhance the quality of reviews. The goal is to have each review be as clear, informative, and respectful as feasible, to help authors bring out the best in their academic writing. We encourage other journals to consider similar programs, perhaps learning from our experience.

Neal W. Pollock, PhD
Editor-in-Chief

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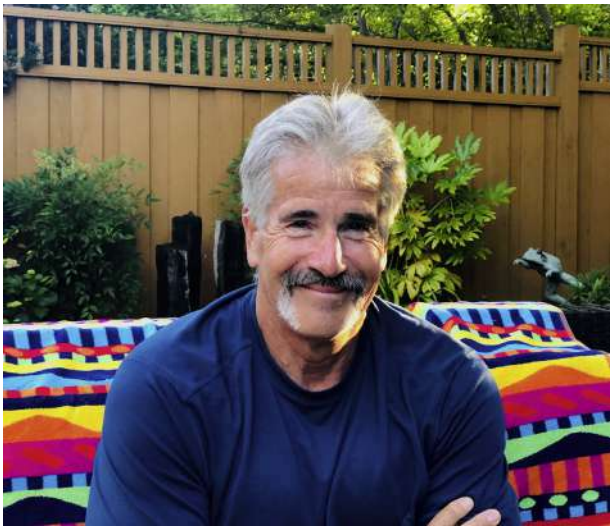
A TRIBUTE

Paul S. Auerbach, MD, MS, FACEP, FAAEM, MFAWM (Hon.)
January 4, 1951–June 23, 2021



Paul S. Auerbach is often called the father of wilderness medicine. Sometimes it annoyed him.

> "I get a lot of credit for wilderness medicine, but that's not fair. I am just the tip of the iceberg. There are so many people doing incredible things out there." (WMS Fireside Chat—Paul Auerbach: Maintaining Joy in Medicine, https://www.youtube.com/watch?v=yze0Tp6rbLU)

Dr Auerbach was born in New Jersey and educated in the eastern United States before making California his family's home and becoming an educator, researcher, writer, speaker, entrepreneur, adventurer, environmentalist, and healthcare leader. His 56-page curriculum vitae demonstrates the breadth of his extraordinary accomplishments, but for his family, friends, colleagues, students, and mentees, he was much more than a list of activities. It was his friendship, support, and endless devotion to making the world a better place that will be his greatest and most enduring gift.

Months before he died, he talked about his mission:

> "Bravery, compassion, and fun—when I look back on my career, and why I became a doctor, those were what was important at the beginning and are the most important at the end." (WMS Fireside Chat—Paul Auerbach: Maintaining Joy in Medicine, https://www.youtube.com/watch?v=yze0Tp6rbLU)

Fun

The motto of the Wilderness Medical Society (WMS) is "Combine your profession with your passion." It is no surprise, then, that Paul helped found the organization; the motto sums up his approach to life. He sought joy in everything he did, combining adventure and curiosity in every project.

> "We need happiness measured in hugs, handshakes, and smiles… a sense of newness and wonder. We need a learning environment. Discovery. Companions and friends. Mates. Loves. Days in the wilderness or at least outside." (WMS Fireside Chat—Paul Auerbach: Maintaining Joy in Medicine, https://www.youtube.com/watch?v=yze0Tp6rbLU)

Dr Peter Hackett described Paul's personality: "He was charismatic, focused, full of positive energy and confidence, unstoppable, and certainly unforgettable. He was a person hard to refuse and easy to love. Sometimes, like an avalanche, it was hard not to be overwhelmed by him. His energy was contagious. If Paul was involved with something, like a bike trip to Bhutan, a trek in the Alps, starting a hospital in Nepal, writing an article or book—whatever it was, if Paul was doing it, you'd want to be part of it."

Dr Auerbach's wilderness medicine accolades were well deserved: In 1983, along with Drs Ken Kizer and Ed Geehr, he founded the WMS and, with Dr Geehr, wrote its first textbook, *Management of Wilderness and Environmental Emergencies* (now *Auerbach's Wilderness Medicine,* in its seventh edition). He was also the founder and first editor of the academic journal *Wilderness & Environmental Medicine*, from 1990 to 1995. In addition, he conducted early wilderness medicine research and, unable to entirely suppress his ironic sense of humor, wrote about farting, officially naming that disconcerting,

gaseous mountaineering condition *high altitude flatus expulsion* (HAFE).

Paul was always looking for another adventure. He never seemed to sit still. "When I would bump into him at meetings, I often had trouble carrying on a long enough conversation because he was off for this bike ride or this great hike in between all of his official responsibilities," said Dr Andrew Luks.

Dr Ed Geehr remembers the young adventurer: "Way back in the 1980s, I took Paul cross-country skiing for the first time. He did fine until the track began to turn, but then he plowed right through the trail, heading for the tree. Just before impact, he tucked his shoulder down and managed a forward flip. His skis catapulted up over his head ('I thought I might be able to take out the tree,' he explained later). One of his skis hung up in the branches of the tree, suspending him upside down, laughing. It took quite a while to disentangle Paul from the tree."

Past WMS president Dr Robert "Brownie" Schoene remembers: "I first met Paul back in the 1980s. That first day, I thought with his big talk and enthusiasm that he was full of hot air. By the end of that meeting week, I realized that he was the real deal. Over the ensuing years, he not only continued to accomplish Herculean tasks with zeal and success, but he was a dedicated friend who never judged. Besides, he was a hell of a lot of fun…"

Even the respected leaders of the WMS were not safe from Paul's play. Dr Luanne Freer, former WMS president, recalled: "Dr Auerbach talked all of the past presidents of the society into publicly posing for a photo wearing silly temporary tattoos. It was Paul's idea to start the 'tradition' of WMS presidents posing together in semi-nude, risqué photos with their personal parts obscured by nature; he then projected these photos in his lectures to audiences of thousands, to the chagrin of those so coerced."

Bravery

That Paul was brave is a given. He did all kinds of things that most people would consider crazy—adventures on land and undersea, rescues (of himself and others), and working in the most stressful environments worldwide. His son, Danny, recalled a childhood incident: "We were driving down the highway going to a Little League game, and we saw a car that was flipped over. Dad pulled over and called a coach to get us. He showed up later at the baseball game, his arm all wrapped up in gauze. He'd crawled through the back window, cutting up his arm to save the guy in the car" ("Paul Auerbach, wilderness-medicine pioneer, dies at 70," https://med.stanford.edu/news/all-news/2021/07/paul-auerbach-wilderness-medicine-pioneer-dies-at-70).

But there is far more to bravery than not fearing injury or death. To be brave is to overcome fear of failure, self-doubt, criticism—to find a purpose and, despite all the passive resistance and active opposition, fight to make it real. That was Paul's approach to life.

> "We're all going to make mistakes. If mistakes render you immobile and disturb you so badly that you can't carry on, you're not going to get very far. You have to spot them, admit them, and then move on." (Michael Killen podcast, Impact of Air Pollution on Human Health: Part 2, August 6, 2020, https://www.youtube.com/watch?v=kOAFJzkDUn4)

His emotional, physical, and moral bravery led him to amazing and diverse accomplishments. Paul graduated magna cum laude from Duke University with a degree in religious studies, received his MD from the Duke medical school in 1977, and completed his emergency medicine training at UCLA in 1980. He immediately began a career in academic emergency medicine while continuing his passion for the outdoors. In 1981, Dr Auerbach joined the emergency medicine faculty at the University of California, San Francisco, School of Medicine. Two years later, still junior faculty, he teamed up to start an entirely new medical specialty.

He left academic medicine for the private sector for a decade, between 1995 and 2004, serving as an executive in major corporations such as Sterling Health, MedAmerica, and KAI Pharmaceuticals, as well as a venture partner for an investment firm in Menlo Park.

Dr Auerbach had an unparalleled career in academic emergency medicine, spent mostly at the Stanford University School of Medicine and ending his career as the Redlich Family Professor Emeritus in the Department of Emergency Medicine. He served in national leadership posts in the American College of Emergency Physicians and the Society of Academic Emergency Medicine. He was a prolific and wildly diverse author—publishing 72 peer-reviewed studies in journals including the *New England Journal of Medicine*, *JAMA, BMJ*, and *Lancet*. He wrote 91 book chapters and almost a dozen books on wilderness medicine, business management, and climate change. He even wrote a novel and, in his spare time, penned popular health pieces for magazines including *Reader's Digest, Field and Stream, Family Circle, Prevention*, and *The Wall Street Journal*.

His reputation as an educator and lecturer was reflected by hundreds of invited presentations worldwide to audiences ranging from laypeople to global leaders, in places as mundane as Menlo Park and Bethesda and as exotic as Kathmandu and Geneva.

Paul consulted for major corporations and worked with the World Health Organization, the World Bank, the Environmental Protection Agency, the Department of Defense, USAID, the National Football League, the National Ski Patrol, and Divers Alert Network. He was a member of the Explorers Club and the Council on Foreign Relations.

Compassion

> "You've got to be compassionate. Always." (Remembering Paul Auerbach, MD, https://www.youtube.com/watch?v=8Iam3DWLKBU)

Paul often spoke of the importance of compassion—not in a sentimental or ironic way, but with a straightforward simplicity of purpose. He helped on a large scale: saving hundreds after a disaster, creating a completely new specialty that could save thousands, and fighting for environmental awareness. But it was really the little things he did that stand out—he served as the team physician for Stanford wrestling, coached his kids' teams, and supported and mentored hundreds of students and young faculty.

> "Anybody that needs my help on anything… I'm delighted to be able to try to help… I want to help. I've always wanted to help. …when you come to this juncture in life, it will be looking back on the people that you helped—that is what is going to give you some peace." (WMS Fireside Chat—Paul Auerbach: Maintaining Joy in Medicine, https://www.youtube.com/watch?v=yze0Tp6rbLU)

Paul was the consummate listener, and when he recognized authentic passion for a project, he opened doors for his mentees to help put them in the right place at the right time. Smiling like a proud father, he gently pushed (sometimes more of a jarring shove) a hesitant young colleague forward and watched them thrive. Paul once encouraged a new author to revise a manuscript for the journal that had been rejected by other reviewers: "Dr Auerbach strongly wished young researchers like me working in far-flung places like Nepal would write more and seek publication. Following his sage advice, that is exactly what I have been doing (and encouraging others to do) since that time many years ago in 1992," says Dr Buddha Basnyat. Receiving a push from Paul was a real vote of confidence in one's abilities. There are hundreds of us whose careers benefited from Paul's selfless support.

But even Paul acknowledged that he was sometimes too passionate about a project, a bit of a hothead, and he could be very demanding of those he mentored: "He did not suffer fools and woe be the person who did not produce or take as strong of a position to advance an organization or support a patient as Paul felt was needed," said his lifelong friend and past WMS president, Dr Howard Backer.

Dr Auerbach loved his patients and loved practicing medicine, even when he was not at work: "Paul was the world's doctor. He would take the time to help any friend or acquaintance in need. No matter where we were, on the ski slope, at dinner, or on an international trip, Paul would receive calls and provide consultation or help with anyone or their family who had a medical problem," said Dr Backer.

Paul's desire to give back did not end with his mentorships and personal friendships but extended to those he had yet to meet. His focus was always on helping, and his work eventually turned to the world.

> "It's just a question of how you can do the greatest amount of good for the greatest number of people. Cause the least amount of harm and just keep working that theme over and over again and just stayed centered…" (Michael Killen podcast, Impact of Air Pollution on Human Health: Part 2, August 6, 2020, https://www.youtube.com/watch?v=kOAFJzkDUn4)

The 2010 Haitian earthquake changed his life. Recognizing the similarities between wilderness and disaster medicine, Dr Auerbach became a disaster responder for catastrophic earthquakes in Haiti and Nepal, where he shared his gifts for organization, inspiration, and leadership to galvanize teams of healthcare workers to help save lives.

Immediately after the 2010 earthquake, Dr Robert Norris, the chair of Stanford Emergency Medicine, asked Paul if he could lead a team to provide medical relief. "We have to go," he responded, and left within days. With a small group of Stanford providers, he plunged into the horror and chaos of a shattered city with hundreds of thousands of dead and injured with essentially no one to care for them.

> "I didn't train to do it; it just came naturally. For some reason, I've always been able to step back, and then when things get really chaotic and scary for other people, from a medical perspective, I generally get calmer." (Remembering Paul Auerbach, MD, https://www.youtube.com/watch?v=8Iam3DWLKBU)

Paul almost worked himself to death. "When we arrived two weeks later, I found him sprawled barely conscious on a pile of broken boxes with an IV in each arm in the courtyard of a crumbled hospital," said Dr Tom Kirsch. "He propped open an eye, smiled wanly, and said 'good luck.'" They had struggled in the

oppressive heat, day and night, for two weeks with few supplies, among piles of bodies, trying to save as many lives as possible. Paul was at the center, coordinating, cajoling, encouraging. The experience almost killed him.

"When Paul returned from Haiti, he was definitely changed by the experience. Like combat, disasters have a way of changing people, some for the better, some for the worse. After he returned, some of the rough edges had been smoothed off. He was more introspective, more compassionate, more caring and more committed," reported his lifelong friend and past WMS president, Dr Mel Otten.

As part of the International Medical Corps team, Paul returned to disaster response after the 2015 Nepal earthquake. Nepal was already a special place for Paul. He had led expeditions to the Himalayas and already understood the health struggles of the Nepali people. Responding to the earthquake was the start. Then, with Nepali physician Dr Rajesh Gongal, he helped develop and raise funds for the first ambulance service in Kathmandu (http://nepalambulanceservice.org). And when his friend Wongchhu Sherpa expressed a dying wish to bring healthcare to his remote village, Paul joined with WMS colleagues Luanne Freer and Eric Johnson to fund and construct the Wongchhu Sherpa Memorial Hospital in Solukhumbu, Nepal (https://wms.org/donation/wongchhu/).

His focus expanded during the last few years: He worked with the National Football League to design and build safer football helmets, and he became an outspoken communicator about the potentially devastating impact of climate change, writing a book, *Enviromedics: The Impact of Climate Change on Human Health*, with WMS past president Dr Jay Lemery. He was working on a project to create an organization to prepare US communities for climate disasters until just weeks before his death.

Paul died peacefully, surrounded by family, on June 23, 2021. He was 70. He is survived by his wife, Sherry, and children Brian, Danny, and Lauren.

Paul embodied bravery, compassion, and fun each day of his life. He was a force of nature, a positive power, a grinning, slightly goofy, scarily intense wilderness physician who made us all better people—always looking beyond himself to his family, his friends, the world. And in that Paul changed the world—from US national parks to Nepal, he made it a safer place for anyone who decides to step off the path and embrace the joy of nature, for the sick and dying in US emergency departments, for those suffering in the aftermath of devastating disasters. But what he really changed were people's hearts. And minds. And purpose. He made us all just slightly better people than we could ever otherwise be—ever optimistic, always looking out beyond himself to the world. Seeking to make things better. Seeking the best.

Fellow Stanford Emergency Medicine faculty member Dr Grant S. Lipman described his impact: "One of the major life lessons Paul taught me was… that through a unique world vision, and belief in our own construct and purpose, we could create something novel and wonderful and magical, and will it into existence. I saw this through Paul's words and actions too many times to count. His ability to grow, redefine, and create something new and poignant was amazing to behold."

We must continue to advance his great loves of wilderness medicine, emergency medicine, and protecting the environment. But more important are the little things, the patients, the students, junior faculty, those who need healthcare even in the most remote parts of the world. We must follow Paul's example and continue to nurture new generations who also want to make the world just a little better. In that perhaps we, too, can find the same joyous, unselfish love of a life well lived. That is all that we can ask for.

Consider honoring Dr Auerbach by making a donation to the Wongchhu Sherpa Memorial Hospital or the WMS Auerbach lectureship (see below).

Thomas D. Kirsch, MD, MPH
Bethesda, MD, USA

Luanne Freer, MD, FAWM
Anacortes, WA, USA

Charitable opportunities

Wongchhu Sherpa Memorial Hospital: https://wms.org/donation/wongchhu/
WMS Auerbach lectureship: https://wms.org/auerbachlectureship/

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ORIGINAL RESEARCH

Impact of a Severe Rocky Mountain Spotted Fever Case on Treatment Practices at an Academic Institution Within a Nonendemic Area

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Introduction—Rocky Mountain spotted fever (RMSF) is a bacterial disease associated with morbidity and mortality when untreated. The primary study objectives are to describe clinician diagnostic and treatment practices in a nonendemic area after the occurrence of an unrecognized severe pediatric presumed RMSF case (index case). We hypothesized that inpatient diagnostic testing frequency and initiation of empiric treatment will increase after the index case.

Methods—We performed a retrospective chart review of patients aged less than 18 y evaluated for RMSF at Penn State Hershey Children's Hospital between 2010 and 2019. We divided the study population into 2 groups (preindex and postindex) and evaluated patient characteristics, RMSF testing completion, and timing of doxycycline administration.

Results—Fifty-four patients (14 [26%] preindex and 40 [74%] postindex) were included. Age (median [25th percentile, 75th percentile]) decreased from 14.5 y (8.6, 16) preindex to 8.3 y (3.6, 14) postindex. Twelve (86%) preindex and 31 (78%) postindex patients received empiric doxycycline (P=0.70). Four years after the index case, a decrease in empiric and urgent initiation of doxycycline administration was noted. One case of severe RMSF was diagnosed 4 y after the index case.

Conclusions—Our study found that inpatient RMSF testing increased after the index case, but not all patients received empiric treatment. This may represent an underappreciation of RMSF severity even after a recent devastating case. We suggest that when severe rare but possibly reversible diseases, such as RMSF occur, all clinicians are educated on the diagnostic and treatment approach to reduce the morbidity and mortality risk.

Keywords: Rickettsia rickettsii, pediatrics, United States, tickborne illness

Introduction

Rocky Mountain spotted fever (RMSF) is a severe tickborne illness caused by the bacteria *Rickettsia rickettsii.*[1] The common symptoms of this disease include fever, headache, myalgia, and various types of rashes (maculopapular to petechial).[2] These nonspecific symptoms, along with a rash that may not be present in all cases, can result in underrecognition.[1]

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When RMSF is not promptly recognized and treated, it can result in multiorgan system failure, including encephalopathy, acute respiratory failure due to acute respiratory distress syndrome, and septic shock.[2-6] Multiple studies have demonstrated that if doxycycline administration does not occur within 5 d of onset of symptoms, patients are at high risk of these complications, including death.[2,7] The standard of care is to initiate therapy early, even if an alternative diagnosis is more likely or the classic findings of RMSF are absent.[2,8] Thus, when a pediatrician works in an RMSF endemic area, a high index of suspicion is required; when suspected, testing and early treatment are necessary.

RMSF is commonly diagnosed in areas of the southeastern and southcentral United States, where climate and

host availability are favorable to the various tick vectors that harbor *Rickettsia.*[4,9] In recent years, however, there are concerns that these environmental risk factors may change due to climate change.[10] This has resulted in an increase in tick bites for not only RMSF but other tick-borne illnesses as well.[10,11] If climate change continues, the frequency of tick bites will likely continue to rise in endemic areas, and ticks may migrate northward, placing nonendemic areas at risk.[10,12]

If a severe case of RMSF occurs in a nonendemic area and is unrecognized, its individual emotional impact can be profound, especially if it results in patient harm.[13] This can include modifications in diagnostic approaches that are less risky.[13] It is unknown, however, how noninvolved clinicians react to similar cases and whether group knowledge of the event improves the care of future patients.[14] Understanding how clinicians react to these rare cases can influence the way a region prepares healthcare providers for the possibility of RMSF (and other rare diseases) and may potentially improve the quality of care of patients who present with signs and symptoms of suspected RMSF.

In 2014, a pediatric patient (referred herein as the index case) presented to our institution's pediatric intensive care unit with severe presumed RMSF. Despite aggressive therapy, the patient died of this illness. The primary objectives of the present study are to examine the diagnostic and treatment practices in a nonendemic area before and after a severe pediatric case of RMSF. We hypothesize that inpatient diagnostic testing frequency and initiation of empiric treatment will increase after the occurrence of a severe case of presumed RMSF.

Methods

This was a retrospective study of pediatric patients seen in the emergency department or admitted to an inpatient service in Penn State Hershey Children's Hospital who were tested for RMSF. Penn State Hershey Children's Hospital is a tertiary care facility located in the central Pennsylvania area. Using the Cerner hospital database, we identified and included the following patients: 1) pediatric patients aged <18 y; 2) patients serologically tested for RMSF via indirect immunofluorescence (our primary mode of diagnostic testing); and 3) patients admitted to the Penn State Hershey Children's Hospital or seen in our emergency department. After we obtained this list of patients, we used our electronic health record to perform a chart review of each case. Data collected included demographics (age, sex, race), timing of RMSF testing, presence of RMSF diagnosis, timing of doxycycline administration (with appropriate administration considered to be within 3 h of suspicion of RMSF), patient outcome, length of stay, primary clinical setting during evaluation (inpatient or emergency department), and identification, timing, and description of a severe case of confirmed RMSF.[15] Retrospective review of the patients' medical records was completed between January 1, 2011 and December 31, 2019.

We divided the study population into 2 groups: preindex and postindex. Preindex was defined as cases in which patients were evaluated for RMSF before and including August 15, 2014, the day on which a pediatric patient with a severe case of presumed RMSF died. Postindex was defined as the cases after, but not including, this event.

The study was reviewed by the Penn State College of Medicine institutional review board and determined to be exempt from institutional review board oversight (STUDY00013124). Summary statistics using median, interquartile range, or proportions were reported for clinical and demographic characteristics of the preindex and postindex patients who were tested for RMSF. Fisher's exact test was applied to compare the proportions before and after the index case. Wilcoxon rank sum test with continuity correction was applied to compare the annual incidences between preindex and postindex periods. Analysis was performed using R v4.0.0. P values <0.05 were regarded as statistically significant.

Results

During the study period, an average of 8829±2138 pediatric patients (age 0−18 y) were seen in the emergency department per year. A total of 54 patients were included in this study. The age (median [25th percentile, 75th percentile]) of patients evaluated was 14.5 (8.6, 16) y preindex and 8.3 (3.6, 14) y postindex. Seven preindex patients (50%) were male, and 27 (68%) postindex were male. Other patient characteristics were similar (Table 1).

The index case patient was a 3-y-old female who presented to an outside hospital with nonspecific upper respiratory infection symptoms, rash, and encephalopathy during the summer of 2014. There was no history of travel, but there was a history of a dog tick bite. The patient was transferred to our institution's pediatric intensive care unit, where she was empirically started on broad-spectrum antibiotics including doxycycline within 95 min. The patient developed progressive encephalopathy, acute respiratory failure secondary to acute respiratory distress syndrome, and profound shock. RMSF testing detected no significant level of *Rickettsia rickettsii* IgG antibody. Based on the findings of fever and generalized rash that began on the wrists and ankles and

Table 1. Demographic characteristics

Patient characteristics	*Preindex case (including index case)*	*Postindex case*
Age, median (25th, 75th percentile), y	14.5 (8.6, 16)	8.3 (3.6, 14)
Race, n (%)		
White	12 (86%)	27 (68%)
Asian	1 (7%)	1 (3%)
African American	1 (7%)	3 (8%)
Other	0 (0%)	9 (23%)
Sex, n (%)		
Male	7 (50%)	27 (68%)
Female	7 (50%)	13 (33%)
Length of stay, median (25th, 75th percentile), d	4.2 (1.7, 12)	5.0 (2.8, 9.6)
Outcome, n (%)		
Survived	13 (93%)	40 (100%)
Died	1 (7%)	0 (0%)
RMSF diagnosed, n (%)	1 (7%)	1 (3%)
Died	1 (100%)	0 (0%)
Location, n (%)		
Inpatient	12 (86%)	36 (90%)
Emergency	2 (14%)	4 (10%)

then spread centrally to involve the legs, buttocks, trunk, and face (with later stages becoming petechial), as well as pediatric infectious diseases consultation, the patient's condition was determined to be consistent with RMSF. After 3 d in the hospital, the patient died of presumed RMSF. On review of the electronic health record, the patient was evaluated 4 d before presentation for a diffuse rash on her feet and fever, diagnosed as a viral exanthem. This was the only pediatric patient with (presumed) RMSF to our knowledge who died during the study period.

Fourteen patients were evaluated for RMSF before the index case. More patients (n=40) were evaluated annually for RMSF after the index case ($P<0.05$). Twelve (86%) preindex patients received empiric doxycycline during testing, compared to 31 (78%) postindex patients, but this was not statistically significant ($P=0.70$). Seven (50%) preindex patients were administered doxycycline within 3 h, compared to 21 (53%) postindex patients, but this was also not statistically significant ($P=1.0$) (Table 2).

Three preindex children who were tested for RMSF were <8 y of age, and all (100%) received empiric doxycycline treatment. Twenty postindex children were <8 y of age, and 16 (80%) received empiric doxycycline treatment ($P=1.0$).

Four years after the index case, in 2019, a decrease in empiric and urgent initiation of doxycycline administration was noted in patients tested for RMSF (Figure 1; Table 3). One case of severe RMSF was diagnosed 4 y after the index case; doxycycline was administered within 3 h after RMSF testing was performed, and the patient survived.

Discussion

This study found that after a severe case of RMSF in an area currently considered nonendemic, diagnostic testing for RMSF appropriately increased. Not all patients after this case, however, received empiric treatment during RMSF testing. In cases in which empiric treatment was initiated, there was no difference in the frequency of doxycycline administration within the 3-h time window between preindex and postindex patients. Our findings may represent an underappreciation of RMSF severity and may have implications for the approach to diagnostic and treatment practices in nonendemic RMSF areas.

RMSF is a vector-borne illness.[2] Therefore, it depends on the presence of ticks and a favorable environment that allows them to thrive.[16] Warmer weather allows the tick to reproduce and facilitates the presence of human hosts.[16] This may contribute to tick bites and the spread of RMSF, even if it is only present in a small population of ticks.[10,11] As temperatures rise across the United States, including Pennsylvania, institutions such as ours need to be prepared to recognize the potential presence of this severe illness, diagnose it, and initiate early treatment to avoid the severe consequences of the disease.[17]

To avoid the morbidity and mortality associated with RMSF, empiric treatment with doxycycline is recommended.[2,7] In pediatric patients, doxycycline is

Table 2. Inpatient Rocky Mountain spotted fever diagnostic and empiric treatment practices

	Preindex case[a]	*Postindex case*	*P value*
RMSF testing, n	14	40	0.04[b]
Empiric doxycycline administration, n (%)	12 (86%)	31 (78%)	0.70
Empiric doxycycline administration within 6 h in suspected RMSF cases, n (%)	10 (71%)	28 (70%)	1.00
Empiric doxycycline administration within 3 h in suspected RMSF cases, n (%)	7 (50%)	21 (53%)	1.00

[a]Includes index case.
[b]*P* value <0.05, based on 2-sided Wilcoxon rank sum test with continuity correction comparing the annual incidences in preindex and postindex periods.

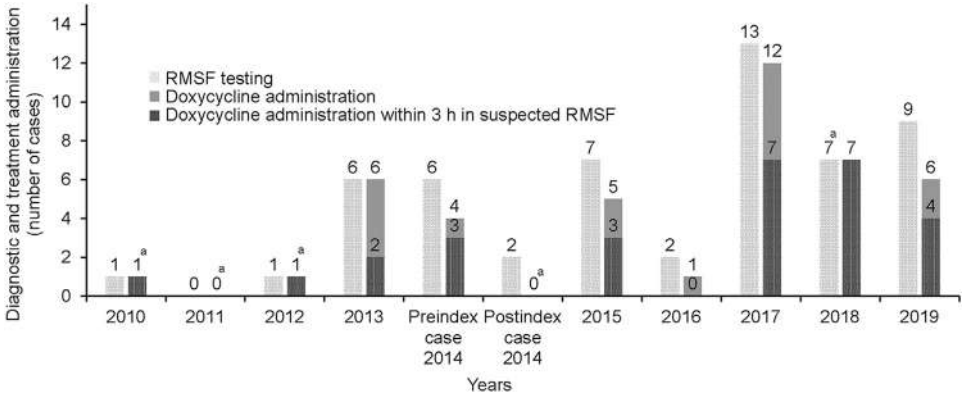

Figure 1. Count of testing and frequency of doxycycline administration (total and within 3 h) in patients with suspected Rocky Mountain spotted fever. [a]Counts of total doxycycline administration and administration within 3 h were equal.

associated with adverse effects, particularly dental staining.[18] Recent American Academy of Pediatrics (AAP) guidelines, however, strongly indicate that the risks of this outweigh the benefits of treatment.[18] Thus, in cases inw hich RMSF is suspected, the patient should be tested, started on doxycycline within 5 d of symptom onset, and should continue treatment until the results return.

Our study found that despite AAP recommendations, not all patients received empiric treatment, including patients <8 y of age after the index case. This is consistent with previous studies examining RMSF therapeutic practices. Although a majority of providers identified doxycycline as the appropriate antimicrobial through a survey, only 35% chose this antibiotic in children <8 y of age.[19] In Tennessee, an endemic area, it was reported that a high proportion of survey participants were unaware that doxycycline is the treatment of choice for children <8 y of age.[20] Another study reported that treatment delay may be due to a likely decreased awareness of RMSF and reluctance to use doxycycline in children.[21] Our study confirms that this provider-based issue continues to occur. In addition, this study highlights that education efforts are needed to enhance provider awareness that the clinical decision-making process could contribute to a rare but preventable cause of pediatric mortality.

This present study set a time point of 3 h after doxycycline administration as appropriate empiric treatment. This may be considered overly conservative, especially because patients with RMSF have a time window of 5 d.[2] However, when this time point was expanded to 6 h (to evaluate whether any clinicians recognized the possibility of RMSF at a later point), there were still patients who did not receive empiric treatment. In endemic areas, owing to the disease's nonspecific symptomology, treatment is often initiated even if an alternative diagnosis is more likely.[2] In our area, however, RMSF is nonendemic; thus, it may not be considered as a high-priority diagnosis. Nevertheless, because of the severity of

Table 3. Inpatient Rocky Mountain spotted fever diagnostic and empiric treatment practices divided by year

	RMSF testing (n)	*Doxycycline administration in suspected RMSF (n, %)*	*Doxycycline administration within 6 h in suspected RMSF (n, %)*	*Doxycycline administration within 3 h in suspected RMSF (n, %)*
2010	1	1 (100%)	1 (100%)	1 (100%)
2011	0	0 (0%)	0 (0%)	0 (0%)
2012	1	1 (100%)	1 (100%)	1 (100%)
2013	6	6 (100%)	2 (33%)	2 (33%)
Preindex case 2014[a]	6	4 (67%)	4 (67%)	3 (50%)
Postindex case 2014	2	0 (0%)	0 (0%)	0 (0%)
2015	7	5 (71%)	5 (71%)	3 (43%)
2016	2	1 (50%)	1 (50%)	0 (0%)
2017	13	12 (92%)	12 (92%)	7 (54%)
2018	7	7 (100%)	7 (100%)	7 (100%)
2019	9	6 (67%)	5 (56%)	4 (44%)

[a]Includes index case.

RMSF, we argue that if a clinician considers this a possible diagnosis, there likely was a delay in that consideration; thus, this conservative approach should be taken to avoid potential complications. In addition, despite studies showing that treatment for RMSF should be initiated within 5 d of onset of symptoms, initiation of treatment as soon as possible when this disease is suspected is supported.[2]

This study is unique in that we report how our practices changed after the RMSF index case. Because our institution is in an academic center, this case did undergo routine morbidity and mortality conference review focusing on inpatient care (not outpatient care); furthermore, due to the rarity of this diagnosis, it was presented in resident academic conferences. The index case provided a real-life example of the lethality of RMSF, the ease with which the diagnosis was overlooked, and how it should appropriately have resulted in a change in practice (even among noninvolved clinicians). The only significant practice change that was noted, however, was an increase in testing (with only 1 y in which all patients tested received empiric treatment). RMSF immunofluorescence assay (IFA) testing costs approximately $27 USD.[22] At our institution, however, the test is analyzed externally, and the turnaround time can be 2 to 3 d.[23] Thus, although the index of suspicion appropriately increased, definitive action to prevent harm was not undertaken because results are not immediately available and diagnostic testing depends on the patient's antibody response (a negative test does not necessarily mean the patient does not have RMSF).[7]

Various factors likely contributed to the approach our clinicians took to patients with suspected RMSF. First, the AAP guidelines for doxycycline administration in suspected RMSF are relatively recent; thus, not all clinicians may be aware of this new recommendation, especially because clinicians are taught early in their training to avoid doxycycline use in children <8 y of age. Because RMSF is rare in our region, even if clinicians do consider RMSF, it may be considered unlikely, thus decreasing the priority of the timeliness of doxycycline administration. Clinicians may have collected a thorough history and determined that although the patient has symptoms similar to RMSF, the risk factors are likely lower (ie, the patient did not have any recent travel history or tick bites or did not frequent any wooded regions). Finally, most of these children presented in a nonacute fashion (ie, were not in shock, respiratory failure, or encephalopathic, like our index case). Thus, the relatively healthy condition of the patient affected how the clinician ordered treatment (urgently versus not). Based on our study findings, known microbiology testing limitations, and the potential for patient harm in unrecognized cases, we recommend that empiric doxycycline should be administered if a provider suspects or tests for RMSF.

It is unclear why there was only 1 y (2018) in which all patients tested for RMSF received empiric treatment, with a potential decreasing trend in RMSF testing the year thereafter. Possibilities include faculty and resident turnover; the clinicians involved in the case may have no longer been present within the institution (thereby resulting in a collective decrease in the index of suspicion). The personal impact of the event may have diminished with the passage of time. Less experienced clinicians may be present. Finally, this case may no longer be discussed within the residency education curriculum.

There were various limitations in this study. This was a single-center retrospective study with a small sample size. Due to limitations in data retrieval from our electronic health record, variation in provider documentation practices, and potential for omitted data, we focused only on patients who were tested for RMSF rather than those who should have been considered. Thus, we did not review patients who may have been empirically treated for RMSF without testing or who may have had symptoms possibly associated with RMSF. We did not determine whether sepsis-associated organ dysfunction was present. The index case was seen initially in the outpatient setting. Therefore, it is unknown whether a peer review discussion took place after this case or if there was an impact on diagnostic or treatment practices in the outpatient setting. Finally, it is unknown whether the practice changes we have described were truly in response to the index case or to the rise in RMSF cases noted nationwide.

Conclusions

Our study found that inpatient testing for RMSF increased after the index case, but not all patients received empiric treatment. This may represent an underappreciation of RMSF severity in light of a devastating case that should have triggered a change in practice, even among noninvolved clinicians. We suggest that when severe, rare, but possibly treatable diseases such as RMSF occur, all clinicians should be educated on the diagnostic and treatment approach to reduce the risk of morbidity and mortality.

Author Contributions: Study concept and design (CK, GDC, NJT); acquisition of the data (CK); analysis of the data (CK, SZ); drafting of the manuscript (CK); critical revision of the manuscript (GDC, SZ, NJT); approval of the final manuscript (all authors).

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ORIGINAL RESEARCH

The Impact of Extreme Heat Events on Emergency Departments in Canadian Hospitals

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Introduction—Mean daily temperatures in Canada rose 1.7°C between 1948 and 2016, and the frequency, severity, and duration of extreme heat events has increased. These events can exacerbate underlying health conditions, bringing patients to emergency departments (EDs). This retrospective analysis assessed the impact of temperature and humidex on ED volume and length of stay (LOS).

Methods—LOS is an indicator of ED overcrowding and system performance. Using daily maximum temperatures and humidex values, this study investigated the impact of mean 3-d temperatures and humidex preceding ED presentation on the median and maximum ED LOS and patient volume in 2 community hospitals in Montreal, Quebec, during the summer months of 2016 to 2018. Data were analyzed with 1-way analysis of variance with post hoc Fisher least significant difference tests and Spearman correlation tests.

Results—The mean maximum temperature and humidex were 26.1°C and 30.4°C, respectively (n=276 d). Mean 3-d temperatures ≥30°C were associated with higher daily ED volumes in both hospitals (138 vs 121, P=0.002 and 132 vs 125, P=0.03) and with increased median LOS at 1 hospital (8.9 vs 7.6 h, P=0.03). Mean 3-d humidex ≥35 was associated with higher daily ED volumes at both hospitals as well (136 vs 123, P=0.01 and 133 vs 125, P=0.009) with an increased median LOS at 1 hospital (8.6 vs 6.9 h, P=0.0001) with humidex values of 25 to 29.9°C.

Conclusions—Heat events were associated with increased ED presentations and LOS. This study suggests that a warming climate can impede emergency service provision by increasing the demand for and delaying timely care.

Keywords: climate change, environment, emergency medical services, overcrowding, hospital administration

Introduction

The mean daily temperature in Canada increased 1.7°C from 1948 to 2016, and summer temperatures in Quebec rose 1.5°C during the same period.[1] This may be related to the growing frequency, duration, and intensity of extreme heat events in Canada, which are projected to increase in the coming years.[2-4] There are numerous local and international examples of the impact extreme heat events can have on population health, including the 485 heat-related deaths during the 1995 Chicago heat wave, the 2003 European heat wave leading to an estimated 40,000 deaths, and heat events in Quebec linked to 106 deaths in 2010 and 66 deaths during the 2018 Montreal heat wave.[5,6]

Higher temperatures have been shown to exacerbate many health conditions.[7,8] Patients taking certain medications (eg, diuretics, anticholinergics, beta blockers, and antipsychotics) that interfere with core body temperature regulation, increase heat production, or increase fluid loss are predisposed to heat-related morbidity and mortality.[9,10] During heat events, patients with a wide variety of comorbidities, especially cardiovascular, pulmonary, renal, and psychiatric conditions, are more likely to seek medical care and be hospitalized.[11-15] Extreme heat events also exacerbate the existing effects of social determinants of health, influencing individuals who

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have poor mobility, live alone, or experience unstable housing; these populations are particularly vulnerable to the health sequelae of extreme heat.[16] There is building local and international evidence that heat waves lead to excess death and increased burden on health care system capacity.[17-19]

There is significant impetus to reorient healthcare systems so that they can be climate-resilient and meet the expected increasing need for health services as our climate continues to warm and extreme heat events become more common. However, there are limited Canadian data exploring the effect of heat events on emergency department (ED) performance. Thus, the authors sought to determine the impact of heat events on ED volume and length of stay (LOS), a marker of quality of care, system capacity, and hospital performance,[20] in 2 community hospitals in Montreal, Quebec.

Methods

This was a retrospective cohort study comparing historical weather data measured at the Montreal/Pierre Elliot Trudeau International Airport weather station, Quebec and ED performance metrics from 2 community hospitals, Notre Dame Hospital (NDH) and Verdun Hospital (VH), in Montreal. These hospitals had 26 to 35 emergency department beds, served between 45,000 and 50,000 patients annually, and were supported by acute care surgery and critical care services. The hospitals were both 10 to 11 km from the weather station. Daily temperatures, humidex, LOS, and daily ED patient volume were collected for the summer months of June 21 to September 20 from 2016 to 2018. This date range was selected to minimize the potentially confounding influence of extreme cold weather events and the seasonal patterns of spread of influenza that can increase ED volumes. Owing to its retrospective nature and exploration of a deidentified dataset that cannot be linked to specific patients and providers, this study was granted an exemption from the CIUSSS du Centre-Sud-de-L'ile de Montreal research ethics and compliance institutional review board.

Using daily maximum temperatures and humidex values, the authors calculated the mean temperature and humidex from the 3 d before patients' presentation to hospital. The authors sought to determine whether higher 3-d mean temperatures influenced volumes and median and maximum LOS of patients in both the ambulatory and stretcher ED sections of NDH and VH. Humidex is a measurement of how hot the air feels based on temperature combined with humidity. Heat is more harmful when the relative humidity is high because less sweat evaporates from the skin, hindering one of the body's ways of cooling itself. The authors included humidex values because they more accurately represent how uncomfortable a patient is feeling and the true physiologic effects of heat. The 3-d average of temperature and humidex was selected in accordance with previous studies demonstrating its validity in modeling heat wave intensity and predicting excess heat-related morbidity and mortality.[21,22] The authors used 3 a priori identified mean 3-d temperature thresholds (25−27.4°C, 27.5−29.9°C, and ≥30°C) and 3 a priori identified mean 3-d humidex thresholds (25−29.9, 30−34.9, and ≥35). No days were represented twice within these thresholds.

Data were analyzed with 1-way analysis of variance for measures of ED performance (daily median and maximum LOS and ED patient volumes) with either mean 3-d temperatures or humidex preceding ED presentation as the factor. NDH and VH data were analyzed separately. Main effects identified by 1-way analysis of variance were further analyzed with post hoc means analyses with Fisher least significant difference tests of the a priori determined thresholds of mean temperatures and humidex preceding ED presentation. Spearman correlation testing was used to assess the effect of temperature and humidex on ED volumes and LOS in the stretcher and ambulatory sections of both hospitals. Statistical analyses were conducted in R-Studio (Boston, MA) and Prism 8.0 for Macintosh (GraphPad Software, Inc). All data are presented as mean±SD. Statistical significance was set at $P<0.05$.

Results

The mean daily maximum temperatures for the 2016, 2017, and 2018 summers (n=276 days) for the city of Montreal were 26.7°C, 24.2°C, and 27.3°C, respectively. The mean daily maximum humidex values were 30.9, 28.0, and 32.2. The days with mean 3-d temperatures and humidex values above the a priori thresholds are presented in Table 1.

Mean 3-d temperatures ≥30°C were associated with higher daily ED volumes in both hospitals (138 vs 121, $P=0.002$ at NDH and 132 vs 125, $P=0.03$ at VH) (Figure 1). There was an increased median LOS at NDH with elevated mean 3-d temperatures between 27.5 and 29.9°C (8.3 vs 7.6 h, $P=0.02$) and ≥30°C (8.9 vs 7.6 h, $P=0.03$).

Mean 3-d humidex ≥35 was associated with higher daily ED volumes at both hospitals as well (136 vs 123, $P=0.01$ at NDH and 133 vs 125, $P=0.009$ at VH)

Table 1. Number of days with temperatures and humidex above a priori established thresholds

3-d mean temperature (°C) before presentation to the emergency department	*Days (n) within each temperature threshold*	*3-d mean humidex (°C) before presentation to the emergency department*	*Days (n) within each humidex threshold*
<25	89	<25	44
25–27.4	90	25–29.9	71
27.5–29.9	74	30–34.9	123
≥30	23	≥35	38
Total	276	Total	276

(Figure 2). There was an increased median LOS at one hospital with an elevated mean 3-d humidex of 25 to 29.9 (8.6 vs 6.9 h, P=0.0001), 30 to 34.5 (8.3 vs 6.9 h, P=0.0001), and ≥35 (8.5 vs 6.9 h, P=0.0001). There was also an increased maximum LOS at one hospital with an elevated mean 3-d humidex of 25 to 29.9 (48 vs 31 h, P=0.03) and ≥35 (46 vs 31 h, P=0.02).

Higher mean 3-d temperatures were found to be positively associated with ambulatory ED visits at NDH (P=0.017) and stretcher ED visits at VH (P=0.00025) (Figure 3). There were positive associations between higher mean 3-d temperatures and median LOS in the NDH stretcher ED section (P=0.024), as well as maximum LOS in the NDH ambulatory ED section (P=0.042) and the VH stretcher ED section (P=0.024). Mean 3-d humidex showed similar significant positive associations with stretcher ED section visits at NDH (P=0.036) and VH (P<0.0001) but not with volumes at the ambulatory sections of both centers (Figure 4). Increased stretcher ED section median (P=0.0082) and maximum (P=0.048) LOS was found to be associated with increasing mean 3-d humidex at NDH but not at VH.

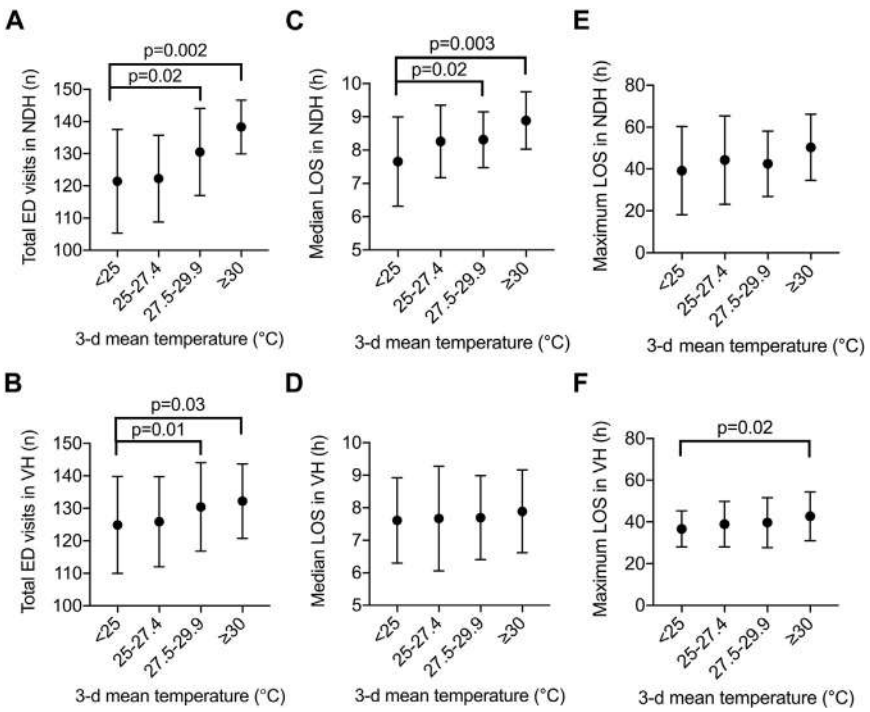

Figure 1. Increased 3-d mean temperatures before emergency department (ED) presentation increases ED volumes and extends ED length of stay (LOS). Daily maximum temperatures were averaged across the 3 d before ED presentation during the summer months of 2016 to 2018 (June 21 to September 20) and were grouped into 3 a priori thresholds (≥25°C, ≥27.5°C, and ≥30°C) to be compared to 3-d mean temperatures <25°C. No days were represented twice within these thresholds. The impact of 3-d mean temperatures was assessed on ED visits at (A) Notre Dame Hospital (NDH) and (B) Verdun Hospital (VH); median LOS at (C) NDH and (D) VH; and maximum LOS at (E) NDH and (F) VH. Data shown are mean±SD. Statistically significant differences are indicated with their respective P-values.

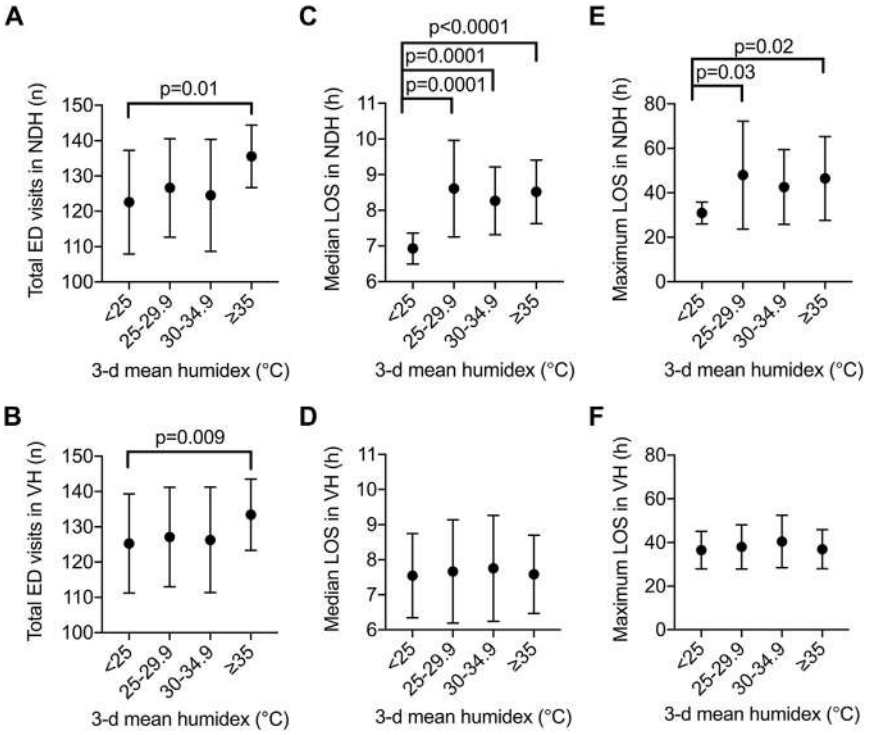

Figure 2. Increased 3-d mean humidex before emergency department (ED) presentation increases ED volumes and extends ED length of stay (LOS). Daily maximum humidex values were averaged across the 3 d before ED presentation during the summer months of 2016 to 2018 (June 21 to September 20) and were grouped into 3 a priori thresholds (≥25, ≥30, and ≥35) to be compared to 3-d mean humidex <25. No days were represented twice within these thresholds. The impact of 3-d mean humidex was assessed on ED visits at (A) Notre-Dame Hospital (NDH) and (B) Verdun Hospital (VH); median LOS at (C) NDH and (D) VH; and maximum LOS at (E) NDH and (F) VH. Data shown are mean±SD. Statistically significant differences are indicated with their respective *P*-values.

Discussion

This study demonstrates that sustained summer heat events with 3 consecutive days of elevated temperatures and humidex leading up to patient presentation to the ED are associated with increased volumes and longer LOS. An escalating impact of mean 3-d temperature and humidex was identified, with mean 3-d temperatures and humidex at the highest thresholds of ≥30°C and ≥35, respectively, showing the most significant increases in ED volumes and LOS across both hospitals. However, impaired ED system performance was noted even when the mean 3-d temperature and humidex preceding ED presentation mildly rose to 27.5 to 29.9°C and 25 to 29.9, respectively. The increased patient volume during heat events may explain the observed increased ED LOS by contributing to an overall busier ED, hampering patient flow through the ED and delaying assessment of patient disposition. Spearman correlation testing provided additional evidence of the disproportionate impact of heat events on the 2 community hospitals; the mean 3-d temperature and humidex were found to be associated with a significantly greater number of measures of ED performance at NDH than at VH. In addition, differential temperature and humidex correlations were observed between the stretcher and ambulatory ED sections at NDH and VH, suggesting that differences in the communities the 2 hospitals serve may contribute to the apparent difference in acuity levels of health conditions exacerbated by heat events.

The associated increase in ED presentations with elevated mean 3-d temperatures and humidex at both centers aligns with a well-established finding in the literature that heat waves significantly increase ED arrivals in populations outside of Canada.[19,23-25] Several factors have been shown to increase ED LOS. Patients with traumatic injuries, patients with higher acuity levels, patients requiring specialist consultation or hospital admission, longer laboratory turnaround time, and higher patient volume have all been shown to increase ED LOS.[26-28] There are limited data on the impact of heat events on ED LOS, especially in Canada. One study demonstrated that prolonged ED LOS is associated with increased pediatric hospital

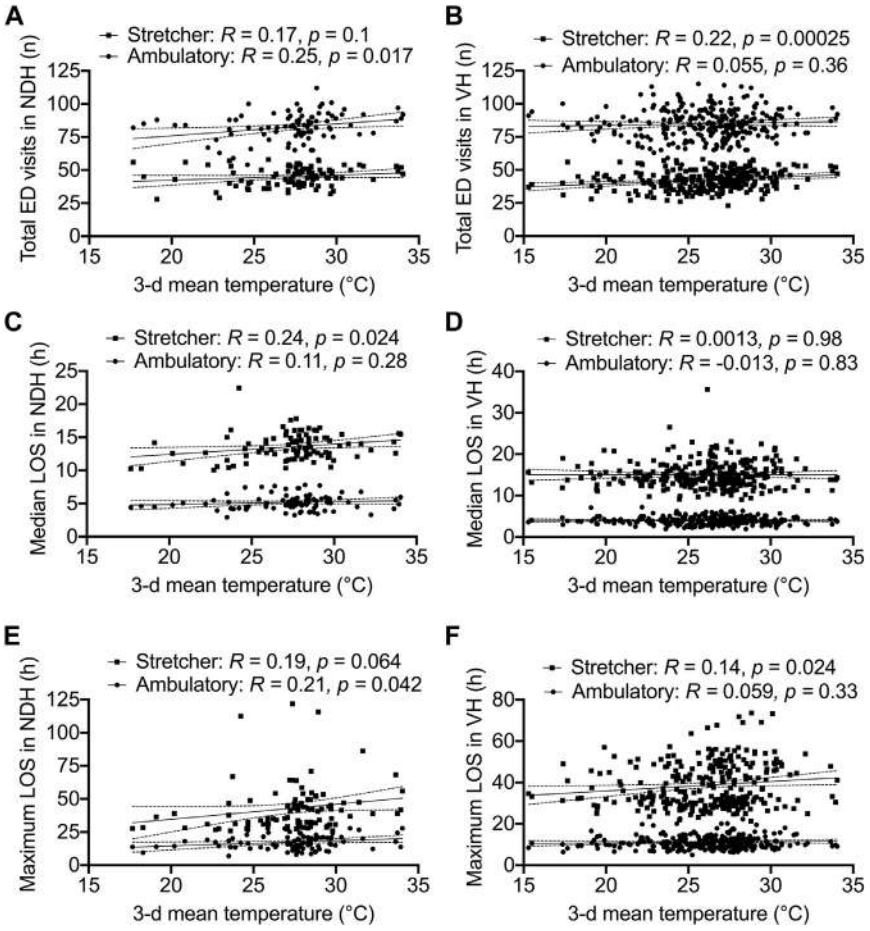

Figure 3. Increased 3-d mean temperatures before emergency department (ED) presentation are associated with differential volumes and length of stay (LOS) changes in the ED ambulatory and stretcher sections. Associations between 3-d mean temperatures and ED visits are shown for (A) Notre-Dame Hospital (NDH) and (B) Verdun Hospital (VH); median LOS for (C) NDH and (D) VH; and maximum LOS for (E) NDH and (F) VH. Spearman's rank correlation analysis R values and *P*-values are shown.

presentations from April to September; however, this study did not demonstrate that specific heat events are associated with ED LOS.[29] Our findings provide local Canadian evidence that is concordant with US data indicating a positive relationship between the mean 3-d heat index and presentation volumes and LOS for patients in the adult and pediatric ED of the Johns Hopkins Hospital in Baltimore, Maryland.[30]

This study's correlation analyses suggested that heatwaves were particularly associated with increased higher-acuity ED presentations, as demonstrated by the observed associations between 3-d temperatures and humidex and increased LOS and presentations particularly in the stretcher sections of both hospitals. Previous health services research has established that longer ED LOS leads to poorer patient outcomes, especially in sicker patients. Delays of ≥4 h in critical care transfers after physiologic deterioration have been associated with a 3.5 times higher mortality rate.[31] Prolonged ED LOS is associated with increased adverse perioperative outcomes for otherwise healthy patients awaiting surgical procedures, as well as reduced thrombolysis and reduced functional status at discharge for patients with cerebrovascular accidents.[32,33] ED LOS is independently associated with increased risk of hospital mortality in patients with sepsis requiring critical care admission.[34] In addition, each 1 h increase in ED LOS has been shown to be associated with a 44% increase in the odds of patients leaving without being assessed, which has been linked to complications and subsequent return to medical care.[20,35] Further research would benefit from investigating the specific ED presentations and resulting diagnoses most affected by heatwaves.

Increased patient presentations to the ED and prolonged ED LOS due to extreme heat events increase the financial burden on the healthcare system. It is possible that the public health system in conjunction with emergency services can dynamically adapt to meet the needs

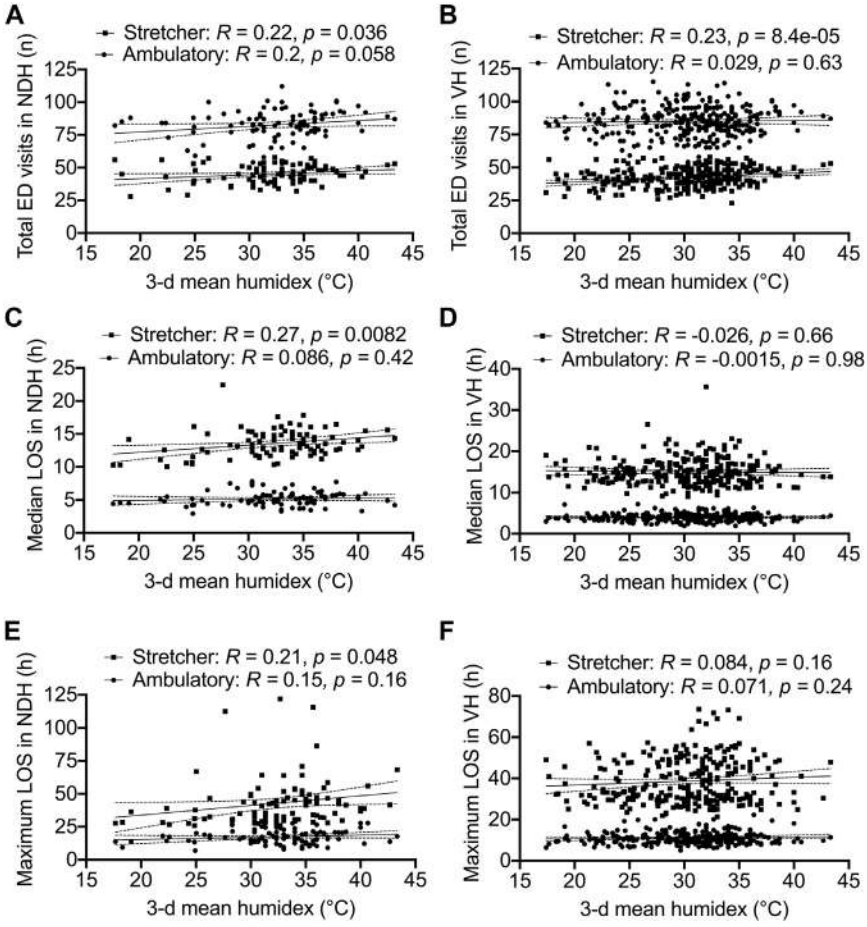

Figure 4. Increased 3-d mean humidex before emergency department (ED) presentation is associated with differential volumes and length of stay (LOS) changes in the ED ambulatory and stretcher sections. Associations between 3-d mean humidex and ED visits for (A) Notre-Dame Hospital (NDH) and (B)-Verdun Hospital (VH); median LOS for (C) NDH and (D) VH; and maximum LOS for (E) NDH and (F) VH. Spearman's rank correlation analysis R values and *P*-values are shown.

of patients during periods of extreme heat. In addition to increasing clinical staffing and providing telemedicine support, interprofessional collaboration to provide community resources (eg, cooling stations and shelters) can aid in preventing return ED visits.[30,36,37]

Of particular interest, heat events affected ED performance metrics in the 2 hospitals in this study differently. The data suggest that even as both VH and NDH saw increased ED volumes in association with sustained heat events, the association between mean 3-d temperature and humidex and median LOS only reached significance at the stretcher section at NDH. A potential explanation for this finding is that the NDH serves a different community with different established risk factors for heat-related mortality, such as reduced access to home air conditioning, adequate ventilation, and shelter from extreme heat.[38,39] From the 2016 Statistics Canada census, residents in the catchment area for NDH were reported to have a much lower median total household income compared to residents in the VH catchment area. Future research would benefit from exploring these factors that may be contributing to the differential vulnerability of various populations and could inform ED quality improvement projects to address climate change-related exacerbation of existing health inequities.

LIMITATIONS

This study should be interpreted in view of several limitations. Although these data are from 2 hospitals, both community hospitals were in the same Canadian city. These findings may not be generalizable to rural EDs. In addition, this study has the limitations inherent to a retrospective study. The associations we found between

environmental variables of temperature and humidex and ED volumes and length of stay are correlative and do not examine the etiologies that led patients to present to the ED. One of the heat events in 2018 coincided with a statutory holiday during which system capacity was already reduced, potentially confounding the results. Finally, specific patient data including presenting complaint, admission to hospital, and consultation with a specialist were not available. These patient characteristics may have contributed to extended LOS in the ED.

Conclusions

This study found that elevated temperatures and humidex exerted an extra burden on ED system capacity by increasing the volume of patient presentations and increasing LOS in the ED in 2 community hospitals in Montreal. EDs are the safety nets of the healthcare system, and climate change threatens to further challenge the timely delivery of emergency healthcare services. Hospitals and EDs need to be climate-ready and resilient.

Author Contributions: Study concept and design (FK, SR); acquisition of the data (SR): analysis of the data (FK, ODL); drafting of manuscript (FK, ODL, SR); critical revision of the manuscript (FK, ODL, SR); approval of final manuscript (FK, ODL, SR).

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ORIGINAL RESEARCH

Closed Reduction of Anterior Shoulder Dislocations Performed by Ski Patrollers in the Alpine Prehospital Environment: A Retrospective Review Demonstrating Efficacy in a Canadian Ski Resort

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Introduction—Shoulder dislocations are common ski hill injuries. Rapid reduction is known to improve outcomes; however, advanced providers are not always available to provide care to these patients. In 2017, nonmedical ski patrollers at Sunshine Village ski resort in Alberta, Canada, were trained to perform anterior shoulder dislocation (ASD) reductions. Program success was determined by a chart review after the 2020 ski season.

Methods—This study retrospectively reviewed data on patients who presented to Sunshine Village ski patrol with a suspected ASD and who met the study inclusion criteria from November 2017 through March 2020. Data were collected from ski patrol electronic patient care records regarding general demographics, reduction technique used, analgesia administration, and reduction success rates.

Results—Ninety-six cases were available for review after exclusions. Trained nonmedical ski patrollers successfully reduced 82 of these cases, resulting in an overall reduction success rate of 89%. Sixty-three (66%) of these patients had experienced first-time dislocations. Eighty-two (87%) patients were male, with a median age of 25 y. The most used technique was the Cunningham method (75%), and analgesia was administered to 70% of patients.

Conclusions—This retrospective study documents the results of a quality assurance review of the treatment of ASD at Sunshine Village ski resort. With a success rate of 89%, the evidence supports the conclusion that nonmedical ski patrollers can successfully perform ASD reductions. We believe training ski patrollers to reduce ASD improved patient care in our austere environment by providing early definitive treatment with a high success rate.

Keywords: wilderness, austere, first responder, skiing/snowboarding, Cunningham's, FARES

Introduction

Skiing and snowboarding are among the favorite pastimes of many outdoor enthusiasts. Unfortunately, injury rates have been reported at around 2 injuries per 1000 rider days.[1–3] It has also been reported that 7% of these injuries are shoulder dislocations,[4] which ranks this injury in the top 5 for alpine skiers and snowboarders.[5] Approximately 19 million people visit Canada's 275 ski resorts annually, resulting in a substantial number of recreational users presenting to ski patrol with anterior shoulder dislocation (ASD). Early reduction of ASD injuries can reduce pain, ease evacuation and transport, improve ease and time to reduction, and potentially reduce neurovascular injury and long-term complications.[6–8] Shoulder reduction is not within the scope of the Canadian Ski Patrol Service, which is the standard of care at ski resorts in Canada, leaving traditionally trained first responders only able to provide supportive care for ASD. Owing to the geographic location of many Canadian ski resorts, injured skiers and snowboarders will often experience significant

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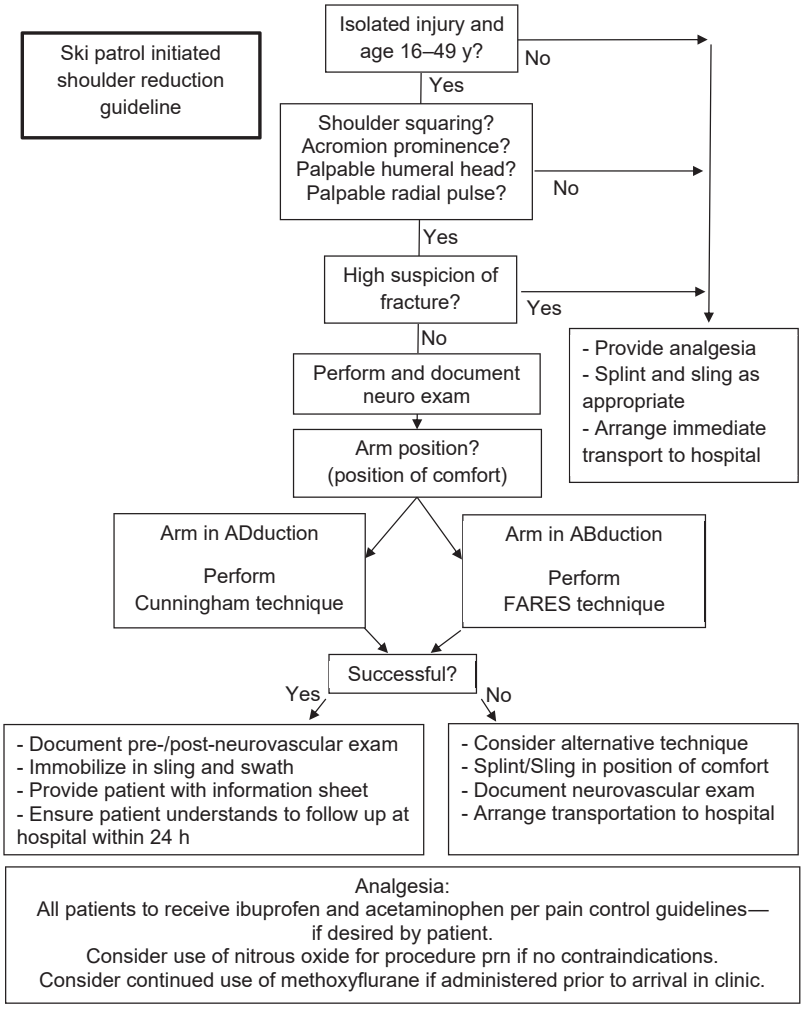

Figure 1. Sunshine Village ski patrol anterior shoulder dislocation protocol. The following is the guideline used by nonmedical ski patrollers at Sunshine Village Ski Resort to assess and treat anterior shoulder dislocations when they occur at the resort.

delays in accessing medical care. Most Canadian resorts do not have physicians available to provide care, nor are they available on transport ambulances. Without consistent physician availability at these resorts, training ski patrollers to reduce obvious ASDs provides an opportunity to improve care among this cohort of patients.

This project was performed at Sunshine Village (SSV) ski resort in Banff, Canada, a large ski resort located in the Canadian Rockies. The resort sees over 550,000 recreational skier/boarder visits annually and has the longest nonglaciated ski season in North America. Resort ski patrol assess and treat all injuries and illnesses that present to the ski resort clinic and treat approximately 1200 patients each season.

Treatment protocols for ski patrol members were adapted to include reduction attempts for clinically obvious ASD in the fall of 2017. The ASD protocol (Figure 1) was developed to provide ski patrollers the option to perform ASD reduction before patient transport to the local hospital and to be used when a physician is not immediately available to provide this treatment. The objective of this protocol was to shorten the time to a definitive procedure, reduce pain and suffering, and reduce complications when faced with prolonged transport times and/or difficult field extrication. This protocol was developed by the ski patrol medical director, an experienced emergency medical services/flight physician. Choice of analgesia, inclusion and exclusion criteria for use of the protocol, training standards, and choice of biomechanical techniques was determined by the medical director based on his personal experience and research.

Searches of the literature on out-of-hospital shoulder reductions by nonmedically trained individuals revealed few results. No evidence could be found on first

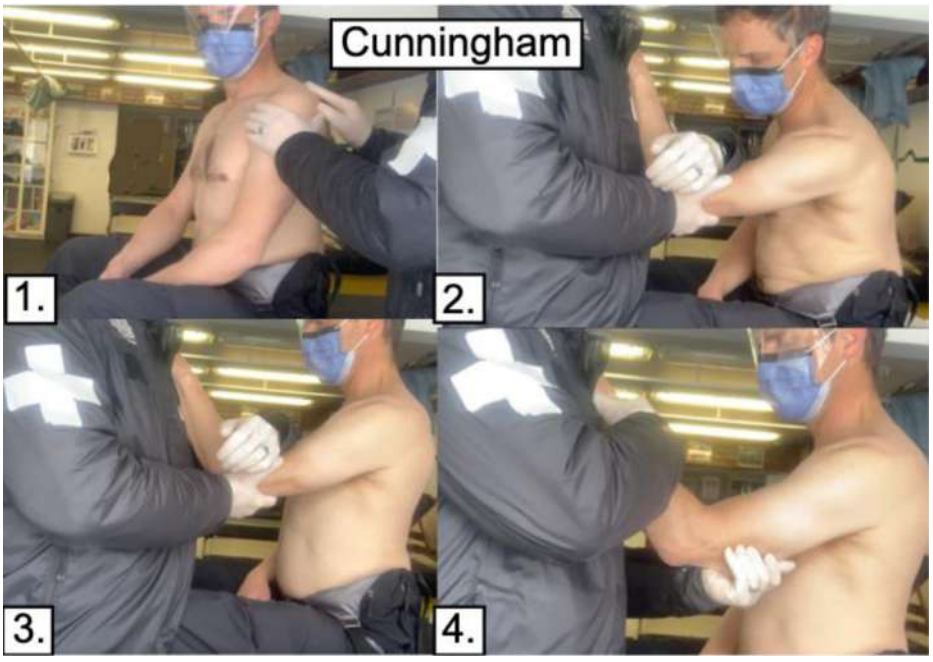

Figure 2. Cunningham technique. The Cunningham technique relies on massage with the arm in a neutral position to reduce spasm and allow spontaneous reduction. This technique relies on good communication with the patient and coaching them to relax and maintain good posture with shoulder blades retracted, which is critical for success. Steps: 1. Perform detailed shoulder assessment. 2. Sit patient upright in a chair opposite the practitioner. The hand of the patient's affected arm should rest on the practitioner's shoulder. 3. The practitioner can rest their arm in the bent elbow space of the patient's affected arm. No more traction is necessary. Constantly encourage patient to maintain good posture and relax. 4. The practitioner then massages the affected shoulder in sequence: first, trapezius; second, deltoid; third, biceps; and, fourth, triceps. Use pressure as tolerated. Repeat. Video available at: https://www.youtube.com/watch?v=yiA3x02CMQA

responders performing reductions as the standard of care in Canadian ski resorts. After the apparent success of the ASD reduction program at SSV, the authors believed the results of the quality assurance review should be shared. The primary objective of this study was to determine whether ski patrollers can be taught to examine and reduce obvious ASDs in a remote hillside clinic.

Methods

This retrospective study was reviewed and approved by the University of Calgary as a quality improvement project. As such, the project was exempt from ethics review by the conjoint health research ethics board of the University of Calgary (Tricouncil policy statement 2018, Chapter 2, Article 2.5).

Our literature search used Medline, BMS Engine, Biomedical Reference Collection, HealthSource: Nursing/Academic, Google Advanced, and CINAHL for "pre-hospital, OR wilderness OR out-of-hospital" AND shoulder reduction AND "paramedic OR rescuer OR EMT OR ski patrol."

Only ski patrollers with 3 or more years of experience at a wilderness first responder level were permitted to partake in this advanced training. Ski patrollers who were trained as advanced care paramedics (EMT-P), registered nurses (RN), or physicians (MD) were not included in the program or subsequent audit.

The training consisted of 3 phases. First, qualified patrollers attended educational sessions on shoulder anatomy, shoulder assessment, and ASD identification. Next, each patroller assisted an on-hill physician in 6 ASD reductions that presented to the medical clinic. Finally, patrollers performed an additional 6 ASD reductions under the direct supervision of an on-hill physician. All cases during this training were then further reviewed by the medical director and ski patrol director before the patroller was approved to independently reduce ASDs. A total of 10 patrollers were trained and provided care over the study period.

Assessment and diagnosis of ASD was performed by the patroller after adequate history, exposure, and examination of the injured patient in the warm environment of the medical clinic. Diagnosis was made by determining the history and signs and symptoms of ASD,

Figure 3. FARES technique. The FARES method requires patients to be supine on an elevated platform for the practitioner to have the mobility to perform the procedure. The oscillations resemble a handshake when performed. Steps: 1. Patient should be lying supine on a raised surface with the affected arm adducted alongside them. 2. Practitioner to take patient's wrist. While maintaining a gentle, steady longitudinal traction, the practitioner should begin to abduct the injured arm. 3. While abducting and maintaining slight traction, small oscillations of the arm should be made 2 to 3 times a second, lifting and lowering the arm 10 to 15 cm. 4. When the arm is abducted to 90°, externally rotate and continue traction, oscillations, and abducting arm. Reduction usually occurs at approximately 120° of abduction. Video available at: https://www.youtube.com/watch?v=RCD0sZREYIg

including the mechanism of injury, pain, limited range of motion, and step deformity of the shoulder complex. All patients were examined for neurovascular deficits, obvious associated fractures, and other injuries. Radiologic confirmation was not performed because imaging was not available at the ski resort clinic.

The 2 positioning and muscle relaxation techniques selected for the protocol were the Cunningham and the FARES, which were chosen to account for subglenoid and subcoracoid dislocations.[9–12] These biomechanical techniques have been described using minimal or no analgesia/sedation, minimal traction and force, and simple positioning to facilitate reductions. Selection of technique was based on patient and arm positioning (Figures 2 and 3).

Success was determined by signs and symptoms, including pain reduction, recovery of normal range of motion, and normal appearance. Patients with unsuccessful reduction attempts by the ski patrol were referred to an on-hill physician, if available, or transferred directly to the hospital after appropriate analgesia and splinting. All patients agreed to immediate post-care follow-up, including referral to a clinic that could provide appropriate imaging and orthopedic consultation.

At SSV, a computer database (EDGEauditor, Orangeville, ON, Canada) was used to record patient information and accident and treatment details on all patients seen by the patrol service. We retrospectively reviewed all patient visits to ski patrol from November 2017 through March 2020. First, we identified all patients with a chief complaint of "shoulder injury." Further screening then included all patients specified as having a dislocation. We then excluded patients who met any of the following criteria: shoulder reduction performed on the ski slope suggesting spontaneous reduction, no reduction attempted owing to risk of clinically significant fractures, and no reduction attempted owing to patient refusal to consent to the procedure. These patients were removed from the study because the protocol for ski patrol reduction excluded these patients from a reduction attempt. We also excluded patients who had an ASD reduction performed by an intermediate or advanced practitioner (MD, EMT-P, or RN) or who received intravenous or intramuscular analgesia. Finally, any patient for whom exclusion criteria could not be determined owing to incomplete documentation was also excluded from the study. Included charts were reviewed for the following

Table 1. Summary of data collected

	Pooled	*Male*	*Female*
Reduction attempted, n (%)	96 (100)	82 (100)	14 (100)
Successful reduction, n (%)	82 (89)	70 (90)	12 (86)
Age, median [interquartile range], y	25 [21–29]	24 [22–29]	26 [24–30]
Sex, male, n (%)	82 (86)	82 (100)	-
Skier, n (%)	36 (38)	33 (40)	3 (21)
Snowboarder, n (%)	59 (62)	48 (59)	11 (79)
First dislocation, n (%)	63 (66)	55 (67)	8 (57)
Reduction technique, n (%)			
Cunningham	55 (74)	46 (74)	9 (75)
FARES	6 (8)	6 (10)	0 (0)
Combined Cunningham + FARES	2 (3)	2 (3)	0 (0)
Other	4 (5)	3 (5)	1 (8)
Cunningham + other	4 (5)	3 (5)	1 (8)
Combined Cunningham + FARES + other	3 (4)	2 (3)	1 (8)
Medication used, n (%)	66 (69)	55 (67)	11 (79)
Type of medication used, n (%)			
Acetaminophen only	2 (3)	2 (4)	0 (0)
Acetaminophen + ibuprofen	7 (11)	5 (9)	2 (14)
Entonox only	40 (61)	33 (60)	7 (50)
Nitrous oxide + acetaminophen	3 (4)	3 (5)	0 (0)
Nitrous oxide + acetaminophen + ibuprofen	9 (14)	8 (15)	1 (7)
Nitrous oxide + ibuprofen	1 (2)	0 (0)	1 (7)
Nitrous oxide + methoxyflurane	1 (2)	1 (2)	0 (0)
Ibuprofen only	1 (2)	1 (2)	0 (0)
Methoxyflurane only	1 (2)	1 (2)	0 (0)
Methoxyflurane + acetaminophen + ibuprofen	1 (2)	1 (2)	0 (0)

information: age, sex, type of sport (skier vs snowboarder), characterization of dislocation (first-time dislocation vs recurrent dislocation), reduction techniques used during attempt (Cunningham, FARES, other), and success of reduction attempt.

Microsoft Excel (Microsoft, Redmond, WA, USA) was used for data collection. Continuous demographic data are presented as median [interquartile range] because the sample group was not evenly distributed within the inclusion criteria. Count data are presented as frequency (percentage). Overall success rates of onsite reductions are presented using frequency (percentage [95% CI]). Logistic regression analysis was completed to understand the association between reduction technique and an unsuccessful reduction. Reduction techniques were grouped into 1 of 3 categories: Cunningham, FARES, or other (includes combined Cunningham + FARES, other, Cunningham + other, and combined Cunningham + FARES + other). Associations between technique and an unsuccessful reduction are given using odds ratios (ORs) with 95% CIs. Statistical analysis was completed using SPSS 25.0 (IBM, Armonk, NY). Statistical significance was defined as $P<0.05$.

Results

A total of 117 patient cases were identified by the ski patrol as having an ASD. After applying the exclusion criteria, we identified 96 patients who had an onsite reduction attempt. Male and female patients were pooled because the total number of female patients included in the study did not provide a large enough sample to be statistically analyzed (Table 1). The median age of patients treated was 25 y (IQR 21–29 y). Males accounted for 86% of patients, and snowboarders and skiers accounted for 62% and 38% of patients, respectively. Sixty-three (66%) patients presented with a first-time ASD injury.

Of the 96 reductions performed onsite, 82 reductions were successful using the ASD protocol (89% [95% CI 81–95%]). Reduction success for first-time dislocations and recurrent dislocations was 90% and 87%, respectively, which did not show a statistically significant difference. The odds of a successful reduction were 1.33 times greater in the first-time dislocation group, but with a very wide CI (95% CI 0.3–5.1).

The most common reduction technique used was the Cunningham (75%), whereas the FARES technique

accounted for 9%. The success rate of the first attempt was 75% (95% CI 65−83%). The odds of having an unsuccessful reduction were not significantly increased by use of the FARES (OR=2.4 [95% CI 0.2−26.4, P=0.46]) or another (combination) technique (OR=2.4 [95% CI 0.4−15.3, P=0.38]). The odds of failure were not significantly different between techniques selected (OR=2.5 [95% CI 0.2−26.4; P=0.46]).

Analgesia was administered in 66 patients, with nitrous oxide being the most common (61%), followed by a combination of ibuprofen, acetaminophen, and nitrous oxide (14%). No analgesia was administered to 31% of patients.

Discussion

Reduction of ASD is not uncommon in the prehospital environment. However, there is limited documentation of protocols or other support for nonmedical personnel performing reductions when physicians are not available. Our literature search found only 6 peer-reviewed articles pertaining to the topic.[7,13−17] All 6 articles report a similar lack of available evidence. This lack of evidence may be a factor in why shoulder reductions have not been widely adopted by medical directors in prehospital settings.

The demographics of our patient cohort appeared to be consistent with the ASD literature, although our age range was slightly lower than most.[14,16,18−22] Our median age (25 y) corresponded with data indicating that younger males (20−30 y) appear to be most likely to experience ASD injuries.[21,23,24] The ratio of skiers and snowboarders in our study, as well as the observation that snowboarders are more likely to injure their upper extremities compared to skiers, is also consistent with other reports.[4,25,26] Finally, the number of patients who experienced their ASD as a first-time injury (66%) is comparable with the literature discussing ASD presentations in the emergency department.[22]

The 3 phases of training established for ski patrollers to independently reduce ASD were chosen to ensure that correct application of the techniques was understood and performed; improper application could reduce success rates and ultimately prolong treatment. A newly developed commercial task trainer (https://www.sawbones.com/) may prove effective in improving our current training regime.

We recognize that complete management for ASD should occur in a facility that can provide imaging and orthopedic follow-up. Most of the population lives in urban settings and can access this standard of care in a reasonable period. In remote and rural locations, meeting this goal is often not possible. It has been suggested that prehospital management of ASD could decrease time to reductions by 130 min versus when performed in the hospital.[13] Unfortunately for ASD patients, prolonged time to reduction (>60 min) has been documented to increase complications and make the injury more painful as muscle spasm increases.[8,27,28] Although analgesia via ibuprofen, acetaminophen, nitrous oxide, and, recently, methoxyflurane provides some relief and is available to some ski patrol services, the definitive treatment for ASD is joint reduction.

Historically, standard methods for reducing ASD have been traction/countertraction and leverage techniques that require procedural sedation. As a result, they commonly require direct physician involvement for medication administration, monitoring, and postprocedural recovery.[29−31] Recent literature continues to support moving away from traditional traction/countertraction and leverage methods and moving toward biomechanical techniques such as FARES and Cunningham for ASD reduction.[12,16,31,32] These techniques have become popular because they are less painful for patients, require fewer personnel, and decrease the risks associated with conscious sedation.[10,12,16,19,29,33] This makes biomechanical reduction techniques ideal for use by nonmedical personnel, or in a low-resource environment.

Results using the Cunningham technique have been extremely varied. Success rates of 35 to 100% exist in the literature.[34,35] The FARES has less published data on its success rate, but 1 randomized control trial found a success rate of 89%.[33] A comparison of the success rate of our ski patrol to the ASD reduction success rates in the literature suggests that ski patrollers can reduce ASD consistently.[16,29,30,32−35]

Although biomechanical techniques may be more tolerable without analgesia, pain management was available for use in this protocol. Administration of over-the-counter ibuprofen and acetaminophen to all patients was encouraged, and nitrous oxide and methoxyflurane were available for patrollers to administer if required. It is noteworthy that none of these medications (acetaminophen, ibuprofen, nitrous oxide, and methoxyflurane) required administration by an advanced practitioner (MD, RN, or EMT-P). Furthermore, most of these medications are available to wilderness first responders, including ski patrollers. Beyond over-the-counter medications such as acetaminophen and ibuprofen, nitrous oxide and methoxyflurane have become popular owing to their quick onset of action and the ability of patients to optimize pain relief in a dose-response manner. Methoxyflurane shows great promise and is only recently available in Canada. Its use by SSV ski patrol as a means of analgesia was introduced halfway through the study period. Because the reduction of ASD may prove more difficult over time,[8] the

relatively short onset of action of inhaled analgesia seems to be preferred by practitioners and is anecdotally very effective within the scope of our review.

The results of this study are likely not a surprise; however, there are often perceived barriers for first responders to perform ASD reductions in the field, which we would like to address here. These barriers include the inability to provide imaging and the risk of causing a fracture or worsening the injury.

The authors acknowledge that reductions without prereduction imaging are controversial. Although common practice may vary, it is still recommended that care of sports-related shoulder dislocations in the emergency department include prereduction x-rays.[24,28,36,37] It is not uncommon, however, for physicians experienced with shoulder reduction, or those in resource-limited settings, to choose to reduce obviously dislocated shoulders before imaging. This practice is also supported in the literature.[16,18,28,38,39]

One stated concern for attempting reduction without prereduction imaging is the presence of a dislocation-associated fracture. Indeed, the frequency of ASD-associated fractures has been reported as high as 30%.[22] However, most of these fractures (which include Bankart lesions, Hills-Sachs lesions, and greater tuberosity fractures) do not affect acute management of the shoulder dislocation.[22,34,40] Although these associated fractures may increase the risk of dislocation recurrence,[8,30,40] they are generally not considered for treatment by orthopedics until after the initial injury has been reduced.[37]

In addition to concerns regarding ASD-associated fractures, concerns such as causing neurovascular or other injury during reduction appear to be unfounded when nontraction, biomechanical-style techniques are employed.[14,28,34,38] To our awareness, there are no case studies or other reports in the literature that indicate biomechanical techniques have been associated with neurovascular or other injuries. In addition to these newer biomechanical techniques being safe, they have also proven to have great first-time-attempt success, even without analgesia or sedation.[12,16,19]

The ASD protocol attempted to ensure patient safety by screening, and subsequently excluding, any patients at high risk of clinically significant fractures. These injuries, which include humeral neck, clavicle, and scapula fractures, significantly increase the difficulty of performing closed reductions without conscious sedation. Providers used the following signs, symptoms, and mechanisms for screening: report of direct trauma to the shoulder, severe disproportionate pain, age over 50 y, crepitus in or near the joint, or obvious deformity not consistent with a "squared shoulder" or Sulcus sign. These criteria are like those in other prehospital reduction studies.[16] Screening assessment tools such as the Quebec rule or the modified Fresno-Quebec rule do not apply well in our context because they automatically exclude any sports-related cause of injury.[36,41] To the authors' knowledge, no validated assessment tool has been developed to determine if a fracture exists. Evidence does demonstrate, however, that trained personnel who have a high clinical suspicion of uncomplicated ASD are often shown to be correct after x-ray confirmation.[18]

LIMITATIONS

There were several limitations to our data, including, most importantly, the study design. As a retrospective chart review, we were unable to compare our results to a control group, compare success rates of reductions on first-time ASD versus recurrent ASD, or investigate the effectiveness of 1 technique after the failure of another. In addition, we were unable to review medical charts from the hospital to determine if any complications were noted. Inconsistent charting was also problematic, resulting in difficulty in determining the precise time from initial injury to reduction, changes in pre- and postreduction pain, and neurovascular examination results. Improved reporting standards now implemented by the SSV ski patrol will hopefully help to answer these questions in the future.

Age exclusion criteria (<16 y and >50 y) may have limited the collection of data from a broader age group. A further limitation is that the ASD injuries included in the study were not confirmed with x-ray imaging and were instead confirmed by practitioner assessment. As a result, we cannot definitively conclude that each shoulder reduction attempt was performed on a true ASD. With regard to data collection, the initial database search criteria for "shoulder injury" may have also failed to capture ASD injuries that had been labeled differently when initially recorded in ski patrol patient records.

In terms of practitioner demographics, there were no female patrollers within the group of 10 patrollers initially trained to reduce ASD. Although patrollers were selected based on factors such as years of experience, this lack of female practitioners may have led to unconscious gender bias when female patients were treated by male practitioners. Furthermore, the small number of female patients included in the study limits the ability to assess any differences in outcome between males and females. As such, future studies will need to have a larger sample size to ensure these data can be captured.

Conclusion

This retrospective study documents the results of a quality assurance review of the treatment of ASD at SSV ski resort. A success rate of 89% supports the idea that

nonmedical ski patrollers can successfully perform ASD reductions. We believe training ski patrollers to reduce ASD improved patient care in our austere environment by providing early definitive treatment with high success rates. Overall, more research is still needed to better document: 1) the actual time saved between initial injury and reduction when performing prehospital reductions, 2) complications of prehospital reductions, and 3) the patient experience of ski hill shoulder reductions compared to traditional hospital management.

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ORIGINAL RESEARCH

Longer Tendon-Bone Distances of the A2 and A4 Annular Pulleys in Experienced High-Level Sport Climbers: Injury or Adaptation?

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Introduction—Experienced high-level climbers are subject to a number of bone and soft tissue changes over the years and are also among the most exposed to pulley injuries. One of the main consequences of pulley rupture is the separation of the flexor tendons from the subjacent phalanges, also known as bowstringing. The purpose of this study was to determine whether this population has asymptomatic bowstringing of the A2 and/or A4 pulleys as determined by tendon-bone distance (TBD) values when compared to nonclimbers.

Methods—High-resolution ultrasound TBD measurements in active forced flexion were made for the A2 and A4 pulley of the ring finger bilaterally. Participants were 21 asymptomatic sport climbers who had 21 consecutive years of climbing at a level above 9.66 in the International Mountaineering and Climbing Federation difficulty metric scale. Control subjects were 21 age-matched nonclimbers.

Results—A significantly longer TBD−25% (0.3 mm) and 35% (0.4 mm) for the A2 and A4 pulleys, respectively−was found in the experienced climbers group (experienced climbers group: A2 1.6±0.5 mm and A4 1.6±0.4 mm; nonclimbers group: A2 1.2±0.1 mm and A4 1.2±0.2 mm).

Conclusions—Our results suggest that bowstringing of A2 and A4 pulleys occurs in asymptomatic experienced high-level climbers, which could be interpreted as either an adaptive mechanism to workloads endured over years of climbing or a consequence of underdiagnosed pulley ruptures.

Keywords: ultrasound, bowstringing, noninjured, asymptomatic

Introduction

Rock climbing is a sport practiced worldwide and has been recently accepted for the 2021 Olympics.[1] With growing global interest in this sport, there has been an increase in the number of climbers, together with an improvement in climbing equipment and protection measures. These have led to a proportional reduction in the number of injuries[2,3] and a change in the most frequent injury types, which have shifted from leg injuries associated with traumatic falls[1] to overuse injuries mainly affecting the shoulder and fingers.[4] The most recent reports show that 33% of all climbing injuries affect the flexor tendon sheath of the hand, especially the A2 and A4 annular finger pulleys of the middle and ring fingers.[1,5] In adult rock climbers, the latter continues to be the most common.[6]

Another important change in the practice of rock climbing has occurred in relation to sport climbing routes, as historic levels of difficulty have been surpassed and levels continue to rise.[7] Currently, the hardest sport climbing grade is 9c measured using the French system, 5.15c/d in Yosemite Decimal System, or 12.33 in the metric scale of the International Mountaineering and Climbing Federation (UIAA),[8] and the increase in difficulty is attributable mainly to developments in strategies and training. Different grading systems exist to rate

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climbing route difficulty, and the UIAA metric scale is a recognized sport-specific scoring system widely used for injury-reporting scientific analysis.[8]

Climbers start training at younger ages, and the trend is that elite climbers are much younger and have more demands and greater workloads, especially at the level of the fingers.[9,10] At this point, it is important to mention that injury type distribution is different in adolescent compared to adult climbers.[11] Whereas A2 and A4 pulley ruptures are the most frequent injuries in adult climbers, epiphyseal growth plate fractures to the base of the middle phalanx are the most prevalent injuries in youth climbers.[12]

High workloads on fingers sustained over years lead to an adaptive process in several anatomic components of the fingers.[7] In adult climbers, adaptive changes in the bone and soft tissues of the fingers have been described. Compared to nonclimbers, radiographic findings of the middle phalanx include 25% higher cortical proportions, up to 6% greater cortical widths, and up to 20% narrower medullary canals.[13] Overall, the A2 pulley is thicker in 69% of climbers, whereas the A4 pulley is thicker in 75% of climbers. The volar plate of the distal interphalangeal joint is also significantly thicker in climbers compared to nonclimbers.[7] Controversy still exists regarding the thickening of the finger flexor tendons at the proximal phalanx as an adaptive response to workload.[7,14]

High-resolution ultrasound (US) is one of the most suitable diagnostic methods for traumatic finger injuries.[15] Both qualitative and quantitative US signs have been proposed for the diagnosis of ruptured annular pulleys, tendon-bone distance (TBD) measurement being among the most recommended.[16] The most accepted TBD threshold to diagnose a complete pulley rupture is 2 mm.[1] However, in rock climbers, TBDs considered pathological are not always accompanied by clinical symptoms or loss of grip strength.[17] This has prompted the hypothesis that either adaptive changes or subclinical injuries could generate finger pulley slackening, related to a certain degree of bowstringing evidenced sonographically by an increased TBD.

The main objective of the present study was to determine whether the TBD of the A2 and A4 pulleys is greater in asymptomatic experienced high-level climbers compared to age-matched asymptomatic nonclimbers. As secondary objectives, we also aimed to determine whether 1) TBD differences exist between the A2 and A4 pulleys, 2) TBD differences exist between the dominant and nondominant hands, and 3) asymptomatic experienced high-level climbers may have TBDs over 2 mm.

Methods

We designed a cross-sectional study in which we recruited noninjured experienced high-level sport climbers to undergo a single US assessment. Inclusion criteria were a lack of clinical symptoms and a minimum

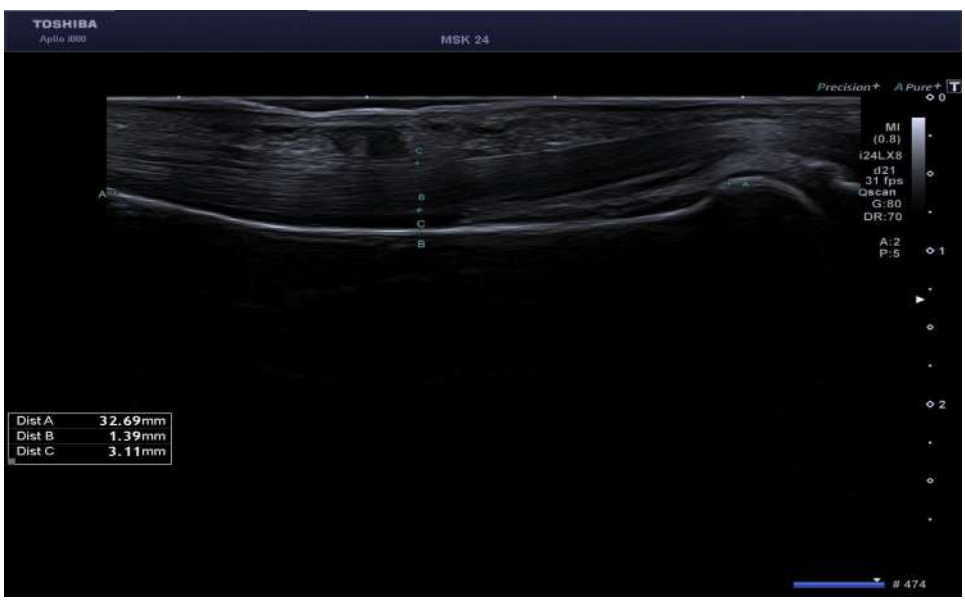

Figure 1. High-resolution ultrasound image of a non-ruptured A2 pulley. The distance B indicates the tendon-bone distance in the midpoint of the proximal phalanx.

Table 1. Demographic data

	Climbers (n=21)		*Nonclimbers (n=21)*		P *value*
	Mean±SD	*95% CI*	*Mean±SD*	*95% CI*	
Age (y)	42±6	39–45	42±7	39–45	0.769[a]
Weight (kg)	68±6	66–71	69±6	67–72	0.558[a]
Height (cm)	173±6	171–176	173±6	171–176	0.914[a]
Highest level in sport climbing[b]	10.5±0.3	10.3–10.6	–	–	–
Years of climbing	26±5	23–28	–	–	–
Years of climbing above 9.66 climbing level	21±4	19–23	–	–	–

[a]*P* value for comparisons between the 2 study groups±Student t test.
[b]UIAA climbing difficulty metric scale.

of 15 consecutive years of climbing at a difficulty level above grade 9.66 on the UIAA metric scale.[8] Exclusion criteria were any previous diagnosis of pulley rupture of the ring finger or hand surgery. The study finally included 21 rock climbers. The control group was composed of 21 healthy nonclimbers who also met the exclusion criteria.

The study was conducted in accordance with the ethical standards of the Declaration of Helsinki.[18] All data were anonymous and confidential. The study protocol received approval by the research ethics committee of the Camilo José Cela University (Spain, EOP-PADME, 22 March 2018). All patients were informed in writing of the study's objectives and procedures and provided written informed consent to participate.

General demographic and climbing data were collected from all participants (age, weight, height, highest level in sport climbing, years of climbing, years of climbing above UIAA 9.66 climbing level). All participants underwent a US assessment using a Canon Aplio i800 equipped with a 24 MHz linear transducer (Canon Medical Systems S.A., Madrid, Spain). US image quality is in part determined by transducer frequency, and a minimum of 14 MHz is recommended.[19] The participant sat in front of the examiner with forearms and hands in supine position and a neutral wrist position. Finger position was standardized as follows: neutral metacarpophalangeal joint, 40° of flexion of the proximal interphalangeal joint, and 10° flexion of the distal interphalangeal joint.[20] A counter-resisted force to the distal phalanx was applied while the patient was instructed to execute maximum flexion force of the finger, under the pain threshold.[20] The TBD of the A2 and A4 pulleys was then measured along the longitudinal axis of the ring finger bilaterally by placing the transducer over the central palmar aspect of the finger, without any tilt and using abundant gel to avoid compression (Figure 1). Measurements were made over the midpoint of the proximal phalanx for the A2 pulley and the medial phalanx for the A4 pulley.[21]

Statistical analysis was performed using SPSS 22.0 software (SPSS Science, Chicago, IL, USA). Descriptive statistics (mean, SD, and 95% CI for continuous measures) were calculated. The Shapiro-Wilk test confirmed a normal distribution of the quantitative outcome measures ($P>0.05$). A t-test was used to analyze intergroup comparisons for age, weight, and height. A repeated-measure analysis of variance mixed linear effects model was used to compare the TBD measurements of the A2 and A4 pulleys in the fingers of both hands in climber and nonclimber groups (Bonferroni correction was used for post hoc analysis). Percentage differences between climbers and nonclimbers were calculated using the following equation: ([TBD climbers − TBD nonclimbers]/TBD climbers × 100). The effect size measure was determined using Cohen's d (large effect, d>0.8; intermediate effect, d=0.5–0.8). Bivariate correlations

Table 2. Experienced high-level climber group TBDs above 2 mm threshold

	Negative (<2 mm)		*Positive (>2 mm)*		*Min. TBD (mm)*	*Max. TBD (mm)*	*Mean±SD*
Pulley	*n*	*%*	*n*	*%*			
A2	34	81	8	19	0.9	3.3	1.5±0.5
A4	33	79	9	21	1	3	1.6±0.4
A2 and A4	67	80	17	20	1	3	1.6±0.4

Min, minimum value; Max, maximum value; TBD, tendon-bone distance.

Table 3. US measurements in the dominant and nondominant hands of climbers and nonclimbers

	Climbers (n=21)			*Nonclimbers (n=21)*			*Difference climbers vs nonclimbers*		P *value*	*d*
	Mean±SD	*95% CI*	*Range*	*Mean±SD*	*95% CI*	*Range*	*Mean*	*%*		
TBD A2 (mm)										
Dominant hand	1.6±0.5	1.4–1.8	1–3	1.2±0.1	1.2–1.3	0.9–1.6	0.3	28	0.003	0.891
Nondominant	1.6±0.6	1.3–1.8	0.1–3.3	1.3±0.2	1.2–1.3	0.9–1.6	0.3	23	0.047	0.609
Total							0.3	25		
Difference (mm)	0.01±0.4	−0.2 to 0.2		−0.03±0.2	−0.1 to 0.04		0.05	167	0.615	0.157
TBD A4 (mm)										
Dominant hand	1.6±0.3	1.5–1.8	1–2.1	1.3±0.2	1.2–1.3	1–1.5	0.4	29	<0.001	1.13
Nondominant	1.7±0.5	1.5–1.9	1.1–3.0	1.2±0.2	1.1–1.3	0.8–1.5	0.5	41	<0.001	1.12
Total							0.4	35		
Difference (mm)	−0.1±0.4	−0.3 to 0.1		0.02±0.1	−0.02 to 0.1		0.1	43	0.175	−0.421
Difference TBD A2–TBD A4										
Dominant hand (mm)	−0.04±0.4	−0.2 to 0.1	−0.7 to 1.3	−0.0±0.1	−0.1 to 0.04	−0.2 to 0.3	−0.02	10	0.818	0.312
Nondominant (mm)	−0.1±0.6	−0.5 to 0.1	−1.3 to 1.7	0.0±0.3	−0.1 to 0.2	−0.3 to 0.7	0.2	123	0.177	0.309

TBD, tendon-bone distance.

among quantitative variables were assessed through Pearson's coefficient. The confidence level was set at 95% and significance at $P<0.05$.

Results

Of the 31 initial climbers (3 female and 28 male), 4 climbers (3 female and 1 male) did not meet the inclusion criteria of climbing for 15 or more consecutive years above a 9.66 level of difficulty. Although female participants were not excluded a priori, only male climbers met the entry criteria. Consequently, the nonclimbing group consisted of male participants. Of the remaining 27 climbers, 4 were excluded: 3 were diagnosed with complete A2 rupture and 1 with A2, A3, and A4 multiple rupture. Demographic data, performance level, years of climbing experience, and consecutive years of climbing at a level higher than UIAA grade 9.66 are provided in Table 1.

A total of 84 pulleys were measured in each group, half A2 and half A4. In the climbers group, 17 pulleys (20%; 8 A2 [1.5±0.5 mm; 0.9–3.3 mm] and 9 A4 [1.6±0.4 mm; 1–3 mm]) were over the 2 mm TBD threshold, whereas in the nonclimbers group, all pulleys were below the limit value (Table 2). US TBD measurements of A2 and A4 pulleys can be found in Table 3. In the repeated-measures analysis of variance including US measurements for both pulleys, significant interactions were detected for both hands and both groups ($P<0.001$).

Data recorded for the A2 pulley indicated an overall 25% greater TBD in climbers compared to nonclimbers. The difference in TBD was 28% greater for the dominant hand ($P=0.003$), and the effect size was large. Similarly, TBD in the nondominant hand was 23% greater ($P=0.047$), and the effect size was intermediate. No significant differences in A2 pulley TBD were detected between the dominant and nondominant hand ($P=0.615$).

Our data for the A4 pulley also indicated an overall 35% greater TBD in climbers compared to nonclimbers. The difference in TBD was 29% greater for the dominant hand ($P<0.001$), and the effect size was large. Similarly, TBD in the nondominant hand was 41% greater ($P<0.001$), and the effect size was also large. No significant differences in A4 pulley TBD were detected between the dominant and nondominant hand ($P=0.175$).

Within the group of experienced climbers, TBDs were similar for A2 and A4, although somewhat greater for A4. TBD differences between A2 and A4 were not significant when comparing the groups of climbers with controls.

Figure 2. Close crimp grip.

Discussion

One of the main roles of the musculoskeletal system is to generate different force vectors and thus achieve functional movements. When the capacity to perform these functional movements is exceeded by a determinate workload owing to chronic repetitive trauma, body tissues may react in 2 ways: claudication and injury, or adaptation.[22] During adaptation, tissue modifications enable the musculoskeletal system to satisfy much more efficiently the biomechanical demands to which the tissue is subjected.[23] Using different imaging techniques, tissue modifications resulting from a process of adaptation or from a mild injury cannot always be distinguished. Thus, discerning between them depends mainly on the presence or absence of clinical symptoms such as pain or functional limitations.[24]

During the different hand grips employed when climbing, bowstringing of the flexor tendons in the fingers submit the annular pulleys to friction, compression, and traction forces.[25] The grip that causes the greatest load on the pulleys is the crimp grip (Figure 2), involving at least a 90° of proximal interphalangeal joint flexion and distal interphalangeal joint hyperextension.[26] This grip, normally used to grasp small finger holds, involves a load 36 times greater for the A2 pulley compared to the slope grip.[27] Furthermore, the force incurred by the distal part of the A2 pulley is threefold that supported by the fingertip.[28] Generally speaking, climbers support a load of 380 N per finger, yet a 70-kg climber in a single-finger grip may support as much as 450 N, exceeding the reported maximum tolerated load of 380 to 400 N for the A2 pulley.[1] Thus, to support this load, a prior period of adaptation is needed.[1] Accordingly, we would expect anatomic and structural modifications in the fingers allowing climbers to adapt to these biomechanical demands. This is consistent with reported adaptive morphologic and radiologic modifications in bone, A2 and A4 flexor tendon pulleys, and volar plates of the interphalangeal joints in high-level climbers without injury symptoms.[7,12]

US TBD THRESHOLD

To diagnose a complete A2 or A4 pulley system rupture, a US TBD of 2 mm has been described in the literature as the best cutoff.[29] However, it should be noted that false positives have been detected among asymptomatic nonclimbers when using this threshold.[30] Although in the present study, this cutoff value was not reached or exceeded in any of the pulleys for the control group, this did occur in 19% (1.5±0.5 mm, 0.9–3.3 mm) of A2 pulleys and 21% (1.6±0.4 mm, 1–3 mm) of A4 pulleys (20% of total pulleys) in the asymptomatic experienced high-level climbers group. Thus, the latter could be considered complete pulley ruptures.[19] A different TBD threshold value that has been proposed, also using active forced flexion finger position, is 5 mm.[20] This value was not reached in any of the measured pulleys. All in all, not always will a climber with a TBD considered diagnostic be symptomatic, although the sum of both factors (pain and elevated TBD) along with a reduction in finger grip strength or other accompanying US signs will suggest the diagnosis of a ruptured pulley.[17]

EXPERIENCED HIGH-LEVEL CLIMBERS TBD VS NONCLIMBERS TBD

Our findings reveal a longer TBD in the A2 and A4 finger pulleys of experienced climbers without symptoms of injury compared to matched healthy nonclimbers. It is certainly feasible that the 25% and 35% longer TBD for A2 and A4 pulleys, respectively, could be part of an adaptive mechanism related to long periods of climbing, as for other adaptive modifications that have been described in bone and soft tissues in this population.[7,12] Another issue worth considering is the possibility of histoarchitectural failure in the retention capacity of the pulley system, or even the formation of small subclinical ruptures over time, as occurs in chronic tendinopathies involving degeneration and intrasubstance tears of the tendon.[31] Future studies should determine whether this increase in TBD occurs progressively as an adaptation process or occurs suddenly after an acute period of

overloading the fingers due to an increased workload, the latter being more suggestive of underdiagnosed pulley tear.

Bowstringing and thickening as physiological adaptations or subclinical entities of A2 and A4 pulleys show similar trends. Pulley thickness was found to be significantly higher in climbers vs nonclimbers,[7] and in the present study the same trend was found for TBD. A4 showed greater changes when submitted to longstanding overloading, with 75% thickening and 35% bowstringing in comparison to A2 (69% and 25%, respectively). Thus, the annular pulleys show a significant tendency to both thicken and slacken.[7] Overall, what seems important about a synchronic thickening and slackening process is that, if a pulley thickens without gaining laxity, the space between the flexor and pulley would be reduced, increasing friction and giving rise to the condition of trigger finger.

A2 TBD VS A4 TBD

We report an overall 10% higher TBD in A4 pulleys compared to the A2 pulleys analyzed, although the difference is not statistically significant. One hypothesis to explain this is the difference in the breaking load between pulleys. The mean perpendicular forces acting on A2 and A4 pulleys were approximately equal (254.8 N and 220.9 N) when performing a crimp grip.[27] The maximum breaking load is 407.5 N for the A2 pulley and 209.5 N for the A4 pulley, which means that A2 is twice as strong as A4.[32] In a maximum crimp grip load, A2 can easily accommodate such forces, whereas A4 is at the limit of its load accommodation capacity. According to this rationale, it is easier for a climber to rupture an A4 pulley than A2 pulley.[33] However, over a minimum of 15 y sustaining high-load repetitive crimp grips and without any pulley rupture, greater asymptomatic bowstringing could be more likely in the A4 pulley than in the A2 pulley.

No difference in TBD was noted here between the dominant and nondominant hand in both climbers and nonclimbers. This was not unexpected because climbing is a relatively symmetric sport.

LIMITATIONS

The main limitations of this study were its small sample size, the lack of female participants, and the absence of intraobserver or interobserver variability. A drawback was that no previous medical records were reviewed when applying the exclusion criteria regarding previous diagnosis of pulley rupture of the ring finger or hand surgery; this information was obtained by interviewing the sample participants, and this might have incurred a self-reporting bias. Therefore, a new study should be developed with the purpose of determining the factors that may modify TBD.

Conclusions

Asymptomatic experienced high-level climbers have significantly longer A2 and A4 flexor pulley TBDs (25% and 35%, respectively) compared to asymptomatic nonclimbers**.** Such differences suggest a subjacent adaptive process to elevated workloads over the years or the possibility of underdiagnosed annular pulley ruptures. The present results should be taken into consideration by sonographers when performing A2 or A4 pulley US assessments in experienced high-level climbers to avoid false positives. The 2 mm TBD threshold to diagnose a complete A2 or A4 pulley rupture could be excessively sensitive for such climbers.

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ORIGINAL RESEARCH

Human-Factor Risk Mitigation in Outdoor Climbing Areas: Survey of Existing Policies in Regulated Climbing Areas

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Introduction—Popular climbing areas in the United States are frequently on public lands under administrative purview of land management entities. During climbing, climbers may be subject to injury. This study aimed to describe existing climbing risk mitigation tactics used to protect climbers by oversight agencies responsible for these public climbing areas and to identify strategies perceived by the oversight agency to be successful.

Methods—We identified publicly managed US bouldering, sport, or traditional climbing areas through a state-by-state search of Mountain Project. We assessed climbing-related visitation, injury, rescue, and risk mitigation strategies using a 10-item survey targeting land-use managers, rangers, outdoor recreation planners, and park managers. Quantitative analysis included univariate and multivariate analysis. Qualitative analyses of survey responses with thematic grouping of mitigation interventions were performed.

Results—One hundred fifty-seven publicly managed US climbing areas were contacted, and 76 (48%) provided data. Thirty-six (47%) of those that provided data stated that programs are in place to reduce climbing-related injury. There were no associations between demographic variables and the presence of risk mitigation strategies. Four themes of climbing risk mitigation strategies emerged: coordination with climbing coalitions (25% of respondents), permitting (22% of respondents), publication of accident reports (22% of respondents), and preventative search and rescue (17% of respondents).

Conclusions—Nearly half of survey respondents reported having climbing risk mitigation programs. There is opportunity to assess the efficacy of risk mitigation strategies through intra-area and inter-area assessments. A centralized climbing injury database may prove useful for assessing the efficacy of and need for risk mitigation techniques.

Keywords: preventative search and rescue (PSAR), injury prevention, National Parks, public lands

Introduction

Rock climbing, mountaineering, and indoor rock climbing are increasingly popular recreational activities in the United States, with over 6 million participants annually.[1-3] Climbing is increasingly accessible to the general population, and participants are transitioning from climbing indoors to climbing outdoors.[1] A range of injuries can occur while climbing, from falls to stress-related musculoskeletal injuries.[1,4] Climbers may also be at risk for exposure-based conditions, including hypothermia, sunburn, and dehydration, depending on conditions and climber preparedness.

Although climbing can be done anywhere rocks are present, popular climbing areas in the United States frequently fall under the administrative purview of the National Park Service (NPS), Bureau of Land Management (BLM), US Department of Agriculture Forest Service (USDAFS), state parks, or city or county parks. There are limited published data describing climbing risk mitigation tactics used by these oversight entities to reduce rates of climbing-related injury and rescue. We sought to describe existing risk mitigation tactics used by oversight agencies responsible for these public climbing

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areas, with the goal of identifying strategies perceived to be successful.

Methods

Research protocols were approved by Stanford University and determined to be exempt after institutional review board review. We identified climbing areas for study via a systematic state-by-state search of the Mountain Project. The Mountain Project is a widely used, regularly updated website serving as a free online guidebook for over 170,000 climbing routes worldwide, relying on regional administrators and real-time user-generated beta, photos, topographic maps, and comments. Navigating through the home page for each state, we captured publicly managed (under the purview of the NPS, BLM, USDAFS, and local city, county, and state park services) climbing areas. With further review of climbing areas, we excluded areas that primarily contained mountaineering or ice climbing routes, aiming for a sample of areas with a predominance of bouldering, sport, or traditional climbing.

Through area web pages (eg, general NPS, BLM, USDAFS, and local city, county, and state park services) and outreach to area ranger stations and offices, we collected contact information for individuals and/or offices with knowledge of or responsibility for climbing-specific risk and objectives in the area. We made ≥3 separate attempts—over at least 2 forms of communication (phone and email)—to contact the individual or office of interest at each area. Once contact was made, we offered interviewees the option of anonymity and the choice to complete our survey either online or by phone.

Our 10-item survey was designed to assess climbing-related visitation, injury, rescue, and risk mitigation strategies. Quantitative data obtained included climbing-specific visitor volumes, frequency of climbing-related injuries, and frequency of climbing-related rescues. Qualitative data collected included climbing-specific risks, tools used to minimize climbing-related injury, and area-specific concerns to provide context for risk mitigation tactics. Survey application and data collection ranged from April 1, 2020 to June 30, 2020.

For quantitative data analysis, we categorized continuous data into high, medium, and low. The visitation index was derived from stratifying reported annual climbing visitation from each area, using logarithmic numerical cutoffs: low (≤500 climbing visitors), medium (500–7000 climbing visitors), and high (>7000 climbing visitors). Land area (in square kilometers) was defined as the complete area of a land management track obtained from official park reports or the GeoHack GIS aggregator.[5-10] Cutoffs were created to reflect natural clustering of the data set: small (<9.5 km^2), medium (9.5–450 km^2), and large (>450 km^2). Regions were defined by the 9 US Census Divisions.[11] A composite risk index (reported as low, medium, or high) was generated by stratifying ratios of injuries and rescues to climbing visitors using logarithmic numerical cutoffs. Groups were as follows: low (<10 risk events [rescue and/or injury] per 10,000 climbing visitors), medium (10–14 risk events per 10,000 climbing visitors), and high (>14 risk events per 10,000 climbing visitors).

Data are presented as median with range. Univariate analysis was performed using the Fisher exact test. Multivariate logistic regression was used to assess predictors of the existence of risk mitigation plans using predetermined quantitative variables, including composite risk index, climbing region, visitation volume, and land area. The a priori *P*-value considered significant was $P \leq 0.05$. For qualitative data, we examined recurrent themes and identified choice quotes from survey respondents encapsulating approaches to risk management, obstacles faced, and tools to administer climbing risk mitigation programs and protocols. We used Stata 15.0 (StataCorp, College Station, TX) for analysis.

Results

We identified 159 climbing areas that met our inclusion criteria. Of these, 2 areas were removed from further evaluation because climbing was officially banned. We did not receive responses from 51 areas (32%). One hundred six (68%) responded, each representing a separate climbing area. Of those, 76 (72%) provided data and 30 (28%) refused to provide quantitative data in response to the survey. Thirty-four (45%) of the 76 that provided data answered 100% of the survey questions. The most common job title of the survey respondent (20 of 76; 26%) was ranger (including chief rangers, district rangers, lead and supervisory climbing rangers, backcountry rangers, and interpretive rangers). Areas of study were distributed across 9 US Census Regions (Figure 1).

Among 39 areas that reported risk-related data, there were 2.1 injuries per 10,000 climbing visitors (range: 0–50) and 0.03 injuries per 10,000 annual visitors (range: 0–8.6). There were 1.1 rescues per 10,000 climbing visitors (range: 0–114) and 0.02 rescues per 10,000 annual visitors (range: 0–0.2). The median land mass of climbing areas of study was 64.8 km^2 (range: 0.02–24,280). There were no associations between

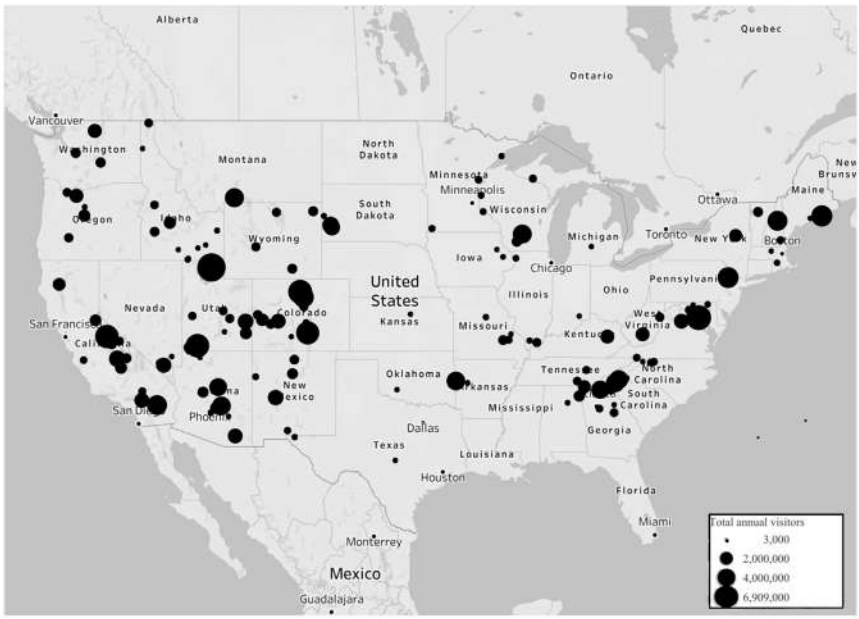

Figure 1. Map of 157 climbing areas of study. Map based on longitude and latitude. Total annual visitation (range: 3000–6,909,000) is scaled in the size of dots.

demographic variables and the presence of risk mitigation strategies (Table 1).

Respondents had heterogeneous responses regarding common geospatial locations of injury. Forty-one percent (31 of 76) stated they were unaware of common locations. Some reported this was based on a general lack of information about climbing-related injury and rescue in their area of governance. Others stated that an overall scarcity of climbing-related injury and rescue rendered them unable to identify any common location-based trends. Among respondents who reported trends in climbing-related injury, 13% (5 of 38) stated more difficult climbing–particularly overhung climbing of high grades (5.11^{+})–contributed to climbing-related injuries and rescues. Two respondents reported increased difficulty of accessible climbing instead contributed to lower rates of injury and less need for rescue. Similarly, 4 respondents (11%) stated that injuries were more common in areas with moderate grades (5.6–5.11) and easy climber access.

RISK MITIGATION STRATEGIES: OVERVIEW

Thirty-six respondents (47%) stated they have programs in place to reduce climbing-related injury and rescue. Four themes of climbing risk mitigation strategies emerged: coordination with climbing coalitions, permitting, preventative search and rescue (PSAR), and publication of accident reports.

COORDINATION WITH CLIMBING COALITIONS

Twenty-five percent (9 of 36) of responding areas' risk mitigation strategies involved coordination with climbing coalitions or alliances. Relationships with climbing coalitions varied, but commonalities included creation of climbing management plans and maintenance of effective communication with the climbing community. These climbing management plans frequently had bolt replacement and trail maintenance components. For instance, a county-managed climbing area in Iowa required their local climbing coalition to perform annual climbing infrastructure (eg, anchors) inspections and provide approval of new routes or infrastructure before route establishment.

Even when formal climbing management plans were not in place, climbing coalitions commonly served as a conduit between administrative agencies and climbing communities. Three respondents (8%) explained that information on bolt and anchor replacement was collected through social media pages (Facebook and Mountain Project discussion boards) operated and monitored by climbing coalitions. Eleven respondents (30%) described climbing area managers engaging in dialogue with climbing coalitions for notification about worn hardware, new hardware proposals, and hardware replacement. Survey respondents reported relying on climbing coalitions to communicate safety information to climbing visitors. Five respondents (14%) stated that climbing coalitions were a "means of stay[ing] connected with

Table 1. Presence of risk mitigation strategies assessed by demographic variables

Variable	*All climbing areas*	*Climbing areas with survey response*		
	n=157	*All areas n=76*	*Areas with risk mitigation n=36*	P-*value*
Land area				0.2
Small	48	25	14	
Medium	58	25	12	
Large	51	26	10	
Region				0.4
New England	11	2	2	
Middle Atlantic	4	1	0	
East North Central	8	1	0	
West North Central	17	6	4	
South Atlantic	23	14	6	
East South Central	7	3	1	
West South Central	5	3	3	
Mountain	58	40	17	
Pacific	21	6	3	
Visitation				
Low	–	9[a]	3[b]	0.3
Medium	–	16[a]	11[b]	
High	–	9[a]	5[b]	
Composite risk index				
Low	–	13[a]	4[b]	0.4
Medium	–	11[a]	7[b]	
High	–	10[a]	8[b]	

[a]The areas represented in these category totals are only those with a complete (100% of questions) survey response (n=34).
[b]The areas represented in these category totals are only those with a complete (100% of questions) survey response (n=19).

climbing communities" to ensure bidirectional communication. Two climbing coalitions were reported to run volunteer injury and rescue groups and provide education and training for climbers. Finally, 6 respondents (20%) noted that local sports stores and local college- or university-led climbing groups were key resources for knowledge building and education around safe climbing.

PERMITTING

Eight respondents (22%) included permitting systems as part of risk mitigation strategies. These respondents all reported that safety information on climbing risks and hazards was featured on climbing permits: part of their permit process required acknowledgement of risks and attestation prior to permit release. Certain areas, notably the Black Canyon of the Gunnison, require permits for all climbing. Two respondents noted that their areas only require permits for high-risk objectives. Two areas have separate sign-in processes at visitor centers, which includes signing liability waivers. This process enables them to create daily climber lists with locations to assist rangers. Three respondents (8%) noted that permits are required for establishment of new routes—particularly if climbers intend to place new hardware.

Ten respondents (28%) stated that commercial climbing supervised by outside guiding companies and organizations required permits. Two respondents (6%) organized their own site-specific guide training courses. Official policies were reported across all entities managed by NPS and the USDAFS—with a few key exceptions—that required permits for guiding. It was also reported that federal policies govern the placement of fixed anchors or other forms of new hardware in designated wilderness areas managed by the NPS, USDAFS, and BLM.[12] Prior authorization, provided by a park plan or case-by-case climber requests, is required for placement of new fixed anchors.[12-14] For replacement of anchors in need of maintenance, authorization is only required in certain cases on a park-determined basis.[15]

PREVENTATIVE SEARCH AND RESCUE

Six respondents (17%) cited PSAR as a risk mitigation strategy. Out of respondents who mentioned PSAR by name, 33% (2 of 6) referred to technical skill training and practice as PSAR, and 33% cited conversations between rangers and climbers. The latter touched on a larger risk mitigation theme involving making ranger staff available for conversations. This came in the form of "Coffee with

a Ranger" programming and ranger-led efforts to contact climbers before they head out to climb. Some examples include climbing rangers contacting climbers in the mornings before they head out for their climbs, advising climbers on route selection, providing information on route closures, and offering technical tips. Six respondents (20%) explained how conversational-based PSAR can arise from more traditional patrol-based search and rescue models. Six respondents (20%) emphasized the importance of the technical training local search and rescue organizations undergo as a means to bolster their climbing risk mitigation response.

PUBLICATION OF ACCIDENT REPORTS

Respondents also reported publicly publishing data on climbing-related injury and rescue as a method of risk mitigation. For example, respondents from El Dorado Canyon SP, Colorado, said they directly share accident data, new regulations, and safety concerns on educational signage in the park. Yosemite NP engages in a similar approach wherein they publish selected search and rescue incident reports on their website bimonthly during the peak season (roughly March to November). Thirty-four areas of study (22%) were represented in accident reports submitted to the 2020 *Accidents in North American Climbing* by rescuers, rangers, and climbers.[15]

LIMITATIONS TO CLIMBING RISK MITIGATION

Ten respondents (13%) endorsed having a fear of creating risk mitigation strategies, because this would inherently acknowledge a governing entity was condoning a risky sport, thus potentially exposing them to litigation. Three respondents (4%) only agreed to complete the survey if guaranteed anonymity in their responses out of fear for litigation. Eight respondents (11%) felt "torn" about the roles of the land management organizations—seeing their formal role as to protect resources, not individuals. Twenty-one respondents (28%) lamented the absence of standard national systems for compiling and reporting climbing-specific injury and rescue data. Respondents stated that without formal reporting mechanisms, many injuries are unaccounted for, particularly when climbers self-rescue. Three respondents reported that climber reticence to disclose events resulting in injury might jeopardize future access to climbing routes.

Discussion

As rock climbing has become more popular, rates of climbing-related injury and rescue have increased.[1-3] Interventions capable of reducing the opportunity for injury and need for subsequent rescue are urgently needed. In surveying popular, publicly managed bouldering, traditional, and sport climbing areas, we found only 47% had programs in place to prevent climbing-related injury and rescue. These programs employed a range of risk mitigation strategies, with 4 themes: coordination with climbing coalitions (25%), permitting (22%), publication of accident reports (22%), and PSAR (17%). Identifying successful risk mitigation strategies, naming challenges, and sharing this information may help ensure useful risk mitigation techniques are adopted broadly.

Engagement with climbing coalitions was reported as advantageous by management entities. Coalitions provided an opportunity to outsource climbing-related risk mitigation efforts, while connecting and aligning management efforts with the needs of the local climbing community. For climbing areas that do not employ climbing-specific rangers, working with climbing coalitions may help ensure that climbing-related concerns and interests are communicated between climbers and land management organizations. These relationships may also provide opportunities for bidirectional information flow: Management entities can use coalitions to inform climbers of regulations and simultaneously receive information from climbers on successful or unsuccessful risk mitigation techniques. An additional benefit of partnering with climbing coalitions could be increased communication with other local entities, such as county search and rescue, which could be brokered through the climbing coalition.

Another commonly reported tactic was permitting. Through development of a permit system, management entities gain perspective on the frequency and volume of climbing use and a roster of climbers in an area. Such a technique may be particularly advantageous in areas where longer or more remote climbs are common, or conversely in areas or on routes with particularly high climber traffic. Having an in-person permit system also provides an opportunity for climber education. When picking up a permit, climbers may have the opportunity to talk with a ranger about their objective and gain information or insight that they might not have otherwise received. This process also serves to develop a personal connection between climbers and the personnel responsible for managing a given land area.

Likewise, the process of sharing accident or rescue data can help inform the climbing community about risks in a given area. However, it was clear from the survey respondents that there is no well-defined mechanism for public management entities to report these accidents, nor is there a central, publicly managed repository where these injuries can be compiled. Development of an anonymized, digital, curated injury repository may prove to

be particularly useful. Building on the success of *Accidents in North American Climbing*, national climbing advocacy groups, public oversight agencies, or wilderness medicine organizations should be encouraged to consider such infrastructure to help guide assessments of climbing risk mitigation techniques.

LIMITATIONS

This research has several limitations. One limitation is in our modest response rate, which is subject to selection and nonresponse bias. There is also potential for recall bias given the survey design. Responses may have been biased due to the method of survey administration, with phone surveys biased toward socially desirable answers. Having individuals provide responses on behalf of land management organizations invited a proxy respondent effect. To encourage diverse responses reflecting strategies in the working context of specific areas, we allowed respondents to define risk mitigation programs on their own terms, which increased subjective variability. We were not able to quantify the amount that a given oversight entity depended on any given risk mitigation strategy, just its presence or absence. Finally, incidents in which climbers self-rescue were not captured in reported injury and rescue numbers.

Conclusions

Forty-seven percent of publicly managed climbing areas responding to our survey reported the presence of programs to prevent climbing-related injury and rescue. Existing programs involve coordination with climbing coalitions, permitting, preventative search and research, and publishing injury and rescue reports. There is considerable opportunity to evaluate the efficacy of risk mitigation strategies through intra-area and inter-area assessments. A centralized climbing injury database may prove useful when assessing the impact of risk mitigation techniques.

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ORIGINAL RESEARCH

The Associations Between Visitation, Social Media Use, and Search and Rescue in United States National Parks

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Introduction—Search and rescue (SAR) is vital for visitor safety in US national parks, which are popular destinations for tourists. Previous studies have described SAR and seasonal visitation patterns, but not in the context of overall visitation. In addition, studies on the association between SAR and developed park areas remain limited. Concurrently, social media can be valuable for sharing information about conservation awareness and the joy of being outdoors. However, social media can potentially be an avenue for users to share risky and dangerous behaviors performed during attempts to obtain photos and videos. The associations between SAR and social media have not been discussed in existing literature.

Methods—Variables included recreational visits, developed site stay visits, backcountry visits, SAR incidents, and tweets. Data from 2017 were obtained from National Park Service visitor use statistics, the SAR incident dashboard, and the University of California, Irvine, Cloudberry application. Correlation analysis was performed using nonparametric Kendall rank correlation coefficients.

Results—Recreational visits were correlated with SAR incidents (r_τ=0.415, P<0.001). Developed site stays were similarly correlated with SAR incidents (r_τ=0.447, P<0.001), as were backcountry visits (r_τ=0.428, P<0.001). Backcountry visits had a stronger correlation with fatalities (r_τ=0.380, P<0.001) compared to developed site stays (r_τ=0.304, P<0.001). Tweets were correlated with SAR incidents (r_τ=0.468, P<0.001), recreational visits (r_τ=0.403, P<0.001), and fatalities (r_τ=0.367, P<0.001).

Conclusions—Our findings demonstrate associations between national park visitation, SAR incidents, fatalities, and Twitter use and provides a concept framework for future prospective studies to further investigate the relationships between visitation, SAR, and social media.

Keywords: wilderness, recreation, technology, internet, emergency medical services, public health

Introduction

Search and rescue (SAR) in US national parks is vital for visitor safety. Public safety in national parks is subject to a changing visitation landscape, with the National Park Service reporting over 300,000,000 visits to parks every year since 2015 (National Park Service, 2020). SAR and seasonal visitation patterns have been previously studied, but associations between SAR and overall park visitation remain limited.[1-4] In addition, few studies discuss the associations between SAR and developed park areas.[5,6]

At a time of marked national park popularity, social media use has become a common avenue for sharing outdoor experiences. Associations between social media posts and visitation to public lands have been well documented.[7-10] In contrast, the literature surrounding social media use and public safety in national parks is limited, although there have been many reports of outdoor injuries related to social media, such as falls from cliffs while taking selfies.[11,12] We sought to research the associations between national park visitation, accessibility, SAR incidents, and social media use. Our study used Twitter, a social media platform that enables users to share their experiences through 280-character "tweets" with attached photos and videos, to investigate the associations between social media and public safety in national parks. We hypothesized that recreational visits are associated with SAR incidents, developed site stays are more strongly associated with SAR incidents compared to backcountry visits, backcountry visits are

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Table 1. Selected national park units that retained the "national park" appendage for Twitter search keywords

Zion National Park	Manassas National Battlefield Park
Olympic National Park	Padre Island National Seashore
Buffalo National River	Kings Mountain National Military Park
Big Bend National Park	Pecos National Historic Park
Acadia National Park	Antietam National Battlefield
Arches National Park	Petersburg National Battlefield
Point Reyes National Seashore	Colonial National Historic Park
Ozark National Scenic Riverways	Gettysburg National Military Park
Colorado National Monument	Great Basin National Park
Badlands National Park	Biscayne National Park
Amistad National Recreation Area	Chickasaw National Recreation Area
Redwood National Park	Cabrillo National Monument
Mojave National Preserve	Theodore Roosevelt National Park
Hot Springs National Park	Dinosaur National Monument

more strongly associated with fatalities than are developed site stays, and tweets are associated with SAR incidents.

Methods

Our study was considered exempt research by the University of California, Irvine institutional review board given that it does not involve human subjects research. For simplicity, the term "national park" in our study includes all individual units managed by the US National Park Service, including national parks, recreation areas, seashores, and lakeshores. Visitation data were obtained from the National Park Service visitor use database using the query builder for public use statistics tool. Search variables included 2017 calendar year recreational visits, concessioner lodging, concessioner camping, tent campers, recreational vehicle campers, and backcountry campers. Backcountry is defined as primitive or wilderness areas that are reached primarily by hiking, boating, or horseback. Developed site stays in our study are defined as the sum of tent campers, recreational vehicle campers, concessioner lodging, and concessioner camping. We made the assumption that developed site stays occurred in more easily reached areas of parks, and backcountry visits occurred in more remote areas.

SAR data were obtained from the US National Park Service SAR Incidents 2017 Dashboard, which provided a comma-separated-value dataset that includes reporting national park units compiled by the law enforcement, security, and emergency services department of the National Park Service. Our study used the number of SAR incidents and fatalities from this dataset. Our inclusion criteria required a park to have a sum of total dollars spent for SAR operations, SAR hours, and SAR costs greater than 0. The assumption was made that parks with 0 SAR costs, 0 operational hours, and 0 SAR incidents did not have their own dedicated SAR unit; these were excluded in the study. Of the 176 national park units included in the SAR dataset, 127 met the inclusion criteria for our study. Four of those parks included did not report fatalities and were excluded from the fatality analysis.

Twitter data were compiled on October 11, 2020 from the University of California, Irvine, department of information and computer sciences Cloudberry application, which collects Twitter post counts based on keyword, hashtag, and geographic data. For 99 parks, the keyword used was the official name of the national park unit, excluding the "national" unit (eg, "Yosemite" and "Grand Canyon" vs. Yosemite National Park and Grand Canyon National Park), which yielded an application-generated list of related keywords that were included in the total tweet count. For the remaining 28 parks, we made the decision to retain the full name of the park

Table 2. Correlations of recreational visits, developed site stays, backcountry visits, and tweets with search and rescue incidents and fatalities

Dependent variables	*Independent variables*			
	Recreational visits	*Developed site stays*	*Backcountry visits*	*Tweets*
SAR incidents				
r_τ	0.415	0.447	0.428	0.468
P-value	<0.001	<0.001	<0.001	<0.001
Fatalities				
r_τ	0.340	0.304	0.380	0.367
P-value	<0.001	<0.001	<0.001	<0.001

SAR, search and rescue; r_τ, Kendall's tau correlation coefficient.

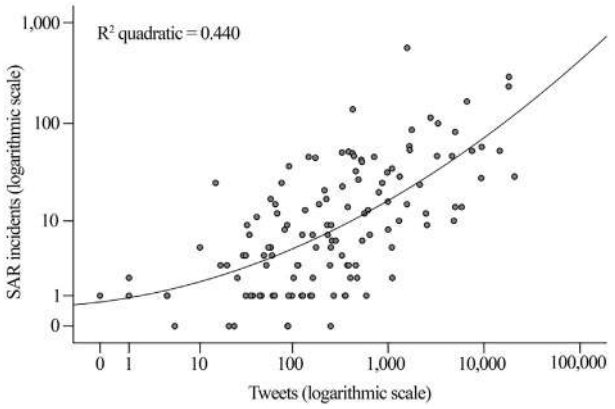

Figure 1. Correlation between tweets and search and rescue incidents.

because excluding the national park appendage would yield too broad a search term (eg, "Glacier National Park" was searched instead of "Glacier"). The parks that retained the national park unit designation in the search query are listed in Table 1. Tweets were included from January 1, 2017 through December 31, 2017 in the United States.

Independent variables were recreational visits, developed site stays, backcountry visits, and tweets. Dependent variables were SAR incidents and fatalities. Because the data were not normally distributed, the associations were analyzed using the Kendall tau correlation coefficient. A correlation was considered statistically significant if $P<0.05$. Kendall tau value interpretations were defined as the following: 0 to 0.10 suggests a weak correlation, 0.11 to 0.30 suggests a weak to moderate correlation, 0.31 to 0.49 suggests a moderate to strong correlation, and 0.50 or higher suggests a strong correlation. SPSS Statistics version 26 (IBM SPSS Statistics for Windows, Version 26.0. Armonk, NY) was used for the analysis.

Results

We analyzed the correlations between 180,216,375 recreational visits, 10,452,835 developed site stays, 1,956,935 backcountry visits, 183,744 tweets, 3,433 SAR incidents, and 181 fatalities that occurred in national parks during 2017. Recreational visits were correlated with SAR incidents ($r_\tau=0.415$, $P<0.001$). Developed site stays and backcountry visits both were correlated with SAR incidents ($r_\tau=0.447$, $P<0.001$ and $r_\tau=0.428$, $P<0.001$, respectively). Backcountry visits had a stronger correlation with fatalities than did developed site stays ($r_\tau=0.380$, $P<0.001$ vs $r_\tau=0.304$, $P<0.001$). Tweets were correlated with SAR incidents ($r_\tau=0.468$, $P<0.001$), recreational visits ($r_\tau=0.403$, $P<0.001$), and fatalities ($r_\tau=0.367$, $P<0.001$) (Table 2).

Discussion

Our large database study found that recreational visits, developed site stays, and backcountry visits were correlated with SAR incidents and fatalities. In addition, developed site stays and backcountry visits had a similar correlation strength with SAR incidents. However, backcountry visits had a stronger correlation with fatalities than did developed site stays. Lastly, we found that tweets were correlated with SAR incidents, recreational visits, and fatalities.

There was a moderate to strong association between recreational visits and SAR incidents. Previous studies have identified an increased number of SAR incidents during peak visitation times such as the summer season and weekend days.[1-3,13] In regard to park popularity and SAR incidents, 4 popular national parks in California accounted for 83% of the state's national park emergency medical services workload.[14] Our results and the existing literature show that visitation may be a helpful marker for park officials to predict SAR demand. Future studies can benefit from focusing on specific popular attractions within parks and SAR incidents associated with these attractions.

Relative ease of access to park areas also has the potential to be associated with safety. We proposed that developed site stays take place in more easily reached areas of parks compared to backcountry visits and hypothesized that developed site stays would be more strongly associated with SAR incidents. Surprisingly, we found that visits to developed and remote areas of parks were similarly associated with SAR incidents. Our original hypothesis was based on a study in Sequoia National Park showing that 81% of 704 rescue incidents occurred within 1 mile of a paved road.[5] In comparison, another study demonstrated that the highest number of medical and trauma events in the national park system occurred in the Intermountain Region and suggested that the higher prevalence of calls for rescue in this region was related to the dangers posed by challenging terrain and minimal shelter from the elements.[6] Backcountry visits inherently carry more risks to safety related to environmental unpredictability and topographic extremes.[15-17] The implications of experience, judgment, and preparedness are extensively documented in the SAR literature.[18-21] The increased dangers of backcountry excursions require a higher level of knowledge and preparedness that may attract the more adept outdoorsperson, who may be less likely to call for rescue in the event of minor setbacks or deviations from plans. In contrast, developed areas of parks may have a

higher proportion of inexperienced visitors, who may be more likely to call for rescue in less urgent or unnecessary circumstances. This observation was demonstrated in multiple SAR studies that found a lack of preparation and errors in judgment to be associated with SAR burden.[18,19] Regarding life-threatening cases, we expectedly found that backcountry visits were more strongly correlated with fatalities compared to developed site stays. A potential mechanism for our finding may be related to both the inherent dangers of the backcountry and the possibility that calls for rescue in the backcountry are made by more experienced and critically injured visitors. Notably, our study did not account for the possibility of less experienced visitors engaging in backcountry activities or more experienced visitors engaging in activities in developed areas. SAR can benefit from future studies that specifically examine and compare the experience levels of those who call for rescue in the front and backcountry.

Tweets were found to have a moderate to strong association with recreational visits. This finding is consistent with the existing literature on social media and outdoor space popularity.[7-10] A study that included outdoor recreational spaces from around the world found strong associations between social media posts and recreational space popularity.[8] Correlations between social media and visits to national forests have also been identified.[22] Regarding public safety, the strongest correlation in our study was between tweets and SAR incidents (Figure 1). There are likely several mediators that contribute to this association, such as an increased number of visitors posting to social media and increased visitation overall being related to SAR incidents, as identified in our study. However, we suggest that social media may be associated with SAR incidents via users motivating others to obtain photos or videos through risky or dangerous means. Existing studies have demonstrated the dangers associated with social media–influenced trends in the outdoors. For example, one study described many cases of selfie-related injuries, including wild animal attacks.[12] A study of similar photo-taking behaviors associated with injury found that the most preferred site of photography was on cliff edges, and the most frequently reported injury was falling from heights.[11] Furthermore, injury, risk-taking behavior, and social media have been described in other contexts. One study found that motivations behind engaging in risky driving demonstrated in videos shared on YouTube, a popular social media video-sharing platform, included factors such as financial reward for garnering higher view counts and peer pressure.[23] We hope that this initial finding of a moderate association between tweets and SAR incidents may be explored in future studies and is useful for park administrators seeking an easily trackable proxy to predict SAR need.

LIMITATIONS

The retrospective design of our study prevents assumptions of causality between the studied variables. Potential confounders such as weather, geography, and cell phone reception were not accounted for in our analysis. Furthermore, our results are shown as nonparametric correlation coefficients because the data were not normally distributed. This limited our ability to yield valid results from a multivariate analysis given that the assumptions required for such analysis, including normal distribution of data and linear relationships, were not met. In addition, we have identified that variables such as tweets and recreational visits may be collinear, and results from a multivariate analysis would potentially be inflated. Because we used databases, the data used to obtain results may be subject to input errors and may not include the entirety of available data. We recognize the potential for bias in the selection of national parks that retained the full national park appendage in the keywords used for tweet searches. Demographic data of users posting to Twitter were not included in the study, and social media use in the context of outdoor recreation may be skewed toward certain populations. Furthermore, we used only Twitter as a measure for social media use, and other social media platforms such as Instagram and Facebook may yield different results in comparing correlations with park visitation and SAR.

Conclusions

Our study found that visitation was associated with SAR incidents, developed and backcountry areas had similar associations with SAR incidents, backcountry areas had a stronger association with fatalities compared to developed areas, and tweets were associated with visitation and SAR incidents. The evolving visitation and social media landscape in national parks warrants further prospective investigation. It is paramount to continue adapting to both the physical and digital landscape of outdoor recreation to ensure the safety of all who take part in the enriching experiences of the great outdoors.

Author Contributions: Study concept and design (ZL); acquisition of the data (ZL); analysis of the data (SS); drafting of the manuscript (ZL, AB); revision of the manuscript (ZL, AB, IAG); and approval of final manuscript (ZL, AB, SS, IAG).

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ORIGINAL RESEARCH

Tree Stand–Related Injuries in Nonadmitted and Admitted Patients at a Level 2 Trauma Center in Michigan: 2015–2019

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Introduction—Tree stand–related injuries (TSRI) are more common than firearm-related injuries in hunters. Most previous studies on the topic used trauma databases that only include admitted patients. This study characterizes injury patterns found in nonadmitted and admitted TSRI patients presenting to a level 2 Michigan trauma center. TSRI prevention interventions are also discussed.

Methods—Cases were obtained through a retrospective chart review of the Henry Ford Allegiance Health trauma registry and EPSi cost-accounting database from 2015 to 2019. Keywords searched included fall, hunter, tree, tree stand, treestand, ICD 9 diagnosis (*E884.9_Other accidental fall from one level to another*), and ICD 10 diagnosis (*W14.XXXA_Fall from tree, initial encounter*). We analyzed age, sex, body mass index, injury severity score, disposition, alcohol use, injuries sustained, reported height of fall, and narrative of fall.

Results—Thirty-three patients were identified. Patient age was 45±13 y (mean±SD). All patients were male. Injury severity score for nonadmitted patients was 2±1 vs 13±11 in admitted patients. Thirty-three percent of cases were nonadmitted; 67% were admitted. The most common injuries sustained were spinal (33%) and lower extremity fractures (15%). The average yearly TSRI case rate was 5.73 per 10,000 hunters in the study area.

Conclusions—Our study found that spinal and lower extremity fractures were the most common injuries sustained. One-third of our patients were nonadmitted and therefore not included in the Henry Ford Allegiance Health trauma registry. Some nonadmitted patients had significant injuries requiring specialist consultation, orthopedic braces, or outpatient surgery.

Keywords: fall, hunting, hunter, elevated platform, trauma

Introduction

Hunting remains a popular avocation for many Americans. According to the National Survey of Fishing, Hunting, & Wildlife-Associated Recreation, it is estimated that between 5 and 8% of all Americans age 16 y and older participate in hunting.[1-3] Percentages vary by geographic region of the United States and year.[1-3] Recent decades have shown a decline in annual hunting participation prevalence estimates. From 2006 to 2016, the number of hunters decreased from 12.5 million to 11.5 million, with a drop in big game hunters from 10.7 million to 9.2 million.[1-3] Deer hunters comprise the overwhelming majority of not only big game hunters, but also US hunters in general.[1-3] These national surveys characterize the archetypal hunter as a middle-aged white male who began hunting in his youth. However, in the past 2 decades the pursuit has gained popularity among women and hunters who adopt the pastime as adults.[1-3]

Tree stands are predominantly used to hunt big game animals, particularly deer. Tree stands confer an advantage to the hunter through scent dispersal, reduced visibility by game, and increased field of view. Stand use is prevalent in midwestern, eastern, and southeastern states owing to the type of habitat available for hunting (eg, agricultural fields, forest). There are 5 main tree

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Figure 1. Bowhunter preparing a hang-on platform tree stand for deer season. Note the hunter's safety harness and attachment to tree near upper back. Photo courtesy Philip J. Lazzara.

stand categories: homemade wooden platforms, metal ladder stands, 2-piece metal climbers, hang-on platforms (Figure 1), and tree saddles. Hunters usually positions themselves 3 to 9 m (10−30 ft) from ground level, finding a balance between prey sight line and shot angle.

The prototypical patient with a tree stand−related injury (TSRI) is a 38- to 45-y-old white male.[4] The most frequent serious injuries are lower extremity or spinal column fractures with or without spinal cord injury.[5-13] The overwhelming majority of hunters who fall are not wearing a fall arrest system or safety harness.[8-16] Some studies show that the fallen hunter is most commonly engaged in archery hunting and falls while descending or ascending a stand in the evening hours.[16,17]

Numerous efforts have been undertaken to make hunting from a tree stand safer, and most focus on hunter education. Since the early 2000s, US state game agencies have included tree stand safety information in the 2-d hunter's education/safety course. Completion of this course is required to purchase a base hunting license and applicable game tags. However, the course is taken only once, potentially leaving many years without formal reminders on gun, archery, or tree stand safety. In 2004, the nonprofit Tree Stand Manufacturer's Association advocated with the Consumer Product Safety Commission to support industry standard equipment specifications for manufactured stands and the inclusion of a fall arrest system in every tree stand sold. In 2007, written material and a DVD on tree stand safety were added as a requirement for manufacturers seeking Tree Stand Manufacturer's Association safety certification.[6] Two states, Alabama and Mississippi, currently have laws mandating safety harness use on public hunting land, which comprises about 7 and 11% of each state, respectively. There is currently no research on the impact of these laws.

The National Electronic Injury Surveillance System (NEISS) shows that TSRI in the United States remain a persistent problem, with 3306 estimated TSRI in 2019. Most previous research has focused only on TSRI patients admitted to the hospital, with data sourced from trauma registries.[5-10,14,15] These sources do not include nonadmitted or patients who present for outpatient services such as urgent care or primary care doctor visits. After an extensive literature review, we found only 4 studies that included nonadmitted and outpatient TSRI patients.[4,11,16,17] This population is of interest not only in understanding the local incidence of TSRI, but also for the possibility that significant injuries occur that do not require admission.

This study characterizes injury patterns found in nonadmitted and admitted TSRI patients presenting to a level 2 Michigan trauma center. We also briefly review prevention efforts and novel interventions aimed at reducing TSRI.

Methods

Cases were obtained through a retrospective chart review of the Henry Ford Allegiance Health (HFAH) trauma registry and EPSi cost accounting database. Keywords searched included fall, hunter, tree, tree stand, treestand, ICD 9 diagnosis *(E884.9_Other accidental fall from one level to another)*, and ICD 10 diagnosis *(W14.XXXA_Fall from tree, initial encounter)*. The results captured both nonadmitted and admitted TSRI patients seen at HFAH or outpatient venues (eg, primary care clinic). Data sets were merged and duplicates removed. The time period of review ran from January 1, 2015 through December 31, 2019.

Inclusion criteria were age >18 y and fall from a tree stand. Variables tracked included age, sex, body mass index (BMI), injury severity score (ISS), abbreviated injury scales, disposition, alcohol use at time of incident, injuries sustained, reported height of fall, and narrative of the fall. The HFAH institutional review board approved the study. Data are reported as mean±SD. All analyses of the study data were performed using IBM SPSS Statistics Version 26. In comparing means across groups, the analysis of variation F-test was used with a cutoff *P*-value of 0.05 to determine statistical significance.

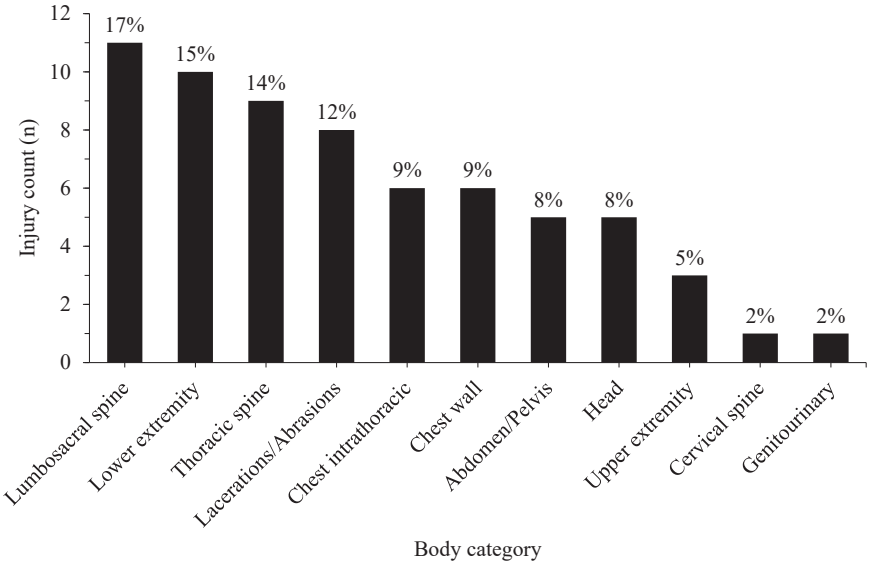

Figure 2. Injury count by body category in 33 patients with tree stand–related injury. Total number of injuries=65.

Results

Thirty-three patients were captured in our data review. All patients were male. Patient age was 45±13 y. BMI for all patients was 30.8±6.6 $kg \cdot m^{-2}$. BMI in nonadmitted patients was 28.3±4.9 $kg \cdot m^{-2}$ versus 32.1±7.1 $kg \cdot m^{-2}$ in admitted patients (P=0.126). ISS for all patients was 9±10, with a minimum of 0 and maximum of 45. ISS for nonadmitted patients was 2±1 versus 13±11 in admitted patients (P=0.003). Reported height of fall for all patients was 5±2 m (17±5 ft), with a minimum of 3 m (10 ft) and a maximum of 9 m (30 ft). Reported height of fall for nonadmitted patients was 5±1 m (17±4 ft) versus 5±2 m (17±5 ft) in admitted patients. Fall arrest systems or harnesses were documented as being used in 4 (12%) cases; in 5 (15%) cases, harnesses were documented as not being used. There was no documentation about harness use or nonuse in the majority of patient charts (n=24; 73%). Alcohol use at the time of the incident was positive by laboratory ethanol test in 3 cases (9%) and negative in 21 cases (64%). Alcohol use was not documented or tested for in 9 (27%) cases.

In the emergency department disposition category, 11 (33%) patients were discharged home after evaluation and treatment (ie, nonadmitted). The remaining 22 (67%) were admitted to the floor (n=9), step-down (n=1), operating room (n=2), surgical intenstive care unit (n=9) or transferred (n=1). Ultimate disposition from the hospital was home for 27 (82%) patients, and the remaining 6 (18%) required some form of continued care (eg, rehabilitation or skilled nursing facility).

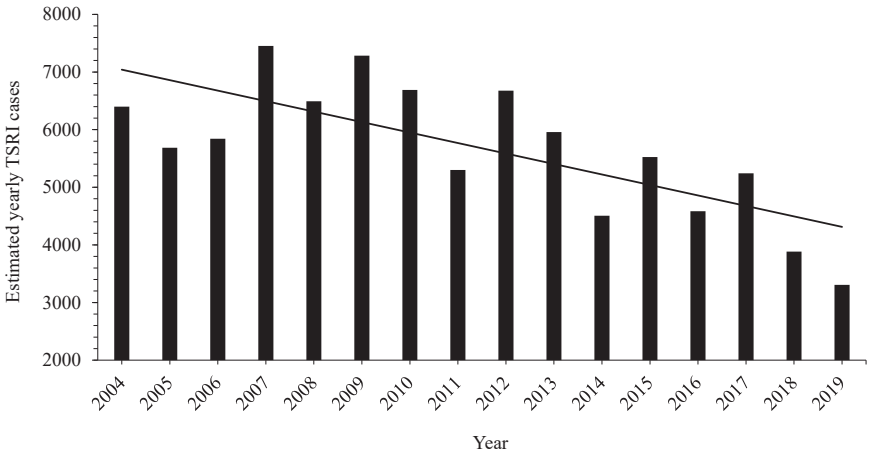

Figure 3. National Electronic Injury Surveillance System estimates of yearly tree stand–related injuries in the United States from 2004 through 2019. The trendline shows an overall decline in tree stand–related injury estimates.

The most common injuries sustained were lumbosacral spine fractures in 11 (17%), lower extremity fractures in 10 (15%), and thoracic spine fractures in 9 (14%). The least likely injuries were cervical spine fractures (n=1; 2%) and genitourinary (n=1; 2%). All patients (6 of 6) who sustained rib fractures had >3 ribs fractured. Of those who were injured, most patients were polytraumas with more than 1 body category involved (n=18; 55%). Nonadmitted patients sustained various injuries, including contusions, abrasions, suspected nasal bone fracture (n=1), medial malleolus fracture requiring outpatient surgery with podiatry (n=1), an L3 compression fracture requiring a back brace (n=1), a proximal fibular shaft fracture that resulted in chronic pain (n=1), and complex scrotal/penis laceration with exposed testicle requiring urology evaluation and repair in the emergency department (n=1). There was 1 fatality in the study. See Figure 2 for injury count and percentages by category.

Discussion

Consistent with prior studies,[5-13] injury patterns in the data set predominantly involved spinal or lower extremity fractures. Harness use, documentation about harness use, and alcohol use at the time of the incident were low and similar to prior research on TSRI.[8-10,12-15] There was a significant portion of cases in the nonadmitted category. Most nonadmitted patients sustained only minor injuries, but some had significant injuries requiring specialist consultation, orthopedic braces, and outpatient surgery. None of these nonadmitted patients were captured by the trauma registry.

Our study showed that BMI was higher in admitted patients compared to nonadmitted patients. Although this observation was not statistically significant, the difference across groups is sufficiently large that in a larger sample size this association might be statistically significant. A prior study showed that patients with a BMI <25 $kg \cdot m^{-2}$ and patients with a BMI >25 $kg \cdot m^{-2}$ had similar ISS and Glasgow coma scale values, but those with a BMI >25 $kg \cdot m^{-2}$ were more likely to go to rehabilitation facilities after their TSRI (36 vs 9%, respectively).[9] Our study did not show a statistically significant difference in ultimate disposition based on BMI >25 $kg \cdot m^{-2}$ (nonadmitted 85% vs admitted 83%; P=1.00).

We found no statistically significant association between reported height of fall and ISS. Although there was no difference in the reported height of fall between the nonadmitted and admitted patients, there was a substantial difference in ISS. Only 2 studies were found that demonstrated an association between reported height of fall and severity of injury. One study found that tree stand falls >3 m (11 ft) were associated with more severe neurologic injury (eg, radiculopathy, central cord syndrome, spinal cord injury).[15] Another found that falls >6 m (20 ft) were associated with a higher ISS.[9]

Our data suggest that relying on a trauma registry alone may underestimate the local incidence of TSRI. One-third of the cases captured were not included in the HFAH trauma registry. Accounting for these "minor" falls is important because higher numbers can highlight the true extent of the problem and galvanize trauma prevention efforts. Another way to capture more TSRI cases is to mimic the compulsory reporting structure for hunting-related firearm injuries, for which police file incident reports with local conservation officers.[8] This is relevant because a hunter is statistically more likely to be injured by a falling from a tree (4.59 per 10,000 hunters)[6,17] than with a firearm (1.66 per 10,000 hunters).[18]

TRACKING TSRI AND THE NEISS

Unlike hunting-related firearm injuries, TSRI are not mandatorily reported to state game agencies in most states, including Michigan. One tool used to estimate TSRI nationally is the NEISS, a database organized by the Consumer Product Safety Commission that tracks US consumer product–related injuries. The database can be found at www.cpsc.gov/library/neiss.html. It comprises 100 variously sized (ie, patient volume/year) and located (ie, rural/urban) emergency departments across the United States that input data from emergency department visits nightly into the system. The database then uses statistical models to estimate the national number of incidents related to specific consumer products, including tree stands used for hunting. NEISS data are limited because the database is a sample of hospitals and therefore of cases; it is not a census, and the estimates have an associated variability subject to sampling error. The NEISS solely identifies patients who sought care in an emergency department, not including those seen in an urgent care center or physician office; thus, the true number of injuries is likely greater than captured in the database. Data may also be skewed toward more serious injuries that were deemed emergencies by presenting patients or providers.[4]

The NEISS estimates of yearly TSRI cases in the United States are declining (Figure 3); however, these data are tempered by the overall decrease in US big game hunters. The following incidence rates use NEISS data and refer to US big game hunters, unless otherwise specified. In 2006, there were 10.7 million hunters and an estimated 5.45 TSRI per 10,000 hunters.[1] In 2016, there were 9.2 million hunters and an estimated 4.98 TSRI per 10,000 hunters.[3]

Previous studies have used NEISS data to analyze both hunting-related firearm injuries[18] and TSRI.[4,17] One epidemiologic study of TSRI used NEISS data from 2000 to 2007 and showed an overall rate of 4.59 TSRI per 10,000 hunters per year.[17] Another study used a novel regional electronic medical record that captured nearly all medical encounters related to TSRI for residents of north and central Wisconsin from 2009 to 2013. This study found an estimated rate of 6 TSRI per 10,000 local Wisconsin hunters in 2009 and 3.6 TSRI per 10,000 local Wisconsin hunters in 2013, which was not a significant change over 5 y (P=0.79).[16] Comparing these epidemiologic TSRI incident rates with much of the prior research is problematic because TSRI incident rates are not calculated or estimated in those studies.[5-10,14,15] Most prior studies on TSRI use trauma databases that exclude nonadmitted patients, making incident rate estimates inherently inaccurate.[5-10,14,15]

According to our data, 33 TSRI cases from 2015 to 2019 amounts to an estimated 6.6 cases per year. HFAH serves as the only hospital in Jackson County, Michigan, and the only level 2 trauma center in a 37-mile catchment area. The estimated mean number of resident Jackson County deer hunters over that time frame was 11,519.[19] Presuming most serious TSRI presented to our hospital, the yearly estimated TSRI case rate was 5.73 per 10,000 Jackson County deer hunters (95% CI 3.94–8.05) from 2015 to 2019, which is consistent with NEISS estimates and previous epidemiologic studies.[16,17]

PREVIOUS PREVENTION EFFORTS

Incorporating tree stand safety material in hunter education classes has not universally demonstrated a decrease in TSRI. A 20-y study from the Pennsylvania trauma registry (1987–2006) showed that TSRI have actually increased significantly despite the inclusion of tree stand safety material in 1999 and reduced numbers of hunters.[14] This may be due to a low rate of safety harness use among TSRI patients, which previous studies documented at 3 to 4%.[10,15] Authors have noted that there is a high degree of underreporting and/or underrecording because harness use is often left out of the medical record.[10,15] One study described a successful TSRI prevention strategy. In 1992, a tree stand safety educational campaign (ie, pamphlets and mailers) in Louisiana that focused on hunt clubs and sporting goods retailers resulted in a reduction in tree stand–related spinal cord injuries over the next 3 y.[20] The results suggest an association, but the study is limited by a lack of randomization and an inability to exclude a myriad of variables. Some prior researchers have proposed state laws to mandate safety harness use.[9] We speculate that such laws would be hard to enforce owing to difficulty in locating hunters, the large amount of man hours needed by state games agencies to find said hunters, and the unpopular aspect of disturbing game during their search. Lastly, a law can be enacted but not followed if a hunter does not buy-in to the importance of fall prevention.

NOVEL PREVENTION EFFORTS

Education is the cornerstone of any system to promote safer behavior. For some high-risk behaviors, education has proven to be not enough. To decrease severe injury from motor vehicle collisions, built-in safety mechanisms are now required or being implemented by manufacturers, such as seat belt warnings and automatic braking. Engineered safety mechanisms might be developed to improve tree stand safety.[21] Engineered solutions to protect hunters are already in place, particularly in terms of firearm safety, such as blaze orange clothing, the gun trigger guard, and the trigger safety mechanism.[22] There are no equivalent measures in TSRI prevention except for warning labels on the tree stand itself—a hunter must still choose to wear a harness. Some novel prevention efforts are reaching hunters though social media. In 2018, the Pennsylvania Game Commission began a highway billboard campaign to reduce TSRI. The billboards feature the silhouette of a tree stand and hunter with the text “wear your harness” and run annually from October 1 to mid-December. Michigan’s DNR marketing strategies are shifting to include social media outreach through Facebook and other platforms that use geographic data from cell phones to target ad campaigns on hunter safety. They also use an email newsletter to propagate videos on firearm, boating, and tree stand safety each season. Currently, there is no research on the impact of these efforts.

LIMITATIONS

Study limitations include the retrospective design, small sample size, exclusion of patients <18 y, incomplete medical documentation (eg, harness use), and reliance on patient reported details (eg, narrative of the fall). We also presumed that most, if not all, serious TSRI in our catchment area presented to our singular county level 2 trauma center to calculate the TSRI case rate for Jackson County. There were likely TSRI that occurred in Jackson County that either presented to another institution or did not present at all to a medical facility (ie, minor injuries), thus limiting our sample size. There is likely a reporting/identification bias because patients with more serious injuries would be more apt to seek medical care.

Conclusions

We found that TSRI have considerable morbidity, mostly involving fractures of the spine and lower extremities. One-third of the patients in our study were nonadmitted, some of whom had significant injuries requiring specialist consultation, orthopedic braces, or outpatient surgery. The primary treatment for TSRI is prevention. Accurate reporting of both nonadmitted and admitted TSRI may allow for tracking the success or failure of prevention measures and can be used to educate the public and medical community alike. The authors recommend that, as a public health measure, and as with hunting-related firearm injuries, states should enact laws mandating reporting of TSRI and that state game agencies implement TSRI education efforts using social media and billboards during hunting seasons.

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WILDERNESS MEDICAL SOCIETY CLINICAL PRACTICE GUIDELINES

Wilderness Medical Society Clinical Practice Guidelines for the Prevention and Management of Tick-Borne Illness in the United States

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The Wilderness Medical Society convened an expert panel to develop evidence-based guidelines for the prevention and management of tick-borne illness (TBI). Recommendations are graded based on quality of supporting evidence according to criteria put forth by the American College of Chest Physicians. The guidelines include a brief review of the clinical presentation, epidemiology, prevention, and management of TBI in the United States, with a primary focus on interventions that are appropriate for resource-limited settings. Strong recommendations are provided for the use of DEET, picaridin, and permethrin; tick checks; washing and drying clothing at high temperatures; mechanical tick removal within 36 h of attachment; single-dose doxycycline for high-risk Lyme disease exposures versus "watchful waiting;" evacuation from backcountry settings for symptomatic tick exposures; and TBI education programs. Weak recommendations are provided for the use of light-colored clothing; insect repellents other than DEET, picaridin, and permethrin; and showering after exposure to tick habitat. Weak recommendations are also provided against passive methods of tick removal, including the use of systemic and local treatments. There was insufficient evidence to support the use of long-sleeved clothing and the avoidance of tick habitat such as long grasses and leaf litter. Although there was sound evidence supporting Lyme disease vaccination, a grade was not offered as the vaccine is not currently available for use in the United States.

Keywords: DEET, Lyme disease, spirochetes, rickettsia, prophylaxis

Introduction

In the United States, 95% of human vector-borne diseases reported to the Centers for Disease Control and Prevention (CDC) per year are attributable to tick-borne pathogens.[1] In turn, the number of cases of tick-borne illnesses (TBIs) reported to the CDC per year in the United States has more than doubled over the past 2 decades, totaling 50,865 in 2019,[2] with formal reporting likely underestimating the disease burden of TBIs.[3] Although Lyme disease is the most commonly reported vector-borne disease in the United States, the incidence of other TBIs, including anaplasmosis, spotted fever rickettsiosis, babesiosis, tularemia, alpha-gal syndrome, and Powassan virus, continues to rise as well.[4]

Given the increased incidence of TBI, it is critical for providers to be comfortable with the prevention and management of tick bites. TBI, however, is a complex landscape with a considerable volume of literature describing relatively rare syndromes and controversial treatment regimens. In an effort to deliver a succinct clinical practice guideline (CPG), the authors have chosen to focus the scope of this article on those issues most

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relevant to the front-line provider with minimal resources: the prevention of tick bites and the management of TBI in settings where diagnostics and therapeutics are limited. Several recent articles are available for the reader interested in a comprehensive review of emerging TBIs or the management of TBI in the hospital setting.[5,6]

Methods

An expert panel was convened through the Wilderness Medical Society clinical practice guidelines committee. Panelists were selected based on clinical interest or research experience. Articles were identified in 2 searches. The first search identified articles through the PubMed and Google Scholar databases using a keyword search with the following terms: tick, tick-borne, tick bite, tick-borne disease, borreliosis, Lyme disease, Rocky Mountain spotted fever. The second search broadened these topics to include a keyword search with the following terms: tick prevention (with permethrin, tick checks, clothing, and habitat management), DEET, and Lyme vaccine. These searches were supplemented by manually searching the references of the obtained articles, existing practice guidelines, and CDC references. All articles were peer reviewed. Methodological scope included randomized controlled trials, observational studies, and case series from both human and animal trials. Given the broad scope of TBI and the limited scope of this CPG, review articles have been cited mainly to augment topics not fully covered by this CPG. However, primary literature was used to derive the graded recommendations within this guideline. All literature searches were performed by study authors, without restrictions placed on date of publication or country of origin. The panel used the American College of Chest Physicians (ACCP) (online Supplementary Table)[7] classification scheme for grading evidence and recommendations. There were instances in which the ACCP schematic did not adequately describe our recommendations. Recommendations based on reasonable clinical practice, but not supported by data, were classified as "expert opinion."

Ticks and Human Illness

TICKS AS A VECTOR FOR DISEASE

Transmission of TBIs to humans requires an interaction between pathogen, vector, and host; each tick species can serve as a vector for a number of different bacterial, viral, and parasitic pathogens (Tables 1 and 2)[8] and may seek out different hosts for blood meals in each of their 3 life stages (https://www.cdc.gov/ticks/index.html) (Figure 1).[1] Disease transmission can occur between vector and host directly through the feeding process, or between tick vectors co-feeding on the same host.[1] As a result of these vector-host interactions, the geographic distribution of TBIs closely reflects the distribution of the tick vectors that transmit disease (Figure 2).[8]

TICK BEHAVIOR AND DISEASE TRANSMISSION

Ticks find their hosts by sensing heat, exhalations, vibrations, and odors. Once a host has been found, ticks burrow their hypostome under the skin using a cutting movement.[9] Most tick bites are painless. Once the tick is feeding, a complex molecular cascade facilitates the transmission of pathogens from the gut of an infected tick vector to the host. Prostaglandins in tick saliva inhibit the host's local immune response, and tick salivary apyrase maintains blood flow into the bite site, stimulates local vasodilation, and prevents platelet aggregation. Other tick salivary enzymes inhibit the coagulation cascade, enhancing blood flow to the lesion.[10] Importantly, there is considerable variability in the time it takes to transmit pathogen from tick to human. In Lyme disease, disease transmission is believed to take >36 h[11]; in contrast, Rocky Mountain spotted fever can be transmitted in approximately 15 min.[12]

INCIDENCE OF TICK-BORNE ILLNESS

As of 2019, Lyme disease accounted for more than 69% of more than 50,000 reported cases of TBI, whereas anaplasmosis represented 16% and spotted fever rickettsiosis represented 10%.[2] Although summer has been associated with the highest risk of Lyme disease, transmission is possible year-round.[13] For example, hunters must remain vigilant for tick bites through the fall season based on seasonal tick collection surveys.

Environmental factors that influence tick and host distribution, feeding patterns, and survivability are rapidly changing. Habitat fragmentation, urbanization, and deforestation all affect the distribution and migration of tick vectors and hosts. Climate change, with its associated temperature and precipitation fluctuations, has enabled tick expansion to regions that have previously experienced little TBI.[14–16] It is thought that these anthropogenic changes are contributing to the expanding range of tick habitat as well as an increase in tick abundance in existing habitats.[17]

TICK IDENTIFICATION, COINFECTION, AND MAJOR TBIS OF THE UNITED STATES

Ticks commonly encountered in the United States are shown in Figure 3 (https://www.cdc.gov/ticks/index.

Table 1. Pathogenic organisms and geographical distribution of important US arthropods implicated in tick-borne diseases[8]

Tick	*Distribution*	*Pathogen*	*Disease*	*Comments*
Blacklegged tick – *Ixodes scapularis*	Eastern US	*Borrelia burgdorferi* *Borrelia mayonii* *Anaplasma phagocytophilum* *Borrelia miyamotoi* *Ehrlichia muris eauclariensis* *Babesia microti* Powassan virus	Lyme disease Anaplasmosis Relapsing fever Ehrlichiosis Babesiosis Powassan virus	Greatest risk of being bitten exists in the spring, summer, and fall in the NE, upper MW, and mid-Atlantic. However, adult ticks may be out searching for a host any time winter temperatures are above freezing. All life stages bite humans, but nymphs and adult females are most commonly found on people
Lone star tick – *Amblyomma americanum*	Eastern US Southern US	*Ehrlichia chaffeensis* *Ehrlichia ewingii* *Francisella tularensis* Heartland virus Bourbon virus	Ehrlichiosis Tularemia Heartland virus Bourbon virus STARI Alpha-gal allergy	Greatest risk of being bitten exists in early spring through late fall. A very aggressive tick that bites humans. Adult female is distinguished by a white dot ("lone star") on her back. Nymph and adult females most frequently bite humans.
American dog tick – *Dermacentor variabilis*	East of Rockies, Pacific Coast	*Francisella tularensis* *Rickettsia rickettsii*	Tularemia Rocky Mountain spotted fever	The greatest risk of being bitten occurs during spring and summer. Adult females are most likely to bite humans.
Brown dog tick – *Rhipicephalus sanguineus*	Worldwide	*Rickettsia rickettsii*	Rocky Mountain spotted fever	Dogs are the primary host for the brown dog tick in each of its life stages but may also bite humans or other mammals.
Groundhog tick – *Ixodes cookei*	Eastern half of US	Powassan virus	Powassan virus	All life stages feed on a variety of warm-blooded animals (groundhogs, skunks, squirrels, raccoons, foxes, weasels) and occasionally humans and domestic animals.
Gulf Coast tick – *Amblyomma maculatum*	SE and mid-Atlantic states, AZ	*Rickettsia parkeri*	*Rickettsia parkeri* rickettsiosis	Larvae/Nymphs feed on birds and small rodents; adults feed on deer and other wildlife. Adults can transmit disease to humans.
Rocky Mountain wood tick – *Dermacentor andersoni*	Rocky mountain states	*Rickettsia rickettsii* CO tick fever virus *Francisella tularensis*	Rocky Mountain spotted fever CO tick fever Tularemia	Adults feed on large mammals; larvae/nymphs feed on rodents. Adults can transmit disease to humans.
Soft tick – *Ornithodoros spp.*	Western US, TX	*Borrelia hermsii* *Borrelia turicatae*	Tickborne relapsing fever	Rustic cabins, cave exposure. Ticks emerge at night, feed while people sleep.
Western blacklegged tick – *Ixodes pacificus*	Pacific Coast	*Anaplasma phagocytophilum* *Borrelia burgdorferi* *Borrelia miyamotoi*	Anaplasmosis Lyme disease Relapsing fever	Larvae/Nymphs feed on lizards, birds, rodents; adults feed on deer. Nymphs/Adult females most often bite humans.

html). Tick identification can be challenging given the variety of tick life cycles and the dramatic anatomic changes that occur with engorgement. Moreover, in a significant proportion of TBIs, the patient will not recall a tick or tick bite.[18] Providers must be prepared to diagnose and manage TBI without definitive tick identification.

Several tick species are able to carry multiple pathogens (Table 1). In 1 study, nearly 25% of *Ixodes* were coinfected with some combination of the bacteria or parasites causing Lyme disease, anaplasmosis, or babiesosis.[19] Although TBI diagnosis is not the focus of this CPG, providers should be aware of high rates of coinfection[20,21]; the presence of 1 TBI should in many instances prompt testing for others. A brief clinical summary of the major TBIs found in the United States follows, supplemented by further detail in Table 2.

Table 2. Clinical syndromes, laboratory diagnosis, and treatment of common bacterial, protozoal, and viral tick-borne illnesses[8,77,104]

Disease	*Causative organism*	*Distribution*	*Incubation*	*Clinical syndrome*	*Laboratory findings*	*Laboratory diagnosis*	*Treatment*
Bacterial tick-borne illnesses							
Borrelioses Lyme disease	*Borrelia burgdorferi* *Borrelia mayonii*	Upper Midwest and Northeastern US 14 states accounted for 95% of Lyme disease reports in 2015: CT, DE, ME, MD, MA, MN, NH, NJ, NY, PA, RI, VT, VA, WI Some cases also reported in northern CA, OR, and WA	3–30 d	Localized: - erythema migrans - flu-like symptoms - lymphadenopathy Disseminated: - multiple secondary annular rashes - flu-like symptoms - lymphadenopathy Rheumatologic manifestations - transient, migratory mono/polyarthritis - migratory musculoskeletal pain - Baker's cyst - recurrent arthralgia Cardiac manifestations - conduction abnormalities - myocarditis, pericarditis Neurologic manifestations - Bell's palsy or other cranial neuropathy - meningitis - motor/sensory radiculoneuropathy - cognitive difficulties - encephalitis, encephalomyelitis, encephalopathy, pseudotumor cerebri Additional manifestations - conjunctivitis, keratitis, uveitis - mild hepatitis - splenomegaly	- Elevated ESR - Elevated hepatic transaminases - Microscopic hematuria/proteinuria - Lyme meningitis: lymphocytic pleocytosis, elevated protein, normal glucose in CSF	2-tiered serologic testing*: - ELISA (IgG/IgM) - Western blot if positive or equivocal * 2-tiered testing often falsely negative early in disease	*Prophylaxis* - doxycycline 200 mg, once *Localized Lyme disease* Adults: - doxycycline 100 mg bid, 10–21 d - cefuroxime 500 mg bid, 14–21 d - amoxicillin 500 mg tid, 14–21 d Children: - amoxicillin 50 mg·kg^{-1} daily divided into 3 doses, 14–21 d - doxycycline 4 mg·kg^{-1} daily divided into 2 doses, 10–21 d - cefuroxime 30 mg·kg^{-1} daily divided into 2 doses, 14–21 d *Lyme meningitis* Adults: - ceftriaxone 2g·d^{-1} IV, 10–28 d - cefotaxime 6 g·d^{-1} IV divided q 8 h, 10–28 d Children: - ceftriaxone 50–75 mg·kg^{-1} daily IV, 10–28 d - cefotaxime 150–200 mg·kg^{-1} daily IV divided q 8 h, 10–28 d *Cranial-nerve palsy without meningitis* Adults: - doxycycline 200 mg·d^{-1} divided bid, 14–21 d - amoxicillin 1500 mg·d^{-1} divided tid, 14–21 d - cefuroxime axetil 1000 mg·d^{-1} divided bid, 14–21 d Children: - doxycycline 4 mg·kg^{-1} daily divided bid, 14–21 d - amoxicillin 50 mg·d^{-1} divided tid, 14–21 d - cefuroxime axetil 30 mg·d^{-1} divided bid, 14–21 d *Carditis* Same oral agents as for localized Lyme disease, same parenteral agents as for Lyme meningitis, 14–21 d *Arthritis* Same oral agents as for localized Lyme disease, same parenteral agents as for Lyme meningitis, 28 d

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Table 2 *(continued)*

Disease	*Causative organism*	*Distribution*	*Incubation*	*Clinical syndrome*	*Laboratory findings*	*Laboratory diagnosis*	*Treatment*
Borrelia miyamotoi disease	*Borrelia miyamotoi*	Upper Midwest, Northeast, and mid-Atlantic states Presumed in CA	Days to weeks	- Fever - Chills - Fatigue - Severe headache - Arthralgia/Myalgia - Dizziness, confusion, vertigo - Rash - Dyspnea - Nausea, abdominal pain, diarrhea, anorexia	- Leukopenia - Thrombocytopenia - Elevated hepatic transaminases	Signs/Symptoms with: - PCR for DNA, or -antibody tests	No comprehensive studies to evaluate treatment regimens; antibiotics for Lyme disease successful in case series
Tick-borne relapsing fever	*Borrelia hermsii* *Borrelia turicatae*	14 western states: AZ, CA, CO, ID, KS, MT, NV, NM, OK, OR, TX, UT, WA, WY	~7 d, followed by febrile episodes lasting ~3 d and separated by ~7 d afebrile periods	- Headache - Myalgia - Chills - Nausea, vomiting - Arthralgia - Facial palsy	- Normal-increased WBC with left shift - Increased serum bilirubin - Thrombocytopenia - Elevated ESR - Prolonged PT/PTT	- Microscopy/Culture while febrile - Peripheral blood smear - Convalescent serologic testing	Adults: - tetracycline 500 mg qid, 10 d - erythromycin 500 mg qid, 10 d - ceftriaxone 2 g·d^{-1} IV, 10–14 d Children: - erythromycin 12.5 mg·kg^{-1} qid, 10 d
Rickettsioses							
Rocky Mountain spotted fever	*Rickettsia rickettsii*	Throughout the US 5 southern states account for 60% of cases: NC, OK, AR, TN, MO	3–12 d	Early (1–4 d): - high fever - severe headache - malaise - myalgia - edema around eyes, hands - nausea, vomiting, anorexia Late (5 d and beyond): - AMS, coma, cerebral edema - pulmonary edema, ARDS - necrosis, requiring amputation - multiorgan system damage (CNS, renal failure) Rash: develops 2–5 d after symptom onset *Early rash* - maculopapular—on wrist, forearms, ankles, spreading to trunk, palms, and soles *Late rash* - petechial—signifies severe disease, develops after day 6	- Thrombocytopenia - Elevated hepatic transaminases - Hyponatremia	4× increase in IgG antibody by IFA in paired serum samples within first week of illness, and 2–4 wk later PCR for DNA in skin biopsy IHC staining from skin/ biopsy	Adults: - doxycycline 100 mg bid Children: - doxycycline 2.2 mg·d^{-1} bid Treat for at least 3 d after fever subsides and symptoms improve, for minimum of 5–7 d Start treatment on clinical suspicion
Rickettsia parkeri Rickettsiosis	*Rickettsia parkeri*	Southeastern and mid-Atlantic states, parts of southern AZ	2–10 d	- Inoculation eschar at site of tick attachment Several days after eschar: - fever - headache - rash (maculopapular/ papulovesicular eruption on trunk/extremities) - muscle aches	- Elevated hepatic transaminases - Mild leukopenia - Mild thrombocytopenia	PCR for DNA in eschar swab, whole blood, skin biopsy 4× increase in IgG antibody by IFA in paired serum samples within first week of illness, and 2–4 wk later	Adults: - doxycycline 100 mg bid Children: - doxycycline 2.2 mg·kg^{-1} bid Treat for at least 3 d after fever subsides and symptoms improve, for minimum of 5–7 d

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Table 2 *(continued)*

Disease	*Causative organism*	*Distribution*	*Incubation*	*Clinical syndrome*	*Laboratory findings*	*Laboratory diagnosis*	*Treatment*
Pacific Coast tick fever	*Rickettsia* sp 364D	CA, OR, WA	Not documented	Eschar, followed by fever, regional lymphadenopathy, headache, myalgia, fatigue	Not documented	PCR for DNA in eschar swab 4× increase in IgG antibody by IFA in paired serum samples within first week of illness, and 2–4 wk later	Adults: - doxycycline 100 mg bid Children: - doxycycline 2.2 mg·kg^{-1} bid Treat for at least 3 d after fever subsides and symptoms improve, for minimum of 5–7 d
Ehrlichiosis and *Anaplasmosis*							
Anaplasmosis	*Anaplasma phagocytophilum*	Upper Midwest, northeastern US 8 states account for 90% of cases: VT, ME, RI, MN, MA, WI, NH, NY	5–14 d	- Fever, chills, rigors - Severe headache - Malaise - Myalgia - GI symptoms: nausea, vomiting, diarrhea, anorexia - Rash	- Mild anemia - Thrombocytopenia - Leukopenia with lymphopenia and left shift - Mild elevation in hepatic transaminases	PCR for DNA in whole blood 4× increase in IgG antibody by IFA in paired serum samples within first week of illness, and 2–4 wk later IHC staining of organism from skin, tissue, or BM biopsy Morulae in granulocytes on blood smear	Adults: - doxycycline 100 mg bid, 10–14 d Children: - doxycycline 2.2 mg·kg^{-1} bid, 10–14 d Start treatment on clinical suspicion
Ehrlichiosis	*Ehrlichia chaffeensis, ewingii, muris eauclairensis*	Southeastern, south-central US, 3 states account for 35% of cases: OK, MO, AR	5–14 d	- Fever, chills - Headache - Malaise - Muscle pain - GI symptoms: nausea, vomiting, diarrhea, anorexia - AMS - Rash	- Thrombocytopenia - Leukopenia - Anemia - Mild elevation in hepatic transaminases	PCR for DNA in whole blood 4× increase in IgG antibody by IFA in paired serum samples within first week of illness, and 2–4 wk later IHC staining of organism from skin, tissue, or BM biopsy	Adults: - doxycycline 100 mg bid Children: - doxycycline 2.2 mg·kg^{-1} bid Treat for at least 3 d after fever subsides and symptoms improve, for minimum of 5–7 d Start treatment on clinical suspicion

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Table 2 *(continued)*

Disease	*Causative organism*	*Distribution*	*Incubation*	*Clinical syndrome*	*Laboratory findings*	*Laboratory diagnosis*	*Treatment*
Other Tularemia	*Francisella tularensis*	All states except HI	3–5 d (range 1–21 d)	- Fever, chills - Headache - Malaise, fatigue - Anorexia - Myalgia - Chest discomfort, cough - Sore throat - Vomiting, diarrhea, abdominal pain Ulceroglandular: - localized lymphadenopathy - cutaneous ulcer at infection site Oculoglandular: - photophobia - excessive lacrimation - conjunctivitis - preauricular, submandibular, or cervical lymphadenopathy Oropharyngeal: - severe throat pain - exudative pharyngitis or tonsillitis - cervical, preparotid, and/or retropharyngeal lymphadenopathy Pneumonic: - nonproductive cough - substernal tightness - pleuritic chest pain - hilar adenopathy, infiltrate, or pleural effusion on chest x-ray Typhoidal: - any combination of general symptoms (without localizing symptoms or other syndrome)	- Leukocytosis - Elevated ESR - Thrombocytopenia - Hyponatremia - Elevated hepatic transaminases - Elevated creatine phosphokinase - Myoglobinuria - Sterile pyuria	Isolation of *F tularensis* from clinical specimen or 4× increase in serum antibody titer to *F tularensis* antigen between acute and convalescent specimens Detection of *F tularensis* in clinical specimen by DFA or PCR, or single positive antibody titer to *F tularensis* antigen	Adults: - streptomycin 1 g IM bid, min 10 d - gentamicin[a] 5 mg·kg^{-1} IM/IV daily, min 10 d - ciprofloxacin[a] 400 mg IV or 500 mg po bid, 10–14 d Doxycycline 100 mg bid, 14–21 d Children: - streptomycin 15 mg·kg^{-1} IM bid, min 10 d - gentamicin[a] 2.5 mg·kg^{-1} IM/IV tid,[b] min 10 d - ciprofloxacin[a] 15 mg·kg^{-1} IV or po bid, min 10 d

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Table 2 *(continued)*

Disease	*Causative organism*	*Distribution*	*Incubation*	*Clinical syndrome*	*Laboratory findings*	*Laboratory diagnosis*	*Treatment*
Protozoan, viral, and other tick-borne illnesses							
Protozoa							
Babesiosis	*Babesia microti*, *Babesia spp.*	Upper Midwest and Northeastern US	1–9 wk	- Fever, chills, sweats - Malaise, fatigue - Myalgia, arthralgia, headache - GI symptoms: anorexia, nausea, abdominal pain, vomiting - Dark urine - Less common: cough, sore throat, emotional lability, depression, photophobia, conjunctival injection - Mild splenomegaly, hepatomegaly, jaundice	- Decreased hematocrit - Thrombocytopenia - Elevated serum creatinine and BUN - Mildly elevated hepatic transaminases	- Identification of intraerythrocytic *Babesia* parasites on peripheral blood smear, or - PCR, or - isolation of *Babesia* parasites from whole blood by animal inoculation Supportive criteria: - *Babesia*-specific antibody titer by IFA	Adults: - atovaquone 750 mg q 12 h AND azithromycin 500–1000 mg day 1, 150–1000 mg daily on subsequent days, 7–10 d Children: - clindamycin 300–600 mg IV q 6 h OR 600 mg po q 8 h AND quinine 650 mg po q 6–8 h, 7–10 d
Viruses							
Powassan virus disease	*Powassan virus*	Northeastern states and Great Lakes region	1–4 wk	- Fever, headache, vomiting, generalized weakness - Usually progresses to meningoencephalitis, may include meningeal signs, AMS, seizures, aphasia, paresis, movement disorders, cranial nerve palsies	- CSF: lymphocytic pleocytosis, mildly elevated protein, normal glucose	Virus-specific IgM antibodies in serum or CSF RT-PCR for viral RNA in acute CSF specimen or tissues	No specific antiviral treatment is available Supportive care
Colorado tick fever	*Colorado tick fever virus*	Western US, primarily CO, UT, MT, WY	1–14 d	- Fever, chills, headache, myalgias, lethargy - 50% have biphasic illness with symptoms remitting after 2–4 d, then recurring 1–3 d later - Conjunctival injection, pharyngeal erythema, lymphadenopathy - Maculopapular/Petechial rash in <20% - Prolonged convalescence with weakness and fatigue - DIC and meningoencephalitis is rare in children, can be fatal	- Leukopenia - Moderate thrombocytopenia	Culture and RT-PCR during first 2 wk of illness Serologic assays (IgM-capture EIA, IFA, plaque-reduction neutralization) on convalescent samples	No specific antiviral treatment is available Supportive care
Heartland virus disease	*Heartland virus*	Midwest, southern US	Unknown	- Fever - Fatigue - Decreased appetite - Headache - Arthralgia - Myalgia - Nausea - Diarrhea	- Leukopenia - Thrombocytopenia - Mild elevation in liver transaminases	Viral RNA and IgM/IgG antibodies	No specific antiviral treatment is available Supportive care

AMS, altered mental status; ARDS, acute respiratory response syndrome; bid, twice daily; BUN, blood urea nitrogen; CNS, central nervous system; CSF, cerebrospinal fluid; DIC, disseminated intravascular coagulation; EIA, enzyme-linked immunoassay; ELISA, enzyme-linked immunosorbent assay; ESR, erythrocyte sedimentation rate; GI, gastrointestinal; IFA, immunofluorescence assay; IHC, immunohistochemistry; IV, intravenous; PCR, polymerase chain reaction; PT/PTT, prothrombin time/partial thromboplastin time; tid, thrice daily; WBC, white blood cell.

[a]Not a Food and Drug Administration–approved use.

[b]Once-daily dosing could be considered in consultation with a pediatric ID specialist.

Bacteria

Anaplasmosis

Anaplasmosis, previously known as human granulocyte ehrlichiosis, is caused by the pathogen *Anaplasma phagocytophilum* carried by *Ixodes* spp ticks. Anaplasmosis is most frequently reported in the Upper Midwest and Northeastern United States, with a distribution that overlaps with Lyme disease. The incubation period is 5 to 14 d, and common symptoms include fever, rigors, headache, myalgias, vomiting, and diarrhea. Rash is uncommon. Doxycycline is first-line therapy.

Ehrlichiosis

Ehrlichiosis is the general name used to describe disease caused by *Ehrlichia chaffeensis*, *Ehrlichia ewingii*, and *Ehrlichia muris eauclairensis* bacteria and is most commonly reported in the Southeastern and South Central United States. It is transmitted by *Amblyomma americanum* (the lone star tick) and *Ixodes scapularis* (the blacklegged tick). Incubation is 5 to 14 d, and symptoms include fever, headache, myalgias, vomiting, diarrhea, and rash. Doxycycline is first-line therapy.

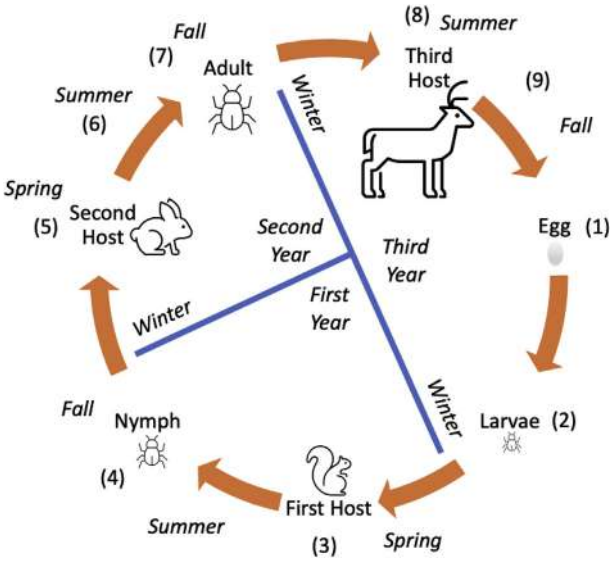

Figure 1. The 3-host life cycle. After the adult female leaves the third host to lay eggs in the fall (1), the eggs hatch into larvae and overwinter (2). The larvae will attach to the first host, usually a small rodent, the following spring (3) and remain attached until late summer, when they drop to molt into nymphs in the fall. After overwintering again, the nymphs will seek out a second host in the spring, again typically a small rodent (5), feed, and then detach later in the summer (6). Nymphs will molt into adults in late summer to fall (7), overwinter, and then seek out a third host in the spring, typically a larger mammal (8). Adult ticks will feed and mate on the third host during the summer, and the female adults will detach in the fall to continue the cycle (9). The 3 hosts are not always different species or different individuals and may be human hosts in all 3 stages.[101]

Lyme Disease and Other Emerging Borrelia Infections

Lyme disease is caused by the spirochete *Borrelia burgdorferi* and is transmitted by *I scapularis* (the blacklegged tick) and *Ixodes pacificus* (the Western blacklegged tick). The majority of cases are reported in the Midwest and Northeast, but Lyme disease is also common in California, Oregon, and Washington. Incubation ranges from 3 to 30 d.

Lyme disease presents in 3 stages: early localized, early disseminated, and late disseminated. In the early localized stage, common symptoms include fever, headache, myalgias, arthralgias, and erythema migrans (EM). EM is estimated to appear in approximately 70 to 80% of cases.[22] The rash is classically described as a "bull's eye" lesion with central clearing that occurs proximal to the site of the tick bite (Figure 4). However, misidentification of EM is relatively common[23]; atypical presentations include lesions with crusts, nodules, or a blueish coloring (Figure 4).

Early disseminated Lyme disease is characterized by multiple annular EM distant from the original tick bite, flu-like symptoms, and neurologic or cardiac manifestations such as cranial nerve palsy, meningitis, or conduction abnormalities. Lyme carditis is a rare, but it is an important cause of mortality[24] and may result in complete heart block as early as 4 d after a tick bite.[25]

Late disseminated Lyme disease occurs months or years after the initial tick bite and is characterized by arthralgias in 1 or more major joints or neurologic symptoms such as sleep disturbances, migraines, vertigo, and numbness in the hands, feet, arms, or legs.

Borrelia miyamotoi is an emerging borrelial infection with a distribution similar to Lyme disease. Although the clinical presentation is similar to Lyme disease, rash is uncommon. An early case series described a hospitalization rate of 24%.[26] *Borrelia mayonii*, recently discovered by researchers at the Mayo Clinic, also has a similar presentation to Lyme disease but has thus far been confined to the Upper Midwest. *B mayonii* infection is associated with higher concentrations of spirochetes in the blood when compared to *B burgdorferi*.[27] Doxycycline is first-line therapy.

Tickborne Relapsing Fever

Tickborne relapsing fever is also a spirochetal disease most commonly caused by *Borrelia hermsii* and *Borrelia turicatae* within the United States. Tickborne relapsing fever is

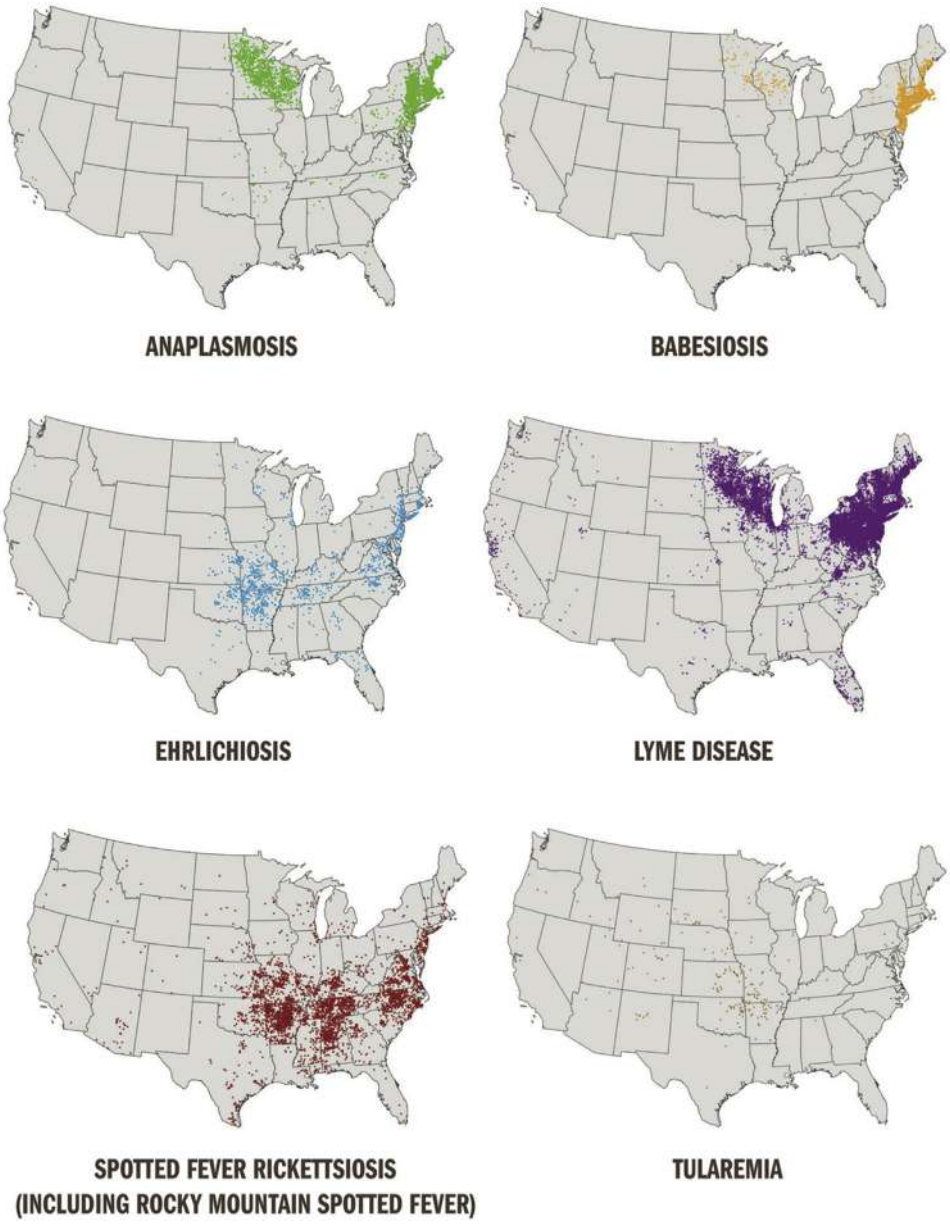

Figure 2. Geographical distribution of US tick-borne illnesses reported to the CDC in 2016.[8] Source: United States Centers for Disease Control and Prevention (public domain).

most commonly reported in western states. The disease is strongly associated with sleeping in rustic cabins or lean-tos where rodents cohabitate. In Texas, the disease is associated with caving. The disease is carried by the soft tick *Ornithodoros* and is characterized by a relapsing and recurring fever, which typically returns every 3 d. Incubation is approximately 1 wk. Other symptoms include headache, vomiting, myalgias, and arthralgias. First-line treatment is tetracycline or erythromycin.

Rocky Mountain Spotted Fever

Rocky Mountain spotted fever is caused by *Rickettsia rickettsii* and can be carried by several tick species. Despite its name, a majority of cases are found in the South Central United States; however, cases have been reported throughout the contiguous United States. The incubation period is 3 to 12 d, and symptoms include fever, headache, malaise, and myalgia. A characteristic maculopapular rash

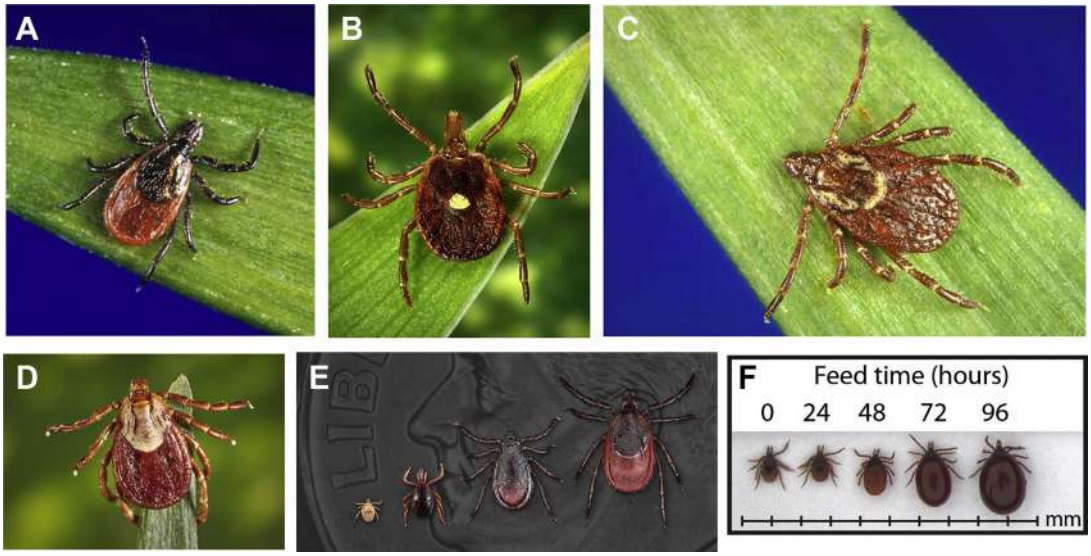

Figure 3. Medically important ticks found in the United States, including (A) *I scapularis*, (B) *A americanum*, (C) *D variabilis*, and (D) *D andersoni*. In addition to species identification, these ticks may present in different (E) life stages and (F) levels of engorgement (Centers for Disease Control and Prevention. Ticks. https://www.cdc.gov/ticks/index.html). Source: United States Centers for Disease Control and Prevention (public domain).

develops 2 to 5 d after the onset of symptoms and may progress to petechiae, heralding more severe disease. Doxycycline is first-line therapy, and delay in treatment beyond 5 d is associated with increased fatality rates.[28]

Tularemia

Tularemia is caused by the bacteria *Francisella tularensis* which is transmitted by several tick species, including *Dermacentor variabilis* (the American dog tick), *Dermacentor andersoni* (the Rocky Mountain wood tick), and *A americanum* (the lone star tick). Tularemia has been reported in all states except Hawaii. In contrast to most other TBIs, tularemia can be transmitted by other vectors, including deer flies and mammals. The incubation period is typically 3 to 5 d. Common symptoms include fever, headache, malaise, and myalgias. A cutaneous ulcer or eschar is classic for tularemia but is not always present. Ultimately, the clinical presentation is dependent on the route of inoculation. Treatment depends on the severity of illness. Doxycycline is first-line therapy for mild symptoms, and streptomycin is reserved for more severe illness.

Protozoa

Babesiosis

Babesiosis is caused most commonly by *Babesia microti* in the United States. Babesia are protozoan parasites of erythrocytes with a life cycle similar to malarial infections. Babesiosis is most frequently found in the Upper Midwest and Northeastern United States, although cases have also been reported on the West Coast. The incubation period is 1 to 9 wk, and symptoms include fever, rigors, headache, myalgias, dark urine, nausea, and diarrhea. First-line therapy is a combination of atovaquone and azithromycin.

Viruses

Colorado Tick Fever

Colorado tick fever is caused by a double-stranded RNA virus from the *Coltivirus* genus. It is transmitted by *D andersoni* (the Rocky Mountain wood tick) and is most commonly reported in Colorado, Utah, Montana, and Wyoming. The incubation period is 1 to 14 d, and early symptoms include fever (which is often biphasic), rash, and conjunctival injection. Prolonged weakness and fatigue are common in adults. Treatment is supportive.

Powassan Virus

Powassan, the only tick-borne encephalitis found in the United States, is caused by a flavivirus related to West Nile virus and dengue, and it is most commonly transmitted by *Ixodes* ticks (the blacklegged and groundhog ticks). The virus is rare, but it is most commonly reported in the Upper Midwest and Northeast. Incubation is 1 to 4 wk. Fever, headache, and vomiting are often followed by an encephalitis syndrome characterized by altered mental status,

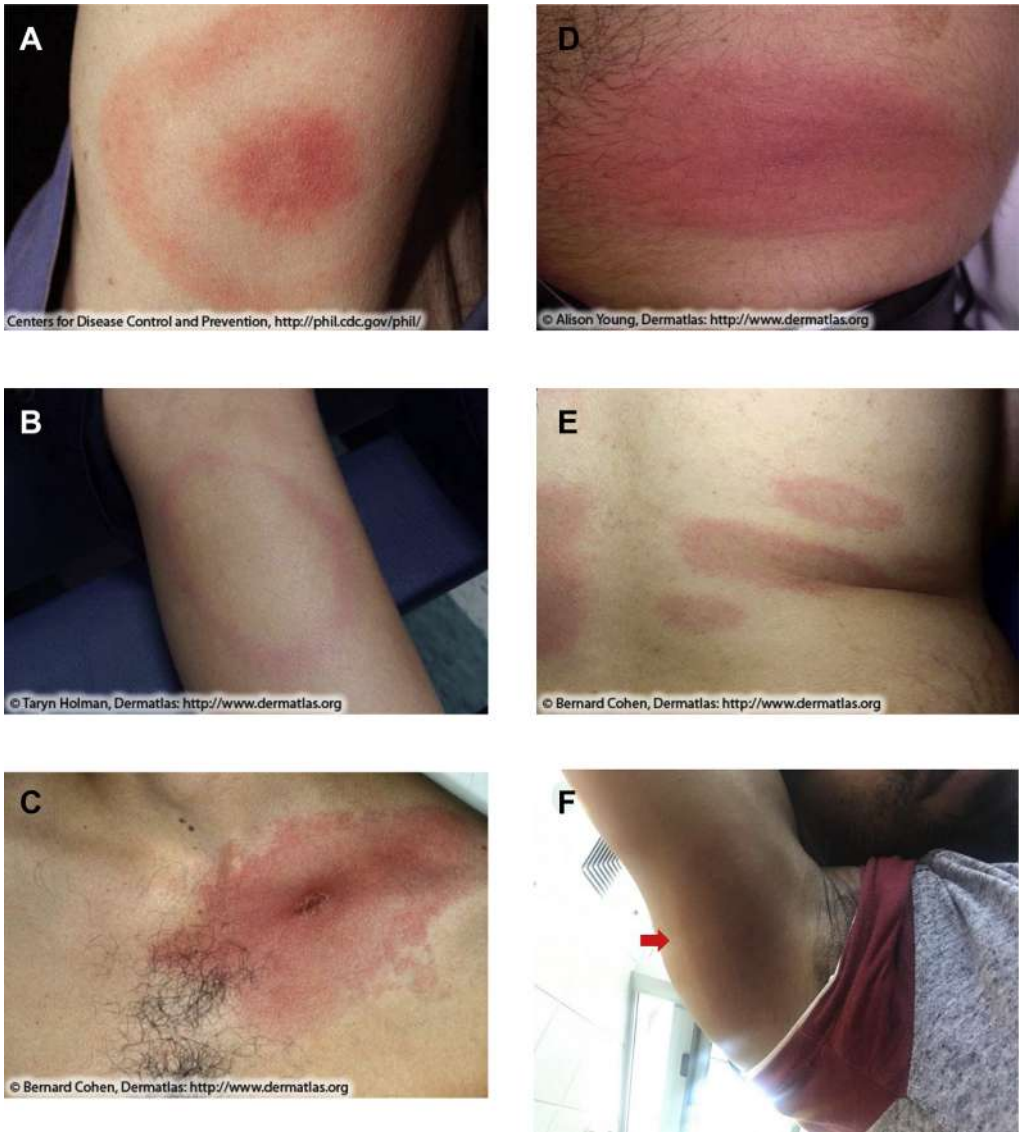

Figure 4. Erythema migrans (EM) is classically described as a flat, blanchable, annular, erythematous skin lesion with or without central clearing, occurring at the site of *B burgdorferi* inoculation. The rash typically develops 3 to 30 d after exposure and can reach up to 30 cm in diameter (Centers for Disease Control and Prevention. Lyme disease. https://www.cdc.gov/lyme/index.html) (A and B).[102] Atypical presentations include vesicular lesions (C), confluent erythematous lesions (D), urticarial lesions, transient EM, and disseminated EM (E), itself a feature of disseminated Lyme disease.[23] EM may be easily misdiagnosed in patients with darker skin (F).[103] Source: United States Centers for Disease Control and Prevention (public domain).

seizure, and localized weakness. The fatality rate approaches 10%. Treatment is supportive.

Other

Alpha-gal Syndrome

Alpha-gal syndrome is a transmissible hypersensitivity reaction most commonly spread by a bite from *A americanum* (the lone star tick) or *I scapularis* (the blacklegged tick). In this recently recognized syndrome,[29] the tick regurgitates the sugar molecule alpha-gal into its human host. In some individuals, the alpha-gal molecule induces a hypersensitivity to meat (and less frequently dairy), which can lead to a spectrum of allergic reactions from hives to anaphylaxis. In contrast to most food-based allergies, which manifest in

minutes, the alpha-gal–induced reaction to meat can be delayed by several hours.

Recommendations for the Prevention and Management of TBIs in Resource-Limited Settings

Frontline providers may encounter TBIs in a variety of scenarios, ranging from the truly austere and remote to an office setting. Our recommendations assume that care is being provided with few resources and therefore focus on simple, medically relevant interventions that can be applied at the individual and community levels to prevent the transmission of TBIs from vector to human host. A cornerstone of preventing TBIs is avoiding tick bites themselves; in a 2019 systematic review, use of insect repellents and protective clothing was associated with lower incidences of Lyme disease when compared to other preventative strategies.[30] However, strategies such as tick checks, timely tick removal, prophylactic antibiotics, vaccination, and education are important in a multimodal strategy for TBI prevention (Table 3). Many large-scale tick control strategies such as vegetation management and host population management are outside of the scope of medical practice and therefore not addressed in this CPG.

LONG-SLEEVED CLOTHING

Although wearing long-sleeved clothing is recommended by the CDC to limit a tick's ability to latch,[31] this strategy has not been directly studied as a means to prevent TBI. However, the intervention is cheap, practical, and has minimal to no risk; therefore, the authors support the use of long-sleeved clothing when traveling in tick habitat.

Recommendation. Wear long-sleeved clothing when traveling in tick habitat. Recommendation grade: Expert opinion.

LIGHT-COLORED CLOTHING

Wearing light-colored clothing has been recommended to improve the visualization of ticks during tick checks, but this relationship has not been formally studied in TBI prevention. In a randomized, cross-over, cohort study of clothing color, the authors found that *Ixodes ricinus* may in fact be attracted to light-colored clothing.[32] In a subsequent case control study of risk factors associated with Lyme disease, the use of light-colored clothing was not associated with a reduced risk of Lyme disease.[33]

Recommendation. Although light-colored clothing may not reduce the risk of tick bites, it does make it easier to identify ticks on clothes during tick checks. Recommendation grade: 2C.

TICK AND INSECT REPELLENTS

Deet

N, N-Diethyl-meta-toluamide, also known as DEET, has been in use since 1956 and has been widely shown to be an effective tick repellent.[34–37] Seizure and neurotoxicity are possible side effects,[38] but with approximately 30% of the US population having used DEET at some point in their lives,[39] the absolute risk is likely quite low.

The concentration of DEET is associated with estimated protection time. For example, 30% DEET will protect the user for an estimated 6 h, whereas 5% DEET offers 2 h of protection. The American Academy of Pediatrics does not recommend the use of DEET in children 2 mo of age or younger, and DEET concentrations of 10% or less should be used in those 12 y of age and younger.[40] Additionally, DEET is known to be corrosive to synthetic or technical fabrics such as Gore-Tex.

Recommendation. DEET is an effective tick repellent. DEET should be reapplied based on the concentration of formulation. Recommendation grade: 1B.

Recommendation. DEET can be used in children over the age of 2 mo. Recommendation grade: 1B.

Picaridin

Picaridin (also known as icaridin) was developed in the 1980s as an alternative to DEET, and it became available in US markets in the mid-2000s. As with DEET, the degree of protection for picaridin is based on concentration[41]: 20% picaridin offers approximately 10 h of protection against arthropods. Although picaridin has a much shorter track record of use than DEET, it appears to be nontoxic and, when compared to DEET, has a superior safety profile.[42] Picaridin is also

Table 3. General tick-borne disease prevention strategies

Personal protection	Wearing long-sleeved shirts and pants, light-colored clothing
	Applying insecticides to skin and/or clothing
	Performing tick checks
	Bathing immediately after spending time outdoors
	Washing and drying clothing at high temperatures
	Prompt tick removal
Community interventions	Educational programs
Medical prevention	Vaccination
	Prophylactic antibiotics
Environmental strategies	Spraying acaricides
	Vegetation management (mowing, clearing leaf litter)
	Host management (fencing, hunting)

odorless and, in contrast to DEET, is not harmful to synthetic fabrics.

Recommendation. Picaridin is an effective tick repellent and is comparably efficacious to DEET. Recommendation grade: 1B.

Recommendation. Picaridin may have a superior safety profile when compared to DEET. Recommendation grade: 2B.

Permethrin

Permethrin is both a repellent and an insecticide that is impregnated into clothing. Permethrin-treated clothing has been thoroughly studied in a number of populations and has been shown to reduce the rate of tick attachment and tick bites in studies of volunteers,[43] military personnel,[44] and forestry workers.[45,46] However, head-to-head studies comparing permethrin to DEET or other repellents suffer from heterogeneous methodologies.[47,48] Overall, permethrin and DEET appear to be comparatively efficacious; the consumer must choose based on cost, relative risk of toxicity, and the timing of their potential exposure. When used in combination, permethrin-treated clothing and topical DEET were found to be more effective in preventing mosquito bites compared to either used alone.[49] Although not specifically studied for tick bite prevention, it stands to reason that the combination of permethrin-treated clothing and topical DEET (or picaridin) may be synergistic to reduce the risk of TBI.

Recommendation. Permethrin-treated clothing is an effective tick repellent. Recommendation grade: 1A.

Recommendation. Permethrin-treated clothing, when used in combination with a skin-based tick repellent such as DEET or picaridin, may further reduce the risk of TBI. Recommendation grade: 1C.

Other Repellents

Several essential oils, citriodiol (oil of eucalyptus), and IR3535 are also commercially available as tick repellents.[50] Others, such as nootkatone, are in development. A review of every available tick repellent is beyond the scope of this CPG. However, each of the aforementioned repellents shares a common trait of either lower repellent efficacy or a significantly shorter duration of action when compared to DEET.[51] Ideally a natural, effective, and nontoxic tick repellent will be available in the future; unfortunately, such a product is not currently available.

Recommendation. Essential oils, citriodiol, nootkatone, and IR3535 have either a lower repellent efficacy or significantly shorter duration of action. Given these attributes, wilderness recreationalists should avoid these products as first-line tick repellents. Recommendation grade: 2B.

TICK CHECKS

Another highly recommended method of personal protection against tick bites is tick checks. The tick check procedure is as follows[52]:

1. Remove clothing for overall visual inspection.
2. Systematically scan the body for ticks, paying special attention to warm places (armpits, knees, under underwear, around hairline of neck, ears, and navel).
3. Remove any identified ticks by grasping at the head of the tick with pointed tweezers or tick remover and pulling perpendicular to the skin (Figure 5).

The data supporting this behavior, however, are scant, and its efficacy has not been rigorously studied. In a study of various personal protective methods in definite, possible, and unlikely Lyme disease patients, no difference in tick check performance was found between patients with Lyme disease and controls, suggesting that inspection is not an effective strategy in preventing Lyme disease.[53] On the other hand, EM-free control subjects were found to be more likely to perform tick checks within 36 h and bathe within 2 h of being outdoors than those with EM, suggesting a protective effect of these behaviors.[54]

Recommendation. Evidence supporting tick checks is contradictory; however, when combined with bathing within 2 h of being outdoors, these measures may help prevent TBI. Recommendation grade: 1C.

Recommendation. Shower or bathe within 2 h of returning from tick habitat. Recommendation grade: 2C.

CLOTHING CARE

In an early study of *A americanum* and *I scapularis* survival after exposure to automatic washer and dryer conditions, all nymphs were killed after 1 h of drying at 40 to 42°C.[55] Although large proportions of nymphs survived hot water washes in this study, a more recent study showed 100% effectiveness of hot water washes at temperatures greater than 54°C (130°F) in killing both nymphal and adult *I scapularis*.[56] These studies did not look specifically at the effect of these interventions on the incidence of TBI, however.

Recommendation. Washing clothes at temperatures over 54°C/130°F and drying clothing in high heat for 10 min kills ticks and therefore may reduce the risk of TBIs. Recommendation grade: 1C.

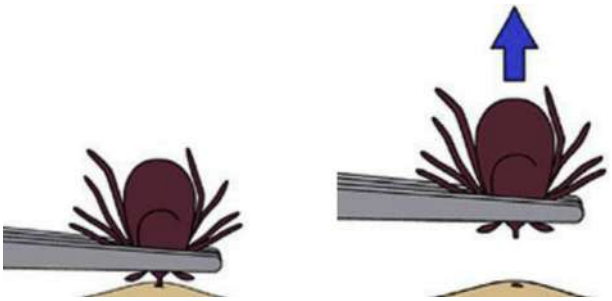

Figure 5. Forceps removal (Centers for Disease Control and Prevention. Ticks. https://www.cdc.gov/ticks/index.html). Source: United States Centers for Disease Control and Prevention (public domain).

OTHER BEHAVIORAL MODIFICATIONS

Although data are limited to support these practices, the CDC recommends several behaviors to avoid either primary contact with ticks or to limit tick latching.

Recommendation. When feasible, avoid areas with high grass or leaf litter. When in tick habitat, walk in the middle of trails to mitigate the chance of contact with ticks. Recommendation grade: Expert opinion.

TICK REMOVAL

A number of anecdotal methods for tick removal have been described,[57–64] inspiring the creation of several commercial devices that use different strategies in grasping and removing embedded ticks. Case series comparing various mechanical tick removal techniques, however, have yielded disparate results.[65–69] Data are mixed on whether the method of tick removal is associated with the occurrence of TBI.[70] However, in a case series, tick removal with forceps was associated with a lower rate of spirochetal and rickettsial infections.[71] Passive methods such as application of petroleum jelly, fingernail polish, 70% isopropyl alcohol, or a hot kitchen match,[67,72] local infiltration of anesthetics,[73] and administration of oral ivermectin[74] failed to exhibit any efficacy in encouraging tick detachment. Overall, mechanical removal is largely accepted by experts,[75] and forceps removal has been endorsed by the CDC (Figure 5)[8]; however, to date no professional organization has adopted strong practice guidelines regarding optimal tick removal techniques.[76]

Recommendation. Mechanical removal by pulling upward, or perpendicular to skin, directly on an embedded tick with forceps is the best currently available method. Mechanical removal using commercial devices may also work, but evidence suggesting superiority does not exist. Recommendation grade: 1C.

Recommendation. Pulling embedded ticks with straight, steady pressure is preferred over a twisting motion.[8] Recommendation grade: 1C.

Recommendation. Passive removal techniques, particularly chemical strategies that involve exposing attached ticks to petroleum jelly, fingernail polish, isopropyl alcohol, gasoline, or methylated spirits are ineffective and not recommended. Recommendation grade: 2C.

Recommendation. Using local or systemic medications such as locally infiltrated anesthetics or systemic ivermectin is not effective in removing or exterminating attached ticks. No evidence exists to suggest any benefit to these strategies. Recommendation grade: 2C.

TIMING OF TICK REMOVAL AND RISK OF LYME TRANSMISSION

Prophylactic strategies for Lyme disease have been modeled on the risk of infection after a tick bite relative to the length of time the tick is attached to its host. Generally, the risk of Lyme disease transmission is correlated with the duration of tick attachment. Although the risk of transmission of *B burgdorferi* after a tick bite in Lyme endemic regions is estimated to be 1 to 3%,[77] the risk increases to 20% when infected *I scapularis* ticks are attached longer than 72 h.[78] Early animal studies of *I scapularis* ticks infected with *B burgdorferi* suggested that at least 48 h of attachment were necessary for transmission of disease.[79–81] However, a recent review highlighted that Lyme transmission is possible within 24 h of attachment and that a definitive study describing a minimum tick attachment time for the transmission of Lyme in humans has not been published.[82] Until these data are available, the CDC recommends tick removal within 36 h of attachment to reduce the risk of Lyme disease.[8]

Recommendation. Once discovered, ticks should be removed as soon as possible. To meaningfully reduce the risk of Lyme disease, ticks should be removed within 36 h of attachment. Recommendation grade: 1B.

PROPHYLACTIC ANTIBIOTICS FOR LYME DISEASE

Efforts were made in the 1990s to determine whether prophylactic antibiotics could prevent the development of Lyme disease in patients after an *I scapularis* tick bite. In a randomized, placebo-controlled trial comparing a single 200 mg dose of doxycycline to placebo in patients who had been exposed to *I scapularis* for less than 72 h, 8 of 247 (3%) placebo patients developed EM, compared to 1 of 235 (0.4%) patients receiving doxycycline, a statistically significant reduction in EM in treated patients.[83] A

more recent meta-analysis including 1082 patients revealed a 2% risk of Lyme disease in placebo patients versus a 0.2% risk of Lyme disease in those receiving prophylaxis, although the number needed to treat to prevent 1 case of Lyme disease was 49.[84] A more recent randomized controlled trial supports the conclusion that doxycycline prophylaxis can reduce the risk of Lyme borreliosis.[85] In their guidelines, the Infectious Disease Society of America recommends a single dose of doxycycline 200 mg within 72 h of tick removal for adults and children older than 8 years if the following high-risk criteria are met: (1) the tick bite was from an identified Ixodes vector species, (2) it occurred in an endemic area, and (3) the tick was attached for ≥36 h.[86]

Recommendation. A single dose of 200 mg doxycycline orally is recommended after a high-risk tick bite if given within 72 h to reduce the risk of Lyme disease. Recommendation grade: 1B.

Recommendation. If a provider is unable to identify the tick, or if the time of attachment is unknown, then a period of "watchful waiting" is recommended instead of prophylaxis. Should the patient develop fever, EM, or arthralgias within 30 d of the presumed tick bite, treatment with doxycycline can then be initiated. Recommendation grade: 1B.

PROPHYLACTIC ANTIBIOTICS FOR OTHER TBI

Currently, there are no compelling data to suggest that antibiotic prophylaxis is effective for any TBI other than Lyme disease.[8]

Recommendation. Providers should not employ prophylactic antibiotics for management of anaplasmosis, ehrlichiosis, Rocky Mountain spotted fever, relapsing fever, or any other TBI. Outside of high-risk Lyme disease exposures, prophylactic antibiotics are not indicated. Recommendation grade: 1C.

THE DECISION TO EVACUATE

The decision to evacuate versus expectant monitoring can be the most important decision the provider can make in resource-limited settings. This decision involves a careful balance of risks and benefits, including consideration of the possible harms of an inaccurate diagnosis and the potential for delayed care. No randomized controlled trials exist comparing field management to evacuation for patients with potential systemic TBI. Available cohort studies from the National Outdoor Leadership School describe a low incidence of TBI in the field, but in nearly all cases those with suspected TBI were evacuated.[87–89] This is appropriate because management of TBI requires accurate clinical and laboratory diagnosis, prompt treatment, and close follow-up. Life-threatening complications such as Lyme carditis could also potentially develop within the timeframe of a multiday backcountry trip. An algorithmic decision tree outlining the field management of tick bites is summarized in Figure 6.

Recommendation. Individuals who develop systemic or high-risk symptoms (fever, generalized rash, arthralgias, cranial nerve palsy, dyspnea, or syncope) related to a suspected TBI should be evacuated to a higher level of medical care. Recommendation grade: 1C.

Recommendation. Individuals who develop symptoms suggestive of Lyme carditis such as dyspnea, dizziness, or syncope should receive a screening ECG as soon as possible and would benefit from a thorough cardiovascular evaluation in an appropriate clinical setting. Recommendation grade: 1C.

VACCINATION

Vaccination strategies offer an attractive option for disease control both at the individual and the public health level. The efficacy of a tick-borne encephalitis vaccine, available in central Europe, Russia, and China, has been well documented.[90,91] Vaccine programs for TBIs in the United States, however, have had limited success. In 1999, 2 large randomized controlled trials of Lyme disease vaccines based on the *B burgdorferi* outer surface protein (OspA), both with and without adjuvant, found the vaccines to be highly efficacious in preventing Lyme disease in endemic populations.[90,92] These vaccines, however, were removed from the market in 2002 due to low sales related to the need for frequent boosters, musculoskeletal side effects, high cost, and litigation.[91]

Vaccine development continues despite these shortfalls. There is commercial and scientific interest in developing a vaccine that could protect against both North American and European *Borrelia* serotypes.[93] Although a candidate vaccine using fused OspA molecules is no longer in development,[94] other candidates involving other OspA and OspC antigens are currently under study.[95–98]

Recommendation. While no vaccine for tick-borne encephalitis is currently available in the United States, vaccines such as Encepur appear to be efficacious for inducing seroconversion against tick-borne encephalitis. Recommendation grade: 2A.

Recommendation. Although not currently available in the United States, Lyme vaccination is efficacious at reducing the risk of infection. Recommendation grade: 2A.

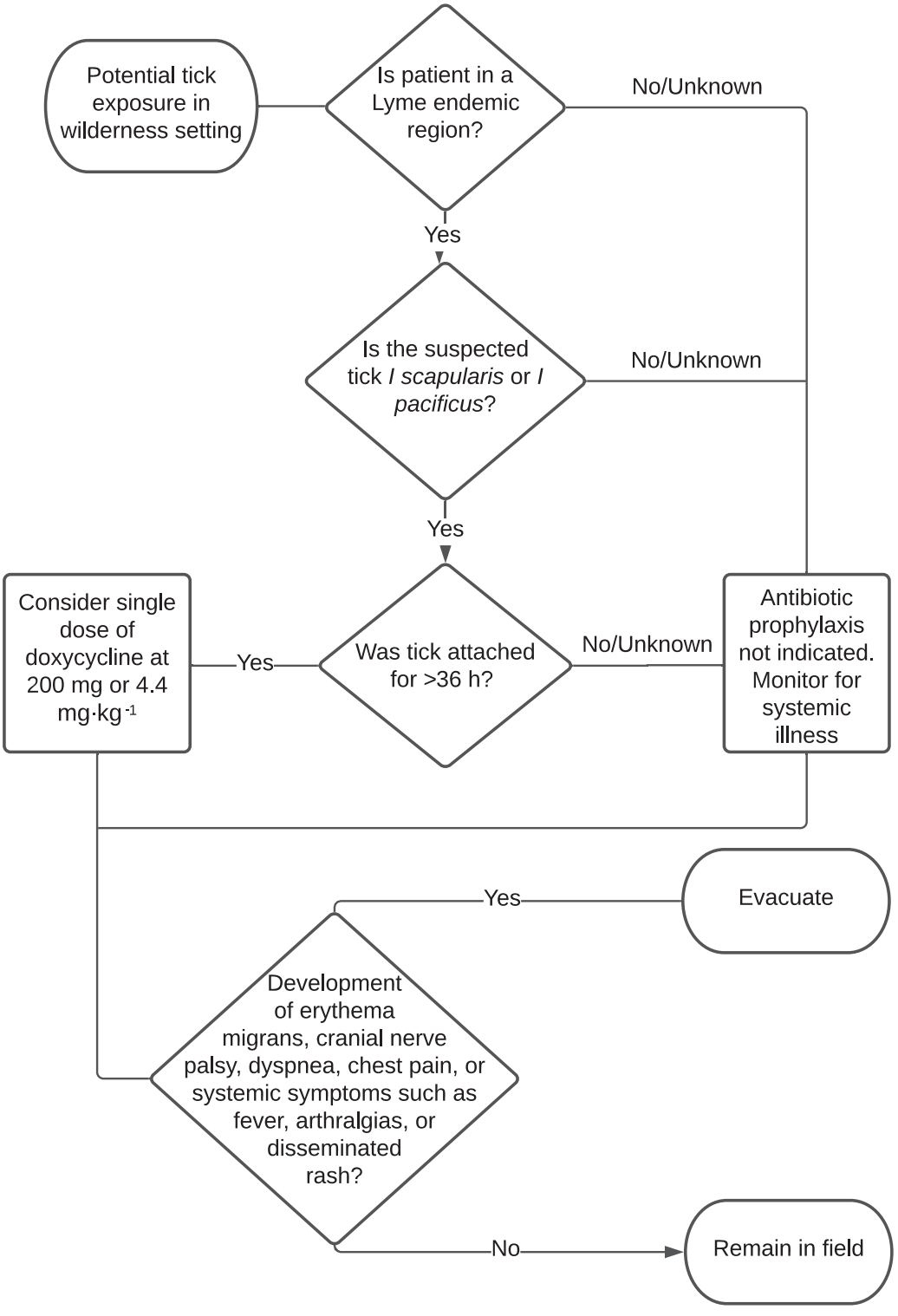

Figure 6. Wilderness management algorithm for tick-borne illness.

EDUCATIONAL PROGRAMS

TBI prevention behavior is inconsistent among individuals; in a cross-sectional study assessing knowledge, attitudes, and behaviors regarding TBI prevention strategies among persons living in Lyme-endemic areas, behaviors ranged widely from the use of tick control products on pets (83%) and tick checks (58%), to lower rates of compliance with showering or bathing after spending time outdoors (42%), applying insect repellents (31%), and using chemical or natural pesticides on yards (23 and 15%, respectively).[99] Simple educational programs, however, have been shown to be effective not only in changing behavior, but also in reducing disease burden. In a randomized controlled trial, individuals who received a 15-min Lyme disease and tick specific educational presentation were found to have lower rates of TBI compared to those receiving a control program.[100] Given the vast range of preventative behavior and the costs of TBIs to individuals and communities, programs such as these provide an effective and economical method for disease prevention.

Recommendation. Educational programs can change behavior and lower rates of TBI and should be encouraged. Recommendation grade: 1B.

Conclusion

TBI is a broad medical topic, influenced by the environment, geography, climate, ecology, and animal and human behavior. Given the interactions between humans and the environment, TBI cannot be completely avoided. However, with certain behavioral and medical adaptations, the overall burden of disease related to TBIs can be reduced. With a changing climate that continues to influence the epidemiology of TBI, promotion and strict adherence to simple prevention measures is important. The recommendations presented in this CPG are largely consistent with those presented by the CDC (https://www.cdc.gov/ticks/index.html) and other practice guidelines,[76] but they specifically highlight concepts most relevant to providers who encounter ticks in backcountry, austere, and limited-resource settings. Despite the limitations of the existing literature, these guidelines provide a starting point for front-line providers to mitigate the transmission and reduce the disease burden of TBIs through low-risk interventions.

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CONCEPTS

Should Airbag Backpacks Be Standard Avalanche Safety Equipment?

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Avalanche airbag backpacks have been shown to be effective at reducing avalanche mortality. However, they are yet to be considered standard avalanche safety equipment, which has long consisted of a transceiver, a shovel, and a probe. This is despite data showing that airbags reduce mortality by decreasing the likelihood of burial. In addition, airbags probably lessen trauma and possibly delay asphyxia. Moreover, the literature suggests airbags reduce mortality at a rate similar to transceivers. For those who work, volunteer, and recreate in avalanche terrain, airbags should be considered standard safety equipment. However, multiple barriers exist for universal adoption, including cost, size, weight, training burden, availability, risk tolerance, and lack of community support and recommendations from professional societies and associations.

Keywords: snow burial, beacon, transceiver, emergencies, self-rescue

Introduction

The avalanche airbag backpack was first patented by German skier Peter Aschauer nearly 4 decades ago. Early inspiration was reportedly based on the experience of forest ranger Josef Hohenester, who found that when he hauled large game on his back, he floated more easily in snow. Aschauer first demonstrated his airbag at the Internationale Fachmesse fur Sportartikel und Sportmode (international trade fair for sporting goods and sportswear) in Munich in 1985. After nearly 4 decades, airbag design and functionality have been refined.

Avalanche airbags work based on the property of particle physics called granular convection. In a flow, particles of larger volume migrate to the top. Airbags increase the volume of users by 150 L or more and thus assist in keeping the user at the top of avalanche debris.

Airbags are widely available in Europe and North America. Airbags use 2 general systems, inflated by either compressed gas canisters or electronic fans. Canister airbags come in multiple configurations and with various gasses, such as air, nitrogen, argon, and carbon dioxide. In some models, compressed air canisters are refillable. Other systems use 1 or 2 single-use disposable canisters. Electronic fan airbags are powered by a rechargeable lithium-ion battery or a capacitor with battery backup. Neither the canister nor the fan system has been demonstrated to be superior in preventing burial. Both add significant weight, bulk, and expense compared to a standard, non-airbag backpack.

The benefits of canister airbags are that they tend to be lighter and less expensive. The downside is that canisters are not easy to refill or replace, are not standard among manufacturers, and can be limited for airline travel and shipping by regulations, especially in North America. For multiple deployments, canisters pose difficulty in terms of practice, rearming an airbag that needs to be used more than once, and sharing extra canisters in groups that use different airbag brands. The addition of extra canisters to a backpack increases both cost and weight.

The benefits of an electronic fan airbag are that multiple deployments and airline transport are simpler. This makes practice easier and less expensive. A potential problem is that they rely on power and electronics, which may be affected by cold temperature and availability of

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power for charging. Fan airbags are also more costly and heavier than canister airbags.

Published Recommendations

Avalanche airbags are used in backcountry areas around the world. Many organizations have provided recommendations regarding their use. Because of the varied infrastructure, legal, and governmental issues around the world, this review will only examine selected current recommendations in North America.

In 2017, a Wilderness Medical Society (WMS) clinical practice guideline reviewed the avalanche literature, which demonstrated the effectiveness of airbags but did not give a solid recommendation.[1] The recommendation was twofold:

> *Travelers entering avalanche terrain should consider using an avalanche airbag. Familiarity and regular practice with airbags is essential. Grade: 1B (strong recommendation, moderate-quality evidence).*
>
> *Airbag backpacks may or may not provide head and neck protection from trauma. Ungraded: No Recommendation.*

Similarly, the International Commission for Alpine Rescue has a recommendation from 2006 that supports airbags.[2] At that time, the majority of members of this global organization were from Europe and the recommendation was completed by consensus:

> *The efficiency of the transceiver in combination with probe and shovel, and of airbag systems has been proven.*

Additionally, the textbook used by the WMS Diploma in Mountain Medicine course omits mention of airbags altogether.[3]

The 3 main North American avalanche organizations—the American Avalanche Association (A3), Canadian Avalanche Association (CAA), and American Institute for Avalanche Research and Education (AIARE)—have no formal recommendation for airbag use on their websites or in their publications, as confirmed by published information, websites, and personal communication with the 3 agencies. For the A3, airbags are part of the core curriculum for only 1 course, titled Avalanche Rescue; this is 1 of 5 courses that range from basic to advanced. For the AIARE, airbags are considered "additional safety equipment" in the student manual.[4] In the CAA risk handbook, airbags are mentioned only once in discussion of risk statistics.[5]

Overview of Literature

Like many topics in wilderness and mountain medicine, the literature on avalanche airbags is sparse. Several key studies regarding airbags will be highlighted. One study found that airbags reduced mortality from 19 to 3%.[6] Another study reported on 245 accidents involving 424 injuries and a 19% overall mortality rate.[7] A subset of the data looked at 66 accidents with both airbag users and non-airbag users. In this subset, all noncritically buried fatalities were due to trauma. Mortality reduction of critical burials decreased from 34 to 11% with use of an airbag.[7] (Critical is defined as the head being under snow and breathing impaired; noncritical is defined as an unobstructed airway.) Of note, airbag deployment failure and device failure were found to be significant in both studies. One study found a 20% failure rate (7 of 35 participants),[6] and another found a 20% failure rate (61 of 307 participants)[7]; this was due to user error, device failure, or device damage during avalanche.

A third study used a series of planned avalanches to test dummies equipped with and without airbags.[8] Of the 5 dummies without airbags, burial depth was a mean of 43 cm, and only 1 of 5 was visible from the surface. In contrast, of 14 dummies with airbags, burial depth was a mean of 15 cm, with all 14 visible from the surface. This study also found that among the 14 airbag dummies, the head was visible for all but 5 and the airway was less than 10 cm below the surface for all but 2.

Similarly, the literature on transceivers is limited and outdated; most studies on transceivers were conducted before widespread use of modern technology, including digital processing and 3 antennas. Nonetheless, avalanche transceivers have been shown to reduce mortality. One study showed transceivers reduced time from burial to companion rescue from 120 to 30 min but did not yield a significant reduction in mortality.[6] Another showed that burial time was decreased from 102 to 30 min and mortality from 68 to 54% with use of a transceiver.[9] A third study confirmed burial time reduction from 125 to 25 min using a transceiver and a mortality risk reduction from 70 to 55%.[10]

Barriers

The important messages from these studies are twofold. First, airbags are successful at decreasing mortality from avalanches, mostly in preventing critical burials. As with any device, user error and equipment failure are substantial problems. Second, airbags' mortality-rate reductions are similar to transceivers, with the limitations discussed previously. However, airbags help prevent burial, whereas transceivers are employed once a person is caught in an avalanche and buried.

Despite these studies, airbags are still not universally adopted as standard avalanche safety equipment. Several barriers exist to universal use of airbags.

AVAILABILITY AND EQUIPMENT CHALLENGES

Airbags can be limited in availability in some regions. This has probably made adoption slow. The availability of canisters, which must be shipped as hazardous material in the United States, is limited. Refilling canisters, which can be done at a retail shop or at home with a high-pressure pump, is also difficult, especially for users who live in rural areas with no access to authorized sales and refilling vendors. Sharing canisters among groups with different brands and models is difficult, as is carrying extra canisters. Fan airbags need to be charged; thus, access to power is needed. Both canister and fan airbags can be affected by cold temperatures. For both fan and canister models, electronic and mechanical parts can fail.

SIZE AND WEIGHT

Airbags are significantly heavier and bulkier than standard, non-airbag backpacks. Whereas an average backpack can weigh as little as 0.5 kg, the lightest airbags are still around 2 kg. This is a significant issue for the weight-conscious mountain traveler who may have a full backpack of mountaineering and rescue equipment.

COST

Airbags are significantly more costly than standard packs. A backcountry ski backpack can cost as little as $100 USD, whereas airbag backpacks range from $700 to > $1000 USD.

TRAINING

Airbags require additional training. For recreational enthusiasts, this means another device to learn how to use and practice with. For professionals, this adds extra training to an already complex mountain safety program. For avalanche schools, this adds more training and higher cost to have all students equipped with airbags.

COMMUNITY USE

The promotion of airbags in local communities via equipment retailers, social media, and professional organizations can play a role. If a community has an important educational organization or mountain shop that promotes airbag use, airbags may be adopted more readily than if the community has no local promotion or awareness.

RISK TOLERANCE

Airbags may increase risk tolerance for users and thus may be both a deterrent and a perceived benefit for users. Two studies suggest some airbag users may take more risks when traveling in avalanche terrain.[11,12]

GUIDELINES

Professional societies and associations, in scientific and lay publications and in textbooks, have neither recommended airbags as standard equipment nor provided much education or guidance about them in curricula. This likely plays a large role in the lack of universal use.

Knowledge Gaps

Aside from preventing burial, other questions worth considering are discussed in the following. These issues are important because they may contribute to lack of adoption of airbags.

DOES BALLOON SIZE MATTER FOR FLOTATION?

A smaller balloon may be less expensive and lighter and thus more likely to be used. The standard is a 170-L balloon. A larger balloon may be more effective at keeping a user atop avalanche debris, but it makes weight and cost higher and design more difficult, including the need for a larger fan or canister. Similarly, a smaller balloon maybe be less expensive and lighter, but it may not work as well to lessen morbidity and mortality.

DO AIRBAGS PROTECT FROM TRAUMA?

Airbag use may be encouraged if data demonstrate protection from trauma, considering trauma accounts for 25% of fatalities.[2] More studies could address this. Anecdotal cases suggest that a 170-L balloon does offer some degree of trauma protection for the head, neck, and torso. The WMS clinical practice guidelines did not reach a conclusion on this issue.[1] Similarly, balloons that extend above and wrap around the head and neck may protect one better than those that do not, but no data exist on this issue.

DO AIRBAGS CREATE AN AIR POCKET AND/OR PROTECT THE AIRWAY TO DELAY ASPHYXIA?

If airbags can help prevent asphyxia, as with trauma, they may be adopted more readily. We know that air pockets and patent airways help prolong survival.[13-15] One brand and model of fan airbag deflates after inflation. In a recent study, researchers buried 12 volunteers with an airbag-created air pocket and measured oxygen saturation, end tidal carbon dioxide, heart rate, and respiratory rate.[16] Eleven volunteers were observed for 60 min. The burial experiment was ended for the 12th volunteer owing to an increased heart rate, increased end tidal carbon dioxide, and anxiety. Participants with a simulated air pocket created by an airbag were able to move their head an estimated 11 cm forward and 7 cm backward.

The study concluded that it is possible that an air pocket from a deflated airbag could prolong survival. Again, more studies are needed to address this question.

SHOULD CANISTERS BE STANDARDIZED?

If canisters and actuation systems were standardized, use of airbags could increase because users would be able to more freely trade canisters. Canisters are available in a variety of sizes, proprietary actuation systems, and gasses. Some companies use different canisters for North American and European versions of their airbags owing to regulations regarding shipping and importing compressed gas canisters.

Conclusions

Airbags have been shown to be effective at saving lives. At first glance, they seem to be equal to transceivers in reducing mortality. Yet studies on airbags are limited, and studies on transceivers are outdated. Even so, with both transceivers and airbags, the absolute mortality rate reduction is small. Notwithstanding these limitations, airbags clearly save lives but are not yet considered standard avalanche safety equipment owing to barriers as described above. Some of these issues can be addressed. As with many products, as demand increases, availability may increase. Cost, weight, and size are largely dependent on the market and the manufacturer, but airbags continue to become less expensive and lighter.

Other difficulties can be addressed largely with education programs. This includes local organizations and retailers to promote use, avalanche education schools to promote training, and professional societies and associations to highlight importance.

Based on the literature, if one wants to maximize safety in avalanche terrain, one should use an airbag in addition to other standard safety tools. An airbag is an effective tool at reducing mortality and is the only tool that helps prevent burial.

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CASE REPORT

Massive Tick Bites Causing Spotted Fever Rickettsial Infection: A Hazard in a Tea Plantation, Sri Lanka

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Tea plantations in Sri Lanka cover the central hills of the island, where spotted fever group (SFG) rickettsial infection is common. In most cases, the history of tick bite is obscure and eschars are not present. A 45-y-old female experienced massive tick bites while working in her tea plantation. She developed fever 2 d after exposure, but the diagnosis of SFG infection was not considered until a skin rash appeared on the eighth day. She had a very high titer of antirickettsial antibodies detected by immunofluorescence assay and responded to doxycycline. Here, we highlight the high risk of exposure to ticks and tick bites within tea estates and its causal relationship to SFG infection, which is increasing in Sri Lanka. Active case detection, notification, surveillance, and community awareness are imperative. Possible preventative measures for tick bites have to be introduced. There is a need to explore the effectiveness of local remedies currently in use.

Keywords: rickettsioses, occupational hazard, environmental hazard, tick infestations, re-emergence

Introduction

Sri Lanka, an island in the Indian Ocean situated at latitudes 5° and 10°N and longitudes 79° and 82°E, has been known for its tea industry from the industry's inception in 1867 in the central highlands when historic coffee plantations began to fail. The tropical climatic conditions and low temperature in the high altitude hilly terrain are conducive for an abundance of both flora and fauna.

Early reports of rickettsial infections in Sri Lanka found that tick-borne spotted fever group (SFG) infection is widely distributed in the hilly central province of the island.[1,2] Classically, patients with SFG rickettsioses present with fever and a skin rash. Fever is generally of abrupt onset and is high grade and intermittent, and the skin rash is erythematous and maculopapular, often with the presence of eschar.[1-4] In most cases of SFG rickettsial infections, history of tick bite is obscure and eschars are not apparent. Therefore, the link of the 2 entities is not very apparent. Tick infestations and tick bites are so common that people tend to ignore them. The ticks commonly attracted by humans are *Amblyomma integrum* or *Dermacentor auratus,* particularly in Sri Lanka (Figure 1). Rickettsiae are groups of gram-negative coccobacilli found as obligatory intracellular pathogens that require eukaryotic cells to proliferate.[3,4] Today, rickettsial infections are endemic in Sri Lanka and burden the health care system with an increasing number of cases.[1,2,5] The aim of this case report is to show the burden of tick infestation in the highlands of Sri Lanka and its direct link to SFG, a diagnosis often overlooked.

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Case Presentation

A 45-y-old previously healthy female from a hamlet in the hilly central province of Sri Lanka presented to the Teaching Hospital, Peradeniya, with a 9-d history of high-grade fever. She was a housewife and worked in her own home-garden tea plantation. Her tea plantation was about 1 acre in size and had provided a living for the family over the past 30 y. Family members together

Figure 1. Adult female tick of *Dermacentor* species (adapted from Kularatne et al, 2018[5]; reprinted with permission from BMC).

Figure 2. Tea plucking in central hills of Sri Lanka (photograph courtesy of Mr. Iroshan Weerakoon, MBA, deputy general manager of Maskeliya Plantations PLC in Sri Lanka).

maintained the tea bushes and plucked leaves alternatively in 4 divided plots. They plucked tea leaves in a quarter of the land and harvested about 350 kg of leaves in the course of a month. The tea harvest was sold to a nearby tea factory to generate their family income. The family house was located at a corner of the tea garden, which had a concrete slab as the roof and a cement floor. Her neighbors also had similar small tea estates, and there were no well-developed roads to access their houses directly by automobile (Figure 2). She did not have pet cats or dogs at home, but it was usual to see wild animals such as monkeys in the daytime and wild boar and small deer at night.

Three days before the onset of fever, she worked in the tea plot plucking tea leaves as usual. In the afternoon, she had to take her son to the local hospital in a rush, postponing her usual bath after the day's work. The next morning, she developed itching of the skin on her abdomen. Immediately, she had her daughter examine her; the daughter found more than 50 tiny ticks firmly attached to her skin where there was itching, including the abdomen and uncovered area of her back between her skirt and blouse.

There were no ticks attached under the areas covered by clothing. The patient applied a medicinal oil called *Sarwawishadhiya* (made of multiple herbal plant extracts) to the tick-infested skin, left it for a while, and rubbed off the ticks. The patient revealed that this particular medicinal oil is available in the local shops and is used for a number of ailments as a quick remedy. It is also believed to be a good repellent of ticks and leeches, according to her experience. The patient believed that wearing long-sleeved tops and long dresses up to the ankle helps to prevent tick bites. Generally, tea pluckers work barefoot and apply lime or coconut oil as repellents on their feet.

Two days after the tick bites, the patient developed a fever and was admitted to a nearby peripheral hospital; she stayed for 3 d and then went home. This initial presentation had been unremarkable except for fever, and no further records could be retrieved. However, fever recurred 2 d later (day 7 after tick bite), and she was readmitted to the same hospital, where she stayed an additional 2 d. Because she was becoming increasingly ill, she was transferred by ambulance to Teaching Hospital, Peradeniya.

In addition to high fever, the patient had severe headache, significant upper and lower limb joint pain, myalgia, difficulty walking, back pain, lethargy, nausea, and vomiting. There was no icterus or lymphadenopathy. Her blood pressure was 100/60 mm Hg, and her pulse rate was 88 beats·min^{-1}. Newly developing skin rash on both upper and lower limbs was noted on the 10th day after tick bite. On the 15th day, she had a distinctive and discrete erythematous macular rash over both upper and lower limbs. She did not have any eschar. She had insomnia due to the joint pain.

Basic laboratory investigation findings were as follows: white blood cell count $10.81 \times 10^3 \cdot \mu L^{-1}$, neutrophils $6.34 \times 10^3 \cdot \mu L^{-1}$, and lymphocytes $3.46 \times 10^3 \cdot \mu L^{-1}$) on the 11th day of illness. The lowest platelet count was $65 \times 10^3 \cdot \mu L^{-1}$ on the eighth day of fever. Erythrocyte sedimentation rate (ESR) was 64 mm·h^{-1}, and the

C-reactive protein level was 157.1 $mg \cdot L^{-1}$. Alanine transaminase was 41 $u \cdot L^{-1}$, and aspartate transaminase was 129 $u \cdot L^{-1}$. No significant abnormality was found in the urine full report. The clinical diagnosis of tick-borne SFG rickettsial infection was made; this was confirmed with strongly positive IgG antibodies against *Rickettsia conorii* on an immune fluorescence antibody test. The titer value was 1:2460. The patient was treated with oral doxycycline 100 mg twice a day for 7 d, which resulted in defervescence and clinical improvement within 24 h.

Discussion

We found tick infestation and bites to be an environmental and occupational hazard in tea plantations in Sri Lanka. Reports of massive tick bites of this nature are not found in the literature, despite their frequent occurrence. In addition to transmission of rickettsial infections, tick bites can lead to skin ulceration, secondary infections, and related complications. Vulnerable populations have their own remedies for tick bites and bite prevention, including wearing protective clothing, applying locally identified plant materials, and avoiding potentially risky areas. However, such methods, particularly the local application of plant materials, have not been specifically tested for their efficacy, and these avenues need to be explored.

A bath with strong soap and water at the end of the day's work helps to remove ticks from the skin. As done in this case, rubbing off ticks is contrary to standard protocol and can irritate the ticks, causing worsened disease. The proper method for tick removal is to grasp the tick as close as possible to the skin to where it is attached, to slowly withdraw the tick from the skin using fine-toothed forceps, and to clean the area once done.[6] People generally do not seek hospital treatment for tick bite except for otoacariasis (ticks in the ear), which is a common problem in the central province and can even cause unilateral facial palsy.[5]

Our patient had a confirmed diagnosis of SFG rickettsial infection with gradual development of known clinical manifestations. She developed a fever 3 d after massive tick bites, which may imply a very short incubation period of SFG rickettsial infection. An unnoticed tick bite in the preceding days could also have been responsible for the SFG infection. In her clinical course, the eruption of the skin rash took about 5 to 8 d and helped with the clinical diagnosis, which was later confirmed with very high titer of immunofluorescence assay (IFA) specific for SFG. She responded well to specific antibiotic therapy with doxycycline and commenced her usual activities. Delays in diagnosis may lead to multiple organ failure and even fatality.[3,7,8] Therefore, a history of tick bite should be inquired in any nonspecific febrile illnesses in Sri Lanka to help in early diagnosis and prompt patient care.

The tick population in Sri Lanka comprises about 31 species under 11 genera.[9,10] Among them, genera such as *Dermacentor*, *Amblyomma*, *Hyalomma*, *Boophilus,* and *Rhipicephalus* are recognized vectors of a range of pathogens causing both animal infections and zoonotic diseases in humans. Ticks take a blood meal by deep attachment to the host over a period of days during their 3 stages of development: larva, nymph, and adult. Ticks in early stages of development are often difficult to notice with the naked eye.[9,10] Because all stages are capable of transmitting disease, there is a high chance of spread of infections due to unnoticed tick bites. Ticks have a wide range of hosts, including domestic animals and humans. Ticks are also found in colonies in the leaves of many plants, including tea bushes, and wait to catch a host, including reptiles, birds, and humans. Farmers and field workers sometimes notice these tick colonies in plants and spray chemicals to kill them. Ticks attach in vascular areas or extremities. Tick attachments are so common in Sri Lankan tea plantations that they are rarely reported; however, when ticks infest the ear canal (otoacariasis), infected individuals come to ear, nose, and throat clinics for removal.[11] There is changing ecology in the hills of Sri Lanka, including temperature changes and expansion of dwellings toward the wilderness, which potentially has led to an increasing tick population and increased contact with humans.[12]

Obvious re-emergence of rickettsial infection in Sri Lanka began in the 1990s but was low in prevalence in some areas. During the early 1940s when Sri Lanka was a British colony, an outbreak of scrub typhus fever was reported.[13,14] Since then, there were no reports until the 1990s when the spread of rickettsial infections (particularly SFG) was detected in the western slope of the hilly central province.[13,15] This was then followed by many publications describing varied epidemiologic and clinical characteristics, including neurologic, hematologic, musculoskeletal, and cutaneous manifestations; however, further research regarding specific pathogens responsible for rickettsioses and the mode of transmission in Sri Lanka is still necessary.[1,2,5,16,17]

Conclusions

The emergence and spread of rickettsioses in Sri Lanka have posed threats across different communities, from rural outback to urban cities. In this case report, we draw attention to the impact of tick bites and their medical complications in a vulnerable community. Knowledge of

tick-borne diseases, particularly in the local setting, is sparse. As demonstrated in this report, tick bites and associated complications are a significant occupational hazard. Hence, it is imperative to raise public awareness about the potential dangers of tick bites and their complications, including transmission of infectious diseases, and the need to minimize or avoid potential encounters. Active case detection, notification, surveillance, and community awareness are imperative. Possible preventive measures for tick bites have to be introduced, and there is a need to explore the effectiveness of potential remedies considered by the local communities; if proven effective, these should be promoted. Moreover, it is of vital importance to increase awareness among clinicians of the varying epidemiologic and clinical manifestations of these infections to help in early detection and precise management to prevent associated morbidity and mortality. This will help in improving the health of labor communities in plantations, who contribute immensely to the economic development of the island.

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CASE REPORT

Successful Non-Extracorporeal Rewarming from Hypothermic Cardiac Arrest: 2 Cases

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Accidental hypothermia (core temperature <35°C) is a complication in persons who have fallen into crevasses; hypothermic cardiac arrest is the most serious complication. Extracorporeal life support (ECLS) is the optimal method for rewarming hypothermic cardiac arrest patients, but it may not be readily available and non-ECLS rewarming may be required. We report the medical course of 2 patients with hypothermic cardiac arrest, each of whom had fallen into a crevasse. They were treated successfully with non-ECLS rewarming using peritoneal and thoracic lavage. We discuss non-ECLS treatment options for hypothermic cardiac arrest and describe successful non-ECLS rewarming in an outlying hospital without ECLS rewarming capability in the Grossglockner region of Austria in 1990 and 2003. Both patients survived neurologically intact. Non-ECLS rewarming in a trauma center without ECLS capabilities is feasible and can result in a good outcome when ECLS is not available. The best non-ECLS rewarming method for hypothermic cardiac arrest patients has not yet been established. Non-ECLS rewarming should be adapted to local capabilities. To obtain more robust evidence, it seems reasonable to pool data on the treatment and outcome of non-ECLS rewarming in hypothermic cardiac arrest patients.

Keywords: cardiopulmonary resuscitation, defibrillation, extracorporeal life support, accidental hypothermia, out-of-hospital cardiac arrest

Introduction

Accidental hypothermia, defined as a core temperature ≤35°C,[1-4] is a serious complication of a fall into a crevasse. The risk of hypothermic cardiac arrest in young healthy persons increases substantially at core temperatures below 28°C.[2,3] Extracorporeal life support (ECLS) is the optimal method for rewarming patients in hypothermic cardiac arrest.[1-4] Access to an ECLS center can be prevented by transport times longer than 6 h[4] or dangerous or impossible transport due to bad weather or darkness. ECLS might not be available when it is being used for other patients, such as when there are multiple patients with hypothermia.[5] Non-ECLS rewarming may be required in a hospital that has no ECLS-capability.[5,6] There are no standard guidelines on how to perform non-ECLS rewarming in a hypothermic cardiac arrest patient. We report the successful non-ECLS rewarming of 2 patients with hypothermic cardiac arrest. Discussions with colleagues made us aware that many previous cases of hypothermic cardiac arrest have not previously been published. Although the incidents in this article occurred in 1990 and 2003, they were never reported. Several important lessons can be learned from these cases.

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Case Reports

CASE 1

On a warm day in August 1990, a 21-y-old male was hiking with 2 friends in the Austrian Alps. He was wearing only a t-shirt, shorts, and light hiking shoes. At 1235, the 3 hikers were traversing a glacier at 2920 m when an ice bridge

collapsed. The hikers were not a roped party, and the victim fell about 15 m into a crevasse. The emergency dispatch center was informed immediately. At 1255, a helicopter winched a mountain rescuer onto the glacier. With the help of some bystanders, the rescuer abseiled (rappelled) into the 0.5 m wide crevasse, where he found the patient mostly buried under snow and ice. The patient was still moving. After 15 min of digging, the mountain rescuer had freed the patient's head. His mouth and the nose had been in an air pocket. The patient was conscious and reported pain. During the next hour, the rescuers dug the patient out of the snow and ice. His cries diminished, and finally, after 45 min, he lost consciousness. The rescue team expedited the evacuation by attaching the patient's feet to a rope (his legs were the least buried) and pulling him feet-first out of the crevasse. He had been in the crevasse for approximately 100 min.

At 1415, on the glacier's surface, the helicopter's emergency physician determined that the patient was in cardiac arrest. Cardiopulmonary resuscitation was started. The pupils were fixed and dilated. The epitympanic temperature was 19.4°C. The patient was initially ventilated with a bag-valve mask before being intubated. Except for several bruises, no other injuries were found. The patient was flown to the nearest hospital, a trauma center without ECLS capabilities, arriving at 1445.

In the emergency department, the electrocardiogram showed asystole. Rectal temperature was 25°C. The patient was transferred to the operating room, where a central venous catheter was inserted. Capillary blood gas analysis revealed pH 7.12, bicarbonate 15 $mmol \cdot L^{-1}$, and potassium 2.8 $mmol \cdot L^{-1}$. At 1530, a laparotomy was performed to start peritoneal lavage with warm normal saline. Thirty minutes later, rectal temperature had increased to 26.7°C. A sternotomy was performed to allow cardiac lavage with warm normal saline. Ventricular fibrillation developed at 1645. Lidocaine was administered intravenously, followed by internal defibrillation. At 1650, the rhythm was converted to sinus bradycardia by defibrillation, and the patient began breathing spontaneously. Cardiopulmonary resuscitation was discontinued after having been administered for approximately 165 min. At 1700, the heart rate was 100 to 130 $beats \cdot min^{-1}$, and blood pressure became measurable for the first time at 80/50 mm Hg. Blood pressure increased to 130/80 over the next 2 min. When the rectal temperature reached 29°C, at 1700, the pupils became less dilated and were reactive to light.

Cardiac lavage was discontinued, but peritoneal lavage was continued until 1830. To hasten rewarming, hemodialysis was started at 1700 with the catheter in the right internal jugular vein. At 1800, the rectal temperature reached 33°C. At 1900, it was 35.2°C, and hemodialysis was discontinued.

The patient was admitted to the intensive care unit. A computed tomography (CT) scan of the head showed no cerebral edema. On Day 2, weaning from ventilation was started, and 2 d later the patient opened his eyes. He started moving his extremities the next day. On Day 6, he was extubated. The patient developed symmetric myoclonus in all 4 extremities, successfully treated with intravenous clonazepam. On Day 17, he developed acute necrotizing pancreatitis with sepsis and respiratory failure. A laparotomy was performed. Fifty-four days after the accident, he was discharged from the intensive care unit. Rehabilitation lasted 3 mo. He made a full neurologic recovery.

CASE 2

On a summer day in 2003, a 40-y-old man was hiking alone on a glacier in the Grossglockner area in Austria when he fell feet-first into a crevasse. His feet were so tightly wedged in the crevasse that he was unable to free them. He was immersed to his shoulders in icy water. He was able to call for help using his mobile phone. The patient was stabilized by the rescue team with ropes around his body. He was pulled out of the crevasse in prone position 2 h after his fall (Figures 1 and 2). The epitympanic temperature was 29°C. The patient was flown with a long line to the site where the emergency physician was waiting. During this short flight, he lost consciousness. The Glasgow Coma Scale score was 7. He was sedated and intubated by the emergency physician on site. A helicopter flew the patient to the hospital in Lienz, Austria.

On admission to a trauma center without ECLS capabilities, the rectal temperature was 26.8°C and blood pressure was 120/80 mm Hg. He had large excoriations on both shoulders and arms from the fall and his attempts to extricate himself from the crevasse. Forced air warming was started with a Bair Hugger (3M, Maplewood, MN, USA). The patient went into ventricular fibrillation during transport to the CT scanner. Cardiopulmonary resuscitation was started. Three defibrillation attempts were unsuccessful. The patient was immediately transferred to the operating room, where a sternotomy and laparotomy were performed. Open cardiac massage and rewarming of the heart and peritoneal cavity with warm normal saline were started. After 2 h, the core temperature was 34°C. At that time, external defibrillation restored sinus rhythm. High doses of catecholamines were needed to maintain adequate blood pressure. Because of intraoperative blood loss and coagulopathy, packed red blood cells and fresh frozen plasma were administered. The post-resuscitation phase was complicated by acute respiratory distress syndrome and acute kidney failure. Intermittent hemofiltration was required until Day 5, followed by hemodialysis. Sedation was

Figure 1. Crevasse rescue of the patient (case 2) in the Grossglockner area, Austria.

discontinued on Day 8, but the patient did not regain consciousness. A CT scan showed signs of possible diffuse hypoxic brain damage. On Day 9, the patient was transferred to a hospital near his hometown. He was still intubated, on assisted spontaneous ventilation, and on renal replacement therapy. The next year, he visited one of the coauthors, personnel at the helicopter emergency medical service base and in the hospital, who had cared for him. He had resumed his job and fathered a healthy child. During the next 4 summers, he visited the area and hiked the local mountains.

Discussion

Although these cases are old, several important lessons can be learned. First, successful non-ECLS rewarming after hypothermic cardiac arrest with good neurologic outcome is possible. This has been reported previously.[7] Non-ECLS rewarming is not always successful.[8]

The measured temperature depends on the site of measurement. In the first patient, an epitympanic temperature of 19.4°C was measured on scene. Forty minutes later, in the hospital, a rectal probe was inserted and a temperature of 25°C was measured. This discrepancy may have been caused by any of several factors.[9] Epitympanic temperatures may be falsely low if the external auditory canal is filled with snow or cold water, and insulation of the measurement site is important to avoid a low measurement.[9,10] Epitympanic measurement is not reliable in cardiac arrest.[2,3] Esophageal temperature measurement closely approximates core temperature in patients with a secured upper airway if the probe is placed in the lower third of the esophagus.[3] Rectal temperatures

Figure 2. Patient is rescued out of the crevasse in a vertical and prone position with ropes wrapped around his body.

may be falsely high because feces cool more slowly than the surrounding rectum,[11] and rectal temperatures may be falsely low during rewarming.[9]

The ideal methods for non-ECLS rewarming of hypothermic cardiac arrest patients have not yet been determined. Several non-ECLS rewarming techniques are available, with various degrees of invasiveness and differing rates of warming. Each technique has advantages and disadvantages, and a combination of techniques may increase the speed of rewarming. Hypothermic patients who are hemodynamically stable should be treated with minimally invasive rewarming, including heated intravenous fluids and forced air warming.[1-4] It is unclear which rewarming methods are most effective for patients in hypothermic cardiac arrest. ECLS rewarming is likely to be more effective than non-ECLS rewarming.[1-4] Survival rates of 23 to 100% have been reported for ECLS rewarming of patients in hypothermic cardiac arrest.[12]

Pooled outcome data for non-ECLS rewarming are not available. Non-ECLS rewarming is the only solution in circumstances in which transport to an ECLS center would be prolonged, unnecessarily hazardous, or impossible.

We do not believe any guidelines were available for accidental hypothermia in 1990, when the first patient experienced a witnessed cardiac arrest on the glacier. He was transported to the closest hospital, without ECLS capabilities, where the team performed a sternotomy to rewarm the heart directly. This was successful. The first article on successful ECLS rewarming after hypothermic cardiac arrest was published in 1997.[13] In 2003, our second patient experienced witnessed hypothermic cardiac arrest in the same trauma center as the first patient. The medical team still remembered the treatment and favorable outcome of the first patient with hypothermic cardiac arrest 13 y earlier. The same non-ECLS rewarming method was chosen to rescue the second patient. Transfer to an ECLS center after in-hospital hypothermic cardiac arrest was not an option because no guidelines had been published. In addition, it was uncertain that transfer to an ECLS center, which would have taken at least 4 h, would have resulted in a better outcome.[1-4,14]

Because cardiac arrest caused by accidental hypothermia is uncommon, prospective studies are not feasible. The International Hypothermia Registry (www.hypothermia-registry.org) has been established. To obtain a large number of patients, pooling data on the treatment and outcome of non-ECLS rewarming in hypothermic cardiac arrest patients might be helpful to gather information regarding rewarming techniques, including rates of rewarming, complications, and outcomes.

LIMITATIONS

Since the time of the 2 case reports, management of hypothermic cardiac arrest has evolved. Guidelines on the management of patients with accidental hypothermia have been published only since the 2010 European Resuscitation Council guidelines on special circumstances, followed by several others.[1-4,14,15] ECLS rewarming is now the method of choice in hypothermic cardiac arrest patients, although ECLS may not always be available. Laparotomy and sternotomy are very invasive and are now rarely performed in central Europe. Because these are old cases, there are gaps in the medical records. One author treated both patients and filled in the gaps to the best of his recollection.

Conclusions

Non-ECLS rewarming of patients in hypothermic cardiac arrest is feasible and can result in good outcomes when ECLS is not available. It is not known which non-ECLS rewarming methods are optimal. The choice of non-ECLS rewarming methods depends on local capabilities, and non-ECLS rewarming should be adapted accordingly. To obtain more robust evidence, it seems reasonable to pool data on the treatment and outcome of non-ECLS rewarming in hypothermic cardiac arrest patients.

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CASE REPORT

Regional Anesthesia for Symptomatic Treatment of Stingray Envenomation

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Stingray envenomation is common in coastal regions around the world and may result in intense pain that can be challenging to manage. Described therapies involve hot water immersion and potentially other options such as opioid and nonopioid analgesics, removal of the foreign body, wound debridement, antibiotics for secondary infection, and tetanus toxoid. However, for some patients, this may not be enough. Peripheral nerve blockade is a frequently used perioperative analgesic technique, but it has rarely been described in the management of stingray envenomation. Here, we report a case of stingray envenomation in an otherwise healthy 36-y-old male with pain refractory to traditional therapies. After admission for pain control, the patient received an ultrasound-guided sciatic popliteal nerve block. Upon completion of the peripheral nerve block, the patient reported rapid and complete resolution of the intense pain, which did not return thereafter.

Keywords: peripheral nerve block, marine injury, animal injury

Introduction

Stingray envenomation is common along coastal regions of the United States, with approximately 2000 cases reported annually.[1,2] The barb from the stingray punctures the patient's skin, releasing a venom containing serotonin, 5'-nucleotidase, and phosphodiesterase, which can ultimately lead to intense pain and peripheral vasoconstriction.[3] Untreated, the envenomation and barb migration can lead to infection, soft tissue necrosis, organ perforation, gangrene, cardiac dysrhythmias, and even death.[3-5] Significant long-term complications from envenomation have also been reported, including chronic pain and complex regional pain syndrome.[6-8] Initial treatment involves hot water immersion (HWI) at approximately 40 to 45°C, although a more recent study suggests 43 to 45°C is optimal,[9] for up to 90 min to denature the heat-labile venom proteins.[1,4,10] Other options include pharmacologic pain management, antibiotics for secondary infection, tetanus toxoid, and foreign body removal/debridement.[1,4,10] The pain associated with the sting can be debilitating, challenging to manage, and potentially resistant to common analgesic medications. Here, we report a case of refractory pain due to envenomation by a southern stingray (*Hypanus americanus*), a species native to the east coast of the United States. The patient was treated with a popliteal sciatic nerve block. After the ultrasound-guided popliteal sciatic nerve block was performed, the patient achieved immediate and long-lasting pain relief.

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Case

An otherwise healthy, opioid naïve, 36-y-old male presented to the emergency department with severe pain in his left ankle after being stung by a stingray while surfing in the Atlantic Ocean off the coast of Virginia Beach, Virginia. The patient reported immediately feeling a sharp and shooting pain over his left lateral malleolus. Shortly thereafter he was brought to the emergency department and over the next 3 to 4 h, his lower leg became erythematous, edematous, and exquisitely tender to palpation. He was initially examined for signs of retained foreign body and received HWI (>40°C, exact temperature unclear) therapy for a little over 80 min

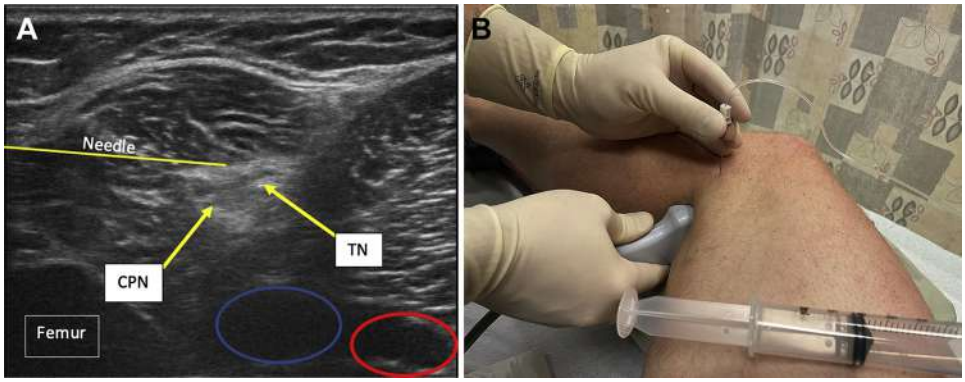

Figure 1. (A) The image on the left shows the ultrasound anatomy of the left sciatic nerve just above the popliteal fossa at the beginning of the sciatic nerve bifurcation into the tibial nerve and common peroneal nerve. Just deep and medial to the tibial nerve and common peroneal nerve are the popliteal vein and artery, circled in blue and red, respectively. (B) The patient is shown in the right lateral decubitus position. An ultrasound-guided sideline popliteal sciatic nerve block is performed with the needle entering from a lateral approach in plane with the ultrasound probe, with local anesthetic deposited around the nerve.

without improvement. Because the patient continued to report little improvement in his pain after 80 min of HWI, he was given 4 mg of intravenous morphine, 1000 mg of intravenous acetaminophen, and 15 mg of intravenous ketorolac. X-rays performed in the emergency department did not reveal any signs of fracture, dislocation, or foreign body (possibly not visible given the small size and cartilaginous nature of the spine).

At approximately 8 h after initial injury, the patient reported persistent intense pain despite prior interventions. At this point, he was admitted for pain control and the acute pain service was consulted for assistance with management. After the orthopedics services reported the injury as being low risk for the development or masking of compartment syndrome, the acute pain service team elected to perform a peripheral nerve block. Ultrasound-guided sciatic nerve block was performed via the popliteal fossa approach. The sciatic nerve was easily identified sonographically in the popliteal fossa and followed to the bifurcation into the common peroneal nerve and tibial nerves (Figure 1A). Under ultrasound guidance, 20 mL of 0.5% ropivacaine was injected in 5 mL aliquots around the nerve directly proximal to the sciatic nerve bifurcation. Immediate relief was achieved after the procedure and continued for the duration of the hospital stay. The patient was started on intravenous cefazolin and doxycycline for secondary infection prophylaxis and was monitored on the internal medicine ward overnight. After 24 h, the patient met discharge criteria with adequate pain control and without signs or symptoms of infection. He was able to ambulate without assistance after resolution of the peripheral nerve block approximately 20 h into admission.

Discussion

The pain from stingray envenomation is potentially debilitating. Although first-line strategies are usually effective,[1] regional anesthesia offers an additional tool for pain control. In our case, the use of HWI in addition to pharmacologic adjuncts was not sufficient to achieve adequate pain control. Although the HWI temperature may have been inadequate to neutralize the venom in this case, peripheral nerve blockade might still be considered in other patients for whom HWI at temperatures of 43 to 45°C is ineffective.[9] That said, regional anesthesia techniques are rarely described for stingray envenomation. Local anesthetic infiltration into the wound site has been described, and 1 other case involving the use of a peripheral nerve blockade was reported, although an upper extremity peripheral nerve block was used.[11,12] Regional anesthesia has significantly changed since the initial description in the 1880s, with improvement in technique and performance due to ultrasound and modern local anesthetics.[13,14] In patients with challenging pain control, such as the combat wounded during the recent wars in Iraq and Afghanistan, regional anesthesia was critically important and effective.[15,16] Regional anesthesia has been described in austere environments outside of the operating room,[17] as well as in the emergency department. For qualified providers in the emergency department, regional anesthesia has been used for patients with hip/femur fracture, shoulder reduction, extremity trauma, and rib fractures.[1,18,19] Furthermore, the ongoing opioid crisis has demonstrated the need for increased utilization of regional techniques for opioid minimization.[20,21]

After stingray envenomation, pain typically reaches its peak within 30 to 90 min of injury and can last 48 h if

untreated.[22] Long-acting local anesthetics such as bupivacaine or ropivacaine could offer analgesic benefit during this time period. Because of the ease of performance of peripheral nerve blocks and the low side effect profile, regional anesthesia should be considered for stingray envenomation refractory to traditional first-line treatment. In addition, peripheral nerve blockade often leads to increased blood flow due to disruption of sympathetic innervation to the local vasculature.[23-25] Theoretically, increased regional perfusion could lead to decreased clinical duration of pain through redistribution of stingray toxins away from the local envenomation site.

Conclusions

We report the use of a popliteal fossa sciatic nerve block for symptomatic treatment of stingray envenomation in a patient with severe refractory pain. The literature supports the use of regional anesthesia for this purpose, however, there is scant evidence for its use. The relative ease of placement and safety of peripheral nerve blocks make their use a potentially valuable tool for such patients.

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Disclosures: The views expressed in this article are those of the authors and do not necessarily reflect the official policy or position of the Department of the Navy, Department of Defense, or the United States Government. LT DeYoung is a military service member. This work was prepared as part of his official duties. Title 17 U.S.C. 105 provides that "Copyright protection under this title is not available for any work of the United States Government." Title 17 U.S.C. 101 defines a United States Government work as a work prepared by a military service member or employee of the United States Government as part of that person's official duties.

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CASE REPORT

Life-Threatening Panfacial Wild Dog Bites in a Child

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Infants and toddlers can sustain grievous craniofacial injuries after dog bites, some of which may be life-threatening. An 18-mo-old male child presented to our emergency department with complex panfacial wounds after being bitten by an unvaccinated wild dog 6 h earlier. Primary management, hemostasis, and rabies postexposure prophylaxis were performed near his home. Initially, he was resuscitated from severe hemorrhagic shock and anemia in a pediatric intensive care unit. After stabilization, early primary repair of all facial injuries was performed. Surgical exploration revealed multiple full-thickness avulsions, lacerations, nasal bone fractures, facial muscle injuries, and right ear necrosis. Gentle tissue-handling and meticulous reconstruction satisfactorily restored his facial soft-tissue contours about 64 h after the bite injury. Postoperatively, recovery was uneventful except for localized soft-tissue infection caused by multidrug-resistant *Pseudomonas*, which resolved with appropriate antibiotics. Nine months later, his face and ear appeared almost symmetrical with well-settled scars. Psychological recovery progressed smoothly, excepting few incidences of panic attacks that were triggered by loud noises. Here, we observed that early primary reconstruction of severely mauled soft tissues after wild dog bites, performed within 72 h with adequate precautions, safely produced satisfying long-term outcomes.

Keywords: bites and stings, dog bite, facial injuries, facial injury treatment, rabies prevention and control

Introduction

With numerous unvaccinated wild, stray, and mixed-breed dogs roaming in the city streets and village neighborhoods of developing countries, a sizeable world population remains at risk of bites by unvaccinated dogs.[1] Age-wise, children constitute the largest segment of victims. Among children, 0- to 1-y-olds are the minority at 5%, and 6- to 12-y-olds constitute the majority (51%).[2] Although infants and toddlers are affected the least numerically, they are maximally vulnerable to craniofacial injuries owing to their short stature, disproportionately large head, tendency to crawl on the ground, extreme curiosity, subdued danger awareness, and inability to defend themselves.[3]

Wound closure after bites by wild, unvaccinated, or unknown dogs remains controversial. Immediate primary suturing of such bites is not recommended.[4] Many surgeons prefer not suturing unvaccinated-dog bite injuries at all, to prevent additional trauma that may increase the risk of rabies. Some surgeons perform delayed primary repair after 5 d, while irrigating the wounds daily to decrease viral load. Another attractive alternative is to loosely appose soft tissues with gentle tissue-handling 48 to 72 h afterward, under cover of antirabies vaccine and rabies immunoglobulin, to control viral spread. This last approach was performed in this case report, and long-term results were observed.

The child's parents provided written, informed consent for the case to be reported in the literature, including all of the content shared.

Case Report

An 18-mo-old male child presented to our emergency department unconscious and severely pale, with several avulsions and lacerations throughout his face. About 6 h earlier, he was reportedly attacked by an unknown wild dog just outside his home. His parents were out of town on an emergency and his babysitter was distracted on a phone call when he crawled outside his home unnoticed.

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Figure 1. An 18-mo-old child after being bitten by a wild dog. (A) Frontal view showing facial avulsions, lacerations, and nasal fracture. (B) Right oblique view showing mauled lips, ear, and face with clots over frontalis. (C) Nasal trauma showing fractured nasal bones. (D) Torn and ripped portions of the right ear.

Profusely bleeding and in severe pain, the injured child was rushed to the nearest health facility. Later, this same wild dog went on to attack 2 more children in the same village, which bordered a forest. The villagers stoned this dog to death 2 d after this incident. Forensic examination performed thereafter did not reveal Negri bodies in the brain, which are indicative of rabies infection. The recommended surveillance of the dog in a registered facility for 10 d after this incident was not possible, however.

A general physician managed the child initially, controlled the bleeding, and administered antibiotics, analgesics, and sedatives. Over the wounds, rigorous irrigation with copious amounts of normal saline for 15 min, disinfection with povidone-iodine while protecting the eyes, and light coverage with sterile dressings (without any wound suturing) were performed. Passive immunization with human rabies immunoglobulin at a dose of 20 IU·kg^{-1} body weight was administered deep inside of and all around the facial wounds. The recommended dose was diluted to 3 times the original volume with sterile normal saline because the extensive nature of the wounds necessitated administration of significant volumes of the immunoglobulin. Active immunization was performed with cell-cultured antirabies vaccine. After primary treatment, the child was referred to our hospital and reached us after a 4-h journey in a private vehicle.

Physical examination at our hospital emergency department revealed multiple avulsions and lacerations all over the child's face, but more so on the right (Figure 1A). Underlying facial muscles appeared mauled, covered with thick clots, and associated with nasal bone fractures (Figure 1B and C). The right ear was severely avulsed, with the helix torn off from the rest of the ear, hanging on a small base (Figure 1D). The child was pale, drowsy, tachycardic, and tachypneic at presentation. Even after receiving adequate oxygen through a face mask, his oxygen saturation levels gradually fell below normal. Arterial blood gas analysis on admission revealed low hemoglobin of 5.2 g·dL^{-1} (normal 12–17 g·dL^{-1}) and high lactate of 13 mmol·L^{-1} (normal 2 mmol·L^{-1}), signifying severe anemia and hemorrhagic shock. The child was intubated to prevent desaturation and was successfully resuscitated at our pediatric intensive care unit. All of his wounds were irrigated at the bedside once every day, and prophylactic broad-spectrum antibiotic combinations of intravenous amoxycillin-clavulanate and amikacin were started empirically. Bedside facial radiograms did not reveal any gross bony injuries, excepting the nasal bone fracture. The patient's parents were counselled extensively about different treatment options. After diligent deliberation, considering risks, benefits, and costs, they chose an early primary repair. Accordingly, after hemodynamic stabilization, surgical exploration was performed with the patient under general anesthesia about 64 h after the bite injury.

Multiple full-thickness avulsions with extensive tissue loss and lacerations throughout the face, with fractured nasal bones, were found on intraoperative examination (Figure 2A). Facial muscles were ripped off the underlying bones in several areas, creating interconnected pockets (Figure 2B and C). Avulsion of the right ear ripped the helix off the concha, necrosing the antihelix and crura in the middle, with demarcation forming between healthy and necrotic tissues (Figure 2D). The injury was classified as a World Health Organization Category III animal bite.[5] According to Lackmann's classification for pediatric dog bites, it was Type IV B.[6]

All wounds were rigorously irrigated under high pressure for approximately 30 min with 0.9% saline-filled

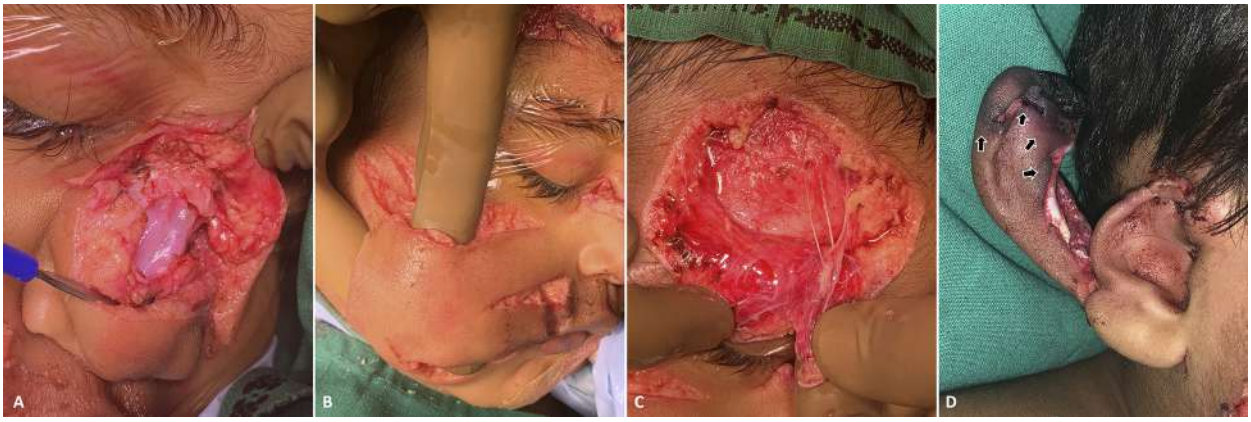

Figure 2. Intraoperative findings. (A) Nasal bone fracture fragments after alignment. (B) Interconnected deep soft-tissue pockets observed over the right midface. (C) Frontalis muscle seen to be ripped off the bone, but the underlying nerves are preserved. (D) Zones of demarcation (black arrows) gradually forming between necrosing and healthy tissues over the right ear.

50-mL syringes fitted with an 18G needle. After protecting the patient's eyes, substantial amounts of 10% povidone-iodine were smeared throughout all wounds for 5 min and flushed with normal saline before repair. Fractured nasal bones were carefully aligned. Soft tissues were meticulously apposed following the principles of wound closure with triple-plane dissection over the forehead and deep-plane augmented facial flaps over the mid and lower face.[7,8] To preserve facial symmetry and harmony, reconstruction of the face progressed from central to peripheral with several tension-free synthetic sutures that were loose enough to allow free drainage from deep tissues, while avoiding excessive soft-tissue dissection (Figure 3A and B). Necrosed areas over the right ear were debrided, and remaining ear flaps were satisfactorily juxtaposed to remold the mutilated ear (Figure 3C).

After discharge, about 1 wk postoperatively, scanty purulent discharge was noticed over a corner of 1 of the midface wounds, with redness and swelling of 2 × 2 cm adjacent areas. Wound swab was culture-positive for multidrug-resistant *Pseudomonas aeruginosa*, resistant to the antibiotics started earlier. After the results of a sensitivity profile, intravenous meropenem was combined with tigecycline for the next few days to control this infection. Antirabies vaccination was completed as scheduled on days 0, 3, 7, and 28.[5] Initially, the involved areas were considerably edematous, but this subsided appreciably within the next 2 wk. Swelling and redness were still visible at the infected area, with about 1 mm of marginal skin necrosis (Figure 4A). Movements over the affected areas were restricted during the first postoperative month and improved gradually afterward. Scars, including the

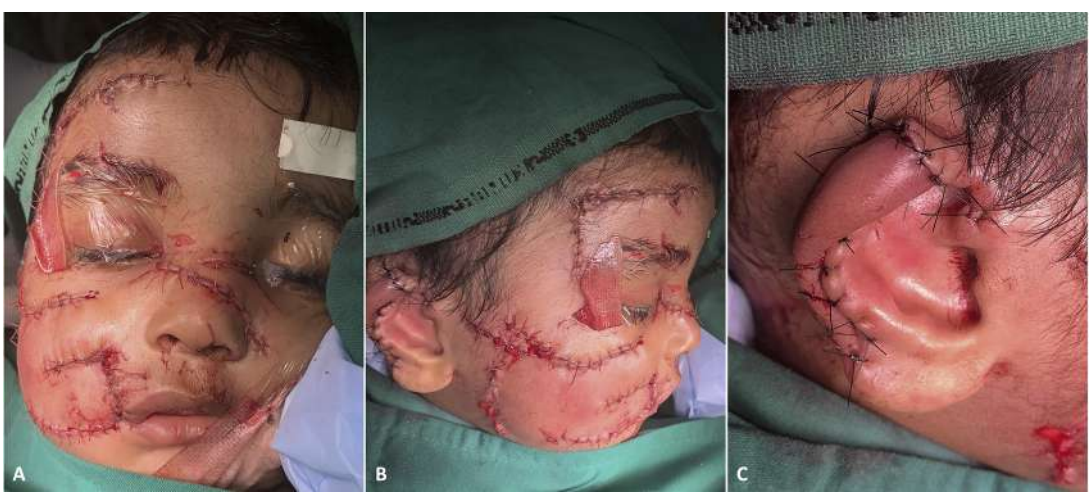

Figure 3. Immediate postoperative results after tension-free apposition of soft tissues. (A) Frontal view. (B) Right lateral view. (C) Right ear.

Figure 4. Postoperative results after 2 wk. (A) Frontal view showing residual redness, swelling, and marginal wound necrosis over right lower midface (this area was infected secondarily). (B) Right lateral view of the healing wounds and scars. (C) Remolded right ear.

cross-hatches, were reddish and overtly visible during the first 2 mo (Figure 4A and B). The remolded right ear appeared well shaped (Figure 4C). The child was withdrawn, afraid, and extremely irritable during the early months, requiring psychological counseling and constant parental care. Otherwise, his recovery was mostly uncomplicated and uneventful.

Nine months postoperatively, his facial appearance and expressions were well balanced and pleasing. Complete reanimation of all avulsed facial muscles resulted in coordinated and harmonious facial movements. Scars settled well and were barely noticeable from afar. Some amount of scar stretching was noticeable in some areas, however (Figure 5A and B). His right ear, although a bit smaller and floppier, appeared nearly symmetrical (Figure 5C). Psychologically, for the first 6 mo, he experienced severe bouts of panic attacks from loud noises, especially the noises of lightning during storms. He improved thereafter, and after 9 mo, while still avoiding all dogs, he would sometimes be fearful after hearing the noise of leaves rustling during high winds. Otherwise, he recovered well under the supervision of a psychologist and a pediatrician. Both of the child's parents expressed deep satisfaction with the entire treatment process, with the final outcome and recovery far exceeding their expectations.

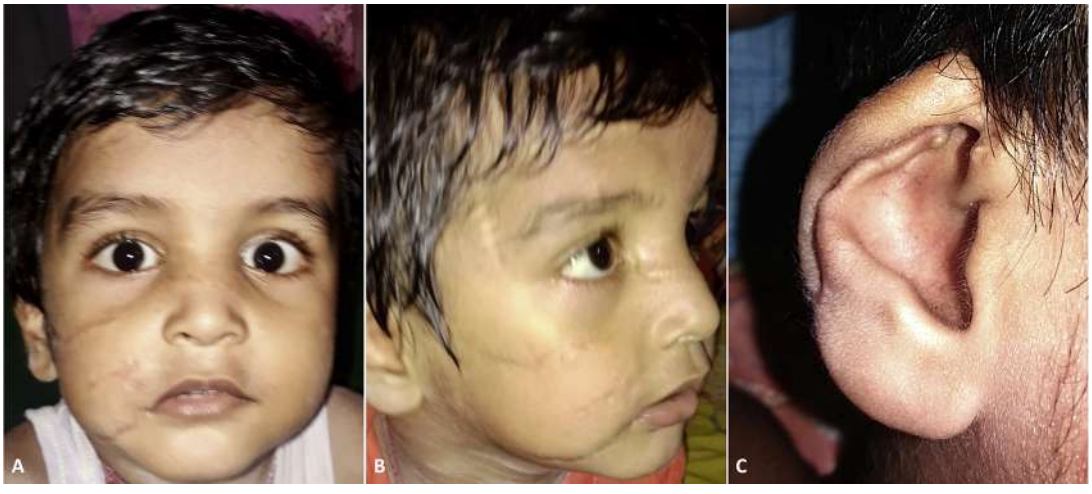

Figure 5. Postoperative results after 9 mo show a near-normal facial appearance and function with scars bettering over time. (A) Frontal view. (B) Right lateral view. (C) Right ear.

Discussion

Dog bites are preventable injuries and yet pose a significant public health problem globally. In children, facial dog bites usually occur after prolonged eye contact with the dog, while bending over the dog, after overt familiarity, or when remaining indoors together for a long time without going out for walks.[2] Close supervision of all child–dog interactions, not leaving any child unattended around any dog, proper vaccination of all dogs, and prompt reporting of all dog bites help to protect children. Dog bites are more common in children than in adults, with 76% occurring in the head/neck region. Cheeks, lips, and nose are commonly affected as the "central target region" in children younger than 18 mo.[9] In comparison, older children usually sustain dog bites to their extremities because they are taller.

Soft-tissue dog bite injuries are classified into 3 major types: laceration, avulsion, and puncture. Typically, a combination of all 3 are present. A biting dog's jaw usually produces a powerful force of 1380 to 3100 kPa (200–450 pounds per square inch), which is strong enough to perforate metal sheets. Frequently, the ripping-and-tearing motion associated with a dog bite devitalizes the wounded soft tissues and damages underlying vessels and nerves.[10] During admission, alerting patients about the chances of wound necrosis, infections, scars, foreign bodies, and rabies helps in overall management.[9] Facial bone fractures after dog bites in children are rare, occurring in about 1 to 2%. The compressive force of dog bites is sufficient to fracture thin facial bones in children, especially over the orbit, nose, and skull. Facial bones in growing children are spongy, have high regenerative potential, and possess osteogenic capabilities. When correctly aligned, these properties frequently result in proper fracture healing.[11]

Soft-tissue infections after dog bites occur in around 10%, with *Pasteurella canis* being the most commonly (50%) isolated organism. Other bacteria such as *Fusobacterium*, *Bacteroides*, *Prevotella*, and *Pseudomonas* may also be present.[12] *Capnocytophaga canimorus* is an uncommon but fearsome slowly growing organism with a prolonged incubation period of 1 to 7 d that causes septic shock and purpura fulminans in susceptible patients, with 25% mortality.[13] Short courses of preemptive antimicrobials are recommended for moderate-to-severe bite injuries over the face and upper limbs. Longer courses are preferred for severe injuries with bony fractures and joint penetration.[4,6] Amoxicillin-clavulanate is effective against most pathogens, including *Pasteurella* and *Capnocytophaga*, and amikacin is effective against gram-negative bacteria including *Pseudomonas*.[4] Thus, combining these 2 antibiotics empirically provides good protection.

From an aesthetic perspective, open wounds allowed to heal secondarily often form unacceptable disfiguring lifelong scars. When compared to nonsuturing, wounds closed within the first 24 h achieve better aesthesis and function with similar secondary infection rates. Wound management started early also results in decreased infection rates and improved aesthetic outcomes, regardless of suturing status.[14] Scalp and forehead are relatively tough tissues, and a limited triple-plane dissection helps in meticulous repair of injuries over these areas.[7] Over the cheeks and lower face, a deep-plane cervicofacial flap-like approach is helpful.[15] Mobilizing the mauled and avulsed segments of facial soft tissues as a large and thick augmented facial flap ensures adequate vascularity to the wound edges while avoiding undue dissection of the adjacent healthy areas.[8] Because facial reconstruction of the central target area is more complicated, the lip vermilion and commissure, nose margins, and nasal columella are reconstructed first. Substantial tissue loss after avulsions may require reconstructive flaps or grafts. If initial surgery is delayed for any reason, delayed primary closure up to 5 d later, with daily wound irrigation, remains a good option.[16]

Conclusions

Major dog bite injuries persist as a global public health problem. Psychological issues are equally significant in affected patients. Here, an early primary meticulous reconstruction of the severely mauled panfacial soft tissues, after taking comprehensive precautions, produced pleasing long-term surgical results in an 18-mo-old child. Vigilance during the postoperative period and a prolonged medical and psychological supervision helped in good patient recovery, yielding excellent patient-reported outcomes.

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CASE REPORT

Traumatic Neck Injury: An Accidental Impalement by a Needlefish

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Encounters between marine animals and humans can result in critical injury and fatal complications. We highlight a 20-y-old male who sustained a penetrating injury to the neck as a result of impalement by needlefish (*Tylosurus* sp) while snorkeling. He sustained a penetrating injury in the posterior triangle of the neck. On presentation, he was stabilized and received empirical antibiotics, analgesia, and antitetanus toxoid injection before being transferred to a tertiary center. On presentation to the tertiary hospital, the patient was hemodynamically stable with no clinical evidence of injury to surrounding neck structures, and this was confirmed using computed tomography. The patient underwent local wound exploration and retrieval of the needlefish beak under general anesthesia. The wound was left open to heal by secondary intention. The patient was discharged with oral antibiotics and went on to make a complete recovery.

Keywords: Belonidae, marine biology, stab wounds, tropical medicine

Introduction

Injury caused by marine animals is usually unexpected. Despite the need for fishing or social activities among the local population and travelers, marine ventures can be life-threatening, especially in tropical and subtropical countries. Activities such as windsurfing, kite surfing, swimming, and diving can result in injury owing to contact with marine animals.[1,2] Stingrays and stonefish can cause direct trauma and envenomation, but there are also infrequent cases of injury by needlefish and swordfish species.[3] Although infrequent, an encounter with these fish can result in critical injury and even fatal complications.[4,5] We present a case of injury to the posterior triangle of the neck after needlefish impalement and discuss our management approach.

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Case Report

A 20-y-old Maldivian male presented to a tertiary care center 4 h after being impaled by a needlefish while snorkeling in the evening. The patient had sustained a penetrating injury to the right side of the neck (Figure 1) with local swelling and ecchymosis. On presentation, he was hemodynamically stable with a pulse of 75 beats·min^{-1} and blood pressure of 120/70 mm Hg. There was no evidence of vascular injury, such as pulsatile bleeding, arterial thrill, or rapidly expanding hematoma. After initial assessment and hemodynamic stabilization by the local clinic, the patient was given ceftriaxone and metronidazole empirically, along with analgesia and antitetanus toxoid (ATT) injection. He was then referred to the tertiary center for definitive management. The results of laboratory investigations, especially renal

Figure 1. After impalement, the body of the fish separated from the beak, which was retained in the wound. The wound entry was noted at the posterior triangle and zone II of the neck.

profile and liver function tests, were unremarkable. Chest radiography (Figure 2) revealed an elongated hyperdense foreign body, which was confirmed using computed tomography (CT) scan. The CT imaging revealed a 3-cm foreign body (Figure 3A) that avoided vital neck structures (Figure 3B).

Upon confirmation that no major structures were involved, the patient was taken to the operation theater for wound exploration under general anesthesia.

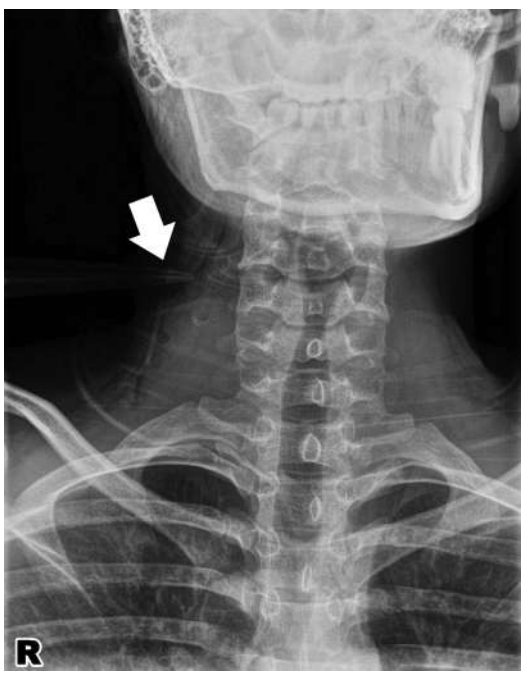

Figure 2. Chest radiograph revealed a sharp, elongated, radiopaque structure (white arrow) at the right lateral neck.

Intraoperative findings (Figure 4A) revealed a 3-cm-long oblique penetrating wound tract on the lateral aspect of the right side of the neck. The intact needlefish jaw was extracted (Figure 4B), and the wound was copiously irrigated with saline. The surgery was completed without complication, and the wound was left open to allow healing by secondary intention given the possibility of retained foreign body and infection. The recovery period was uneventful, as expected, and the patient was discharged home the next day with oral antibiotics (cefuroxime and metronidazole) and analgesia. He was prescribed Algicel Ag dressing changes every other day.

Discussion

Needlefish are silver in color with an elongated body and are tubular in shape, ranging from small to medium size; most needlefish are usually between 3 and 95 cm.[6] Needlefish have a characteristic narrow, pointed jaw with numerous sharp teeth. They belong to the family Belonidae and order Beloniformes.[6] Usually, needlefish swim near the surface; however, if they are startled, frightened, or attracted to bright light, especially at night, they may leap from the water or skip on the surface. They are capable of making short jumps out of the water at up to 65 $km \cdot h^{-1}$ (40 mph).[6] With high velocity, their sharp beaks can spear swimmers, divers, or fishermen, inflicting deep puncture wounds.[4,5] In our case, the type of fish was identified based on visual confirmation by the patient and local bystanders who brought him to the hospital. We did not send for DNA testing because it was unnecessary at the time and a biologist was not available for visual identification.

Injury severity depends on the organs involved. Given the nature of penetrating wounds, the entry point may appear deceptive compared to the underlying injuries it has caused. Injuries from needlefish to the limb, abdomen, and even face have been commonly reported within the literature.[1,2,4,5,7,8] However, there are infrequently reported cases involving the neck.[9,10] To date, there are only 2 other reported cases involving the neck; however, this type of injury has the potential to be fatal. This case is important because it highlights the management of a marine penetrating injury to the neck and key considerations to be made (Table 1).

Management of all penetrating injuries should begin with advanced trauma life support.[11] It may be challenging in a remote area and may require transfer to a higher center of care capable of advanced imaging and surgical intervention, as happened in our case.[12] Penetrating wounds to the neck may injure the vital structures such as the vessels, nerves, trachea, and esophagus, depending on the zone of injury (Figure 5).[13,14] In this

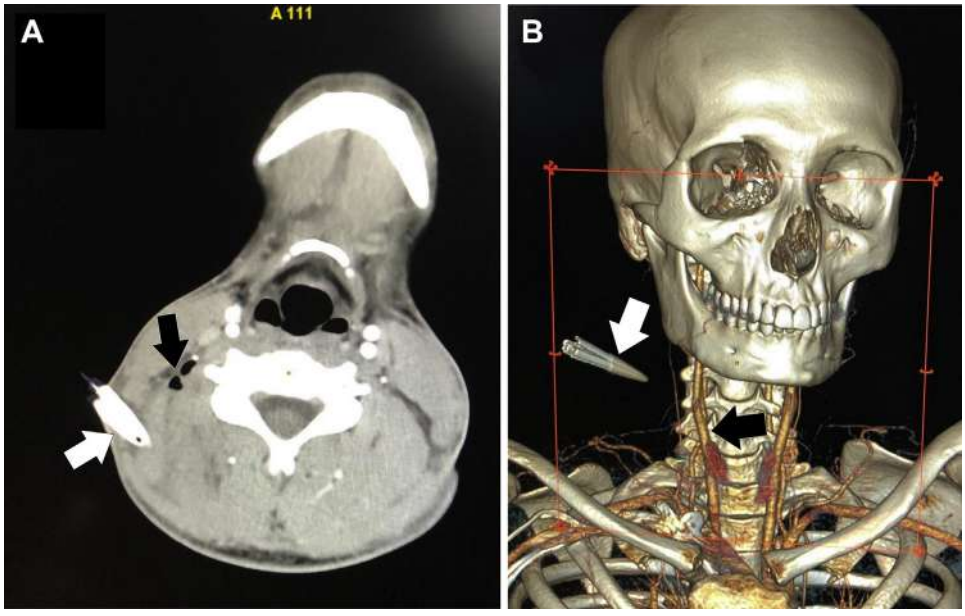

Figure 3. (A) An axial computed tomography angiogram image revealing a hypodense structure (white arrow) measuring 3 × 1 cm, representing the retained beak of the needlefish penetrating beyond the deep cervical fascia with the presence of air (black arrow). (B) A 3D reconstruction revealing the elongated foreign body (white arrow) 6 cm from the carotid vessels (black arrow).

case, the patient sustained an injury in zone II, which lies between the angle of the mandible and the border of the cricoid cartilage, in which injury to the carotid arteries and its branches, as well as the jugular veins, can be life-threatening.[13] The cartilaginous laryngeal skeleton and tracheal injury are difficult to repair but can induce rapidly developed subcutaneous emphysema, which sometimes can lead to fatal outcomes.[15] Injury to the abdomen and thorax can lead to the involvement of visceral organs and their blood supply.

Antibiotic coverage via the parenteral route should be considered empirically to avoid superimposed infection in the dirty wound after blood and swab culture and sensitivity testing. For marine injury in the summer, the 3 most common isolates were *Bacillus* sp, *Enterobacter cloacae*, and *Klebsiella pneumoniae*, which are sensitive to a combination of penicillin or ampicillin with levofloxacin for empiric antibiotic coverage via the parenteral or oral route.[15] Clinicians should consider ATT and its appropriateness in penetrating injuries/foreign body retention. It should be given if the patient has not received a tetanus shot in the last 5 y and if the wound is determined to be dirty.

A comprehensive physical examination is necessary to determine the extent of injury, and imaging can help suggest the approach, technique, and instrument that need

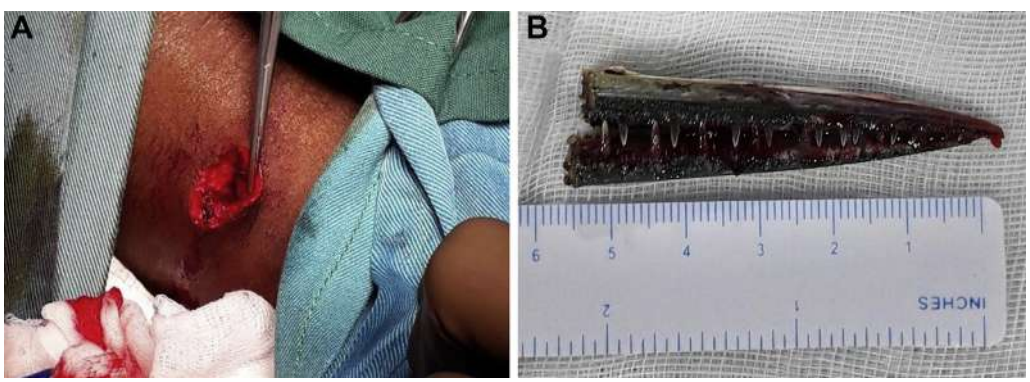

Figure 4. (A) Wound exploration and irrigation. The entry wound measured 1 × 0.5 cm and 3 cm in depth. (B) The needlefish beak was retrieved in a complete form measuring 6 cm in length.

Table 1. Traumatic neck injury caused by needlefish

Case	*Age (y)*	*Sex*	*Level of injury*	*Activity*	*Investigation*	*Type of surgery*	*Antibiotics*	*ATT*
I[9]	53	Female	Zone II	Swimming	Cervical radiograph revealed radiopaque foreign body. CT showed 3.7 cm calcified foreign body	Neck exploration, primary wound closure with drain. The procedure was done with the patient under general anesthesia	Intravenous cefazolin	Not mentioned
II[10]	20	Male	Zone III	Surfing	Radiologic examination (not mentioned) showed no perforation or bone lesion	Manual removal using straight Kelly clamps, performed with patient under local anesthesia	Oral cephalexin	Not mentioned
Current	20	Male	Zone II	Snorkeling	Cervical radiograph revealed radiopaque foreign body. CT showed foreign body at the lateral neck.	Neck exploration and closure with secondary intention. The procedure was done with the patient under general anesthesia	Parenteral ceftriaxone and metronidazole with conversion to oral cefuroxime and metronidazole upon discharge	ATT injection

ATT, antitoxoid therapy; CT, computed tomography.

to be used to retrieve the foreign body.[16] The choice of imaging modality is crucial to evaluate the injury severity, including the depth and local effects, and to look for any retained foreign body.[17] Neck radiography can sometimes be helpful to detect such findings; however, in an inconclusive study, especially in certain types of non-opacity or with small, fragmented pieces of foreign bodies, CT scan will be diagnostic. CT angiogram is mandatory if an injury to the vascular organs is suspected. A sharp foreign body is known to traumatize the vulnerable structures not only at the point and moment of entering but also during the process of withdrawal. A sharp and jagged foreign body may cause more harm during retrieval than during entrance, which is an important consideration from a surgical perspective.

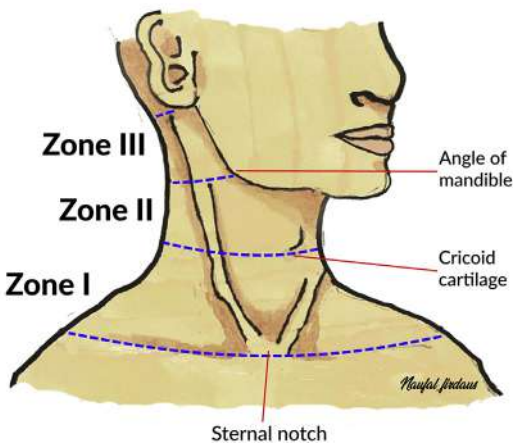

Figure 5. Illustration showing the level of penetrating injury to the neck according to 3 zones. Zone I: the area between the clavicles and the cricoid cartilage. Zone II: the area between the cricoid cartilage and the angle of the mandible. Zone III: the area between the angle of the mandible and the base of the skull.

Penetrating wounds need to be explored irrespective of zone in the presence of severe active bleeding, unresponsive hypovolemic shock, a rapidly expanding hematoma, blowing wound, and major hemoptysis.[13] A surgical examination can be done either under local or general anesthesia depending on the injury severity, based on individualized case judgment by the attending clinician. In our case, surgical exploration was performed with the patient under general anesthesia, owing to violation of the platysma. In this case, deeper vital structure injuries were anticipated compared to the intact platysma wound. EAST Guidelines of 2008 stated as Level I evidence that selective operative management versus mandatory exploration of penetrating injuries to

zone II of the neck is both justified and safe.[18] The wound should be copiously irrigated, debrided, and closed by primary or secondary intention.[13] The size and depth of the wound will influence the need for suturing.

Conclusion

Traumatic encounters with marine animals such as needlefish can result in morbidity and mortality. A comprehensive primary and a secondary survey should be used in all penetrating traumas to rapidly identify additional injuries. Patients with penetrating neck wounds who are stable may be considered for surgical exploration once the platysma is breached. Marine penetrating injuries should be left open for secondary healing with antibiotic coverage to minimize risk of infection.

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CASE REPORT

Differential Diagnosis Between Venomous (*Bothrops jararaca,* Serpentes, Viperidae) and "Nonvenomous" (*Philodryas olfersii,* Serpentes, Dipsadidae) Snakebites: Is It Always Possible?

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Bites of "nonvenomous" snakes can sometimes be mistaken for the bites of venomous snakes. As an example of this confusion, this report describes confirmed bites by *Philodryas olfersii* and *Bothrops jararaca*. In the first case, a 55-y-old man with a history of controlled hypertension was bitten on his right forearm by *P olfersii*. Physical examination revealed extensive edema, erythema, and widespread ecchymoses throughout his right upper limb. Laboratory tests indicated leukocytosis and high D-dimer levels, but normal coagulation, suggestive of a resolved recent coagulopathy. He received only supportive treatment. In the second case, a healthy 35-y-old man was bitten by *B jararaca*. Although the anatomic region of the bite and the results of physical examination were similar to those in the first case, laboratory tests showed mild coagulopathy, leukocytosis, and high D-dimer levels. The patient was treated with antivenom. In both cases, the 20-min whole blood clotting test results were normal. Patients bitten by *P olfersii* may present with local symptoms resembling *B jararaca* envenomation. Without snake identification and the detection of venom-induced consumption coagulopathy, especially in places where the 20-min whole blood clotting test is the only clotting test available, it is almost impossible to establish an accurate and safe differential diagnosis. In this context, the best alternative is to take the risk of prescribing antivenom for a possible *P olfersii* bite rather than failing to do so for a real *Bothrops* bite. Late treatment for *Bothrops* bite can result in severe complications and sequelae.

Keywords: non-front-fanged colubroids, nonvenomous snake, antivenom

Introduction

Non-front-fanged colubroid (NFFC) snakes comprise approximately two-thirds of the described species of advanced snakes.[1] However, the medical significance of the majority of the NFFC taxa is unknown,[1] and their bites can sometimes be mistaken for the bites of front-fanged venomous snakes.[2] Here we describe the cases of 2 confirmed bites, 1 by *Philodryas olfersii* (Lichtenstein, 1823) (Figure 1A), an NFFC snake, and the other by *Bothrops jararaca* (Wied, 1824) (Figure 2A), a pit viper, as examples of how this confusion can occur. Both patients were admitted to the Hospital Vital Brazil (HVB), Instituto Butantan, São Paulo, Brazil, a hospital specializing in the care of patients involved in accidents caused by venomous animals. In both cases, the snakes were brought to the hospital by the patients and identified by technicians or researchers at the Laboratório de Coleções Zoológicas. The identifications were made from voucher specimens that were housed in the herpetologic collection of Alphonse Richard Hoge, Instituto Butantan, São Paulo, Brazil.

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Details of Cases

CASE 1

A 55-y-old man with a history of controlled hypertension was bitten on the dorsal surface of the distal third of his right forearm while trying to capture an adult *P olfersii*

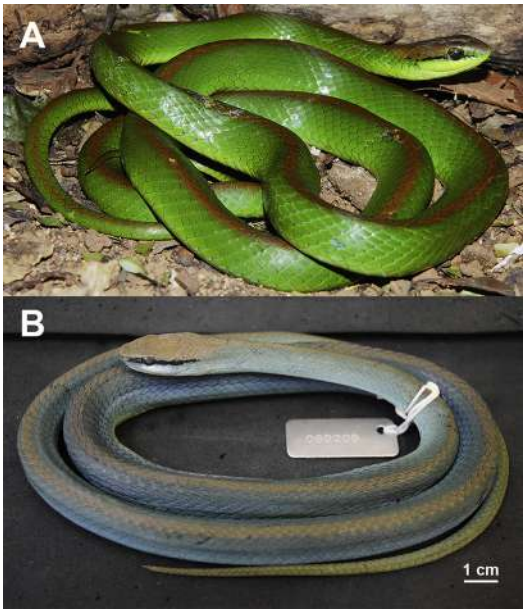

Figure 1. (A) *Philodryas olfersii* (Lichtenstein, 1823), a nonfront-fanged colubroid snake; living specimen photographed in the municipality of Jundiaí (23°11′11"S, 46°53′03"W), about 38 km from São Paulo. (B) *Philodryas olfersii* (Instituto Butantan, São Paulo, Brazil number 89209) was brought by the Case 1 patient. Adult female, total length 87.5 cm, mass 80 g, origin Cabreúva (23°18′27"S, 47°7′59"W), about 87 km from São Paulo. (Photos by Marcelo Ribeiro Duarte).

(female, total length 87.5 cm, mass 80 g) (Figure 1B). The snake was in the backyard of his house, located in a rural area in the municipality of Cabreúva (23°18′27"S, 47°7′59"W), about 87 km from the city of São Paulo. Upon arrival at the HVB, 10 h post-bite, the following data were recorded: blood pressure 155/87 mm Hg, heart rate 111 beats·min^{-1}, axillar temperature 35.8ºC, and oxygen saturation 95% on room air. The patient reported pain extending from the bite site to the underarm. Physical examination revealed the presence of semicircular bite marks and severe edema, erythema, and widespread ecchymoses throughout his right upper limb (Figure 3A–C). He denied having used a tourniquet.

Laboratory test results on admission are presented in Table 1. The fibrinogen level, prothrombin time, international normalized ratio (INR), activated partial thromboplastin time, 20-min whole blood clotting test (WBCT20), and platelet count were all within the normal ranges. However, leukocytosis and increased levels of urea, creatinine, D-dimer, and C-reactive protein (CRP) were observed. All of the initial test results normalized spontaneously by the second day of hospitalization, except for the D-dimer level, which continued to increase, and a subsequent increase in the creatinine kinase level. The patient was discharged after treatment with anti-inflammatory drugs and corticosteroids. Within 2 d, he no longer had any pain, and swelling showed significant improvement. He recovered completely after 3 wk.

CASE 2

A healthy 35-y-old man presented with a bite on the dorsal surface of the distal third of his right forearm from an adult *B jararaca* (female, total length 92.6 cm with mutilated tail, mass 254 g) (Figure 2B) sustained while working in his vegetable garden. His home was located in a rural area in the municipality of Itapecerica da Serra (23°43′03"S, 46°50′58"W), about 30 km from the city of São Paulo. Two hours after the snakebite, he was admitted to the HVB with blood pressure 149/76 mm Hg, heart rate 63 beats·min^{-1}, axillary temperature 36.2ºC, and oxygen saturation 98% on room air. He reported pain and swelling, which quickly progressed from the bite site toward his hand and elbow. On physical examination, he displayed excoriations at the bite site and extensive edema, erythema, and sparse ecchymosis throughout his right upper limb (Figure 3D–F). Laboratory tests on admission (Table 1) showed leukocytosis, coagulopathy with fibrinogen consumption, and thrombocytopenia, despite the normal WBCT20.

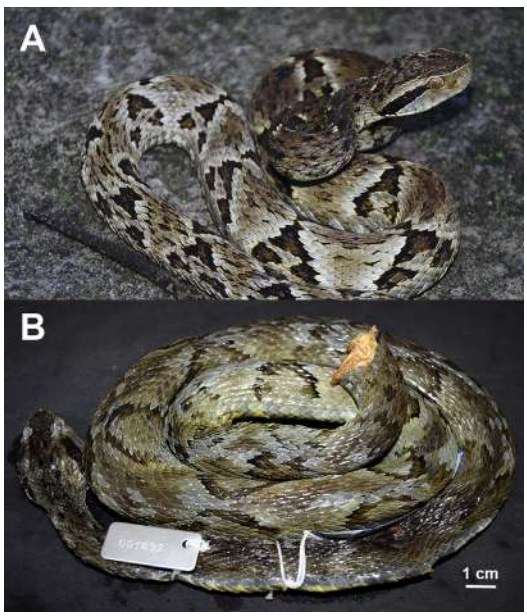

Figure 2. (A) *Bothrops jararaca* (Wied, 1824), a pit viper; living specimen photographed in the municipality of Cotia (23°00′25"S, 47°08′04"W), about 38 km from São Paulo. (B) *Bothrops jararaca* (Instituto Butantan, São Paulo, Brazil number 91432) brought by the Case 2 patient. Adult female, total length 92.6 cm (mutilated tail), mass 254 g, origin Itapecerica da Serra (23°43′03"S, 46°50′58"W), about 30 km from São Paulo. (Photos by Marcelo Ribeiro Duarte).

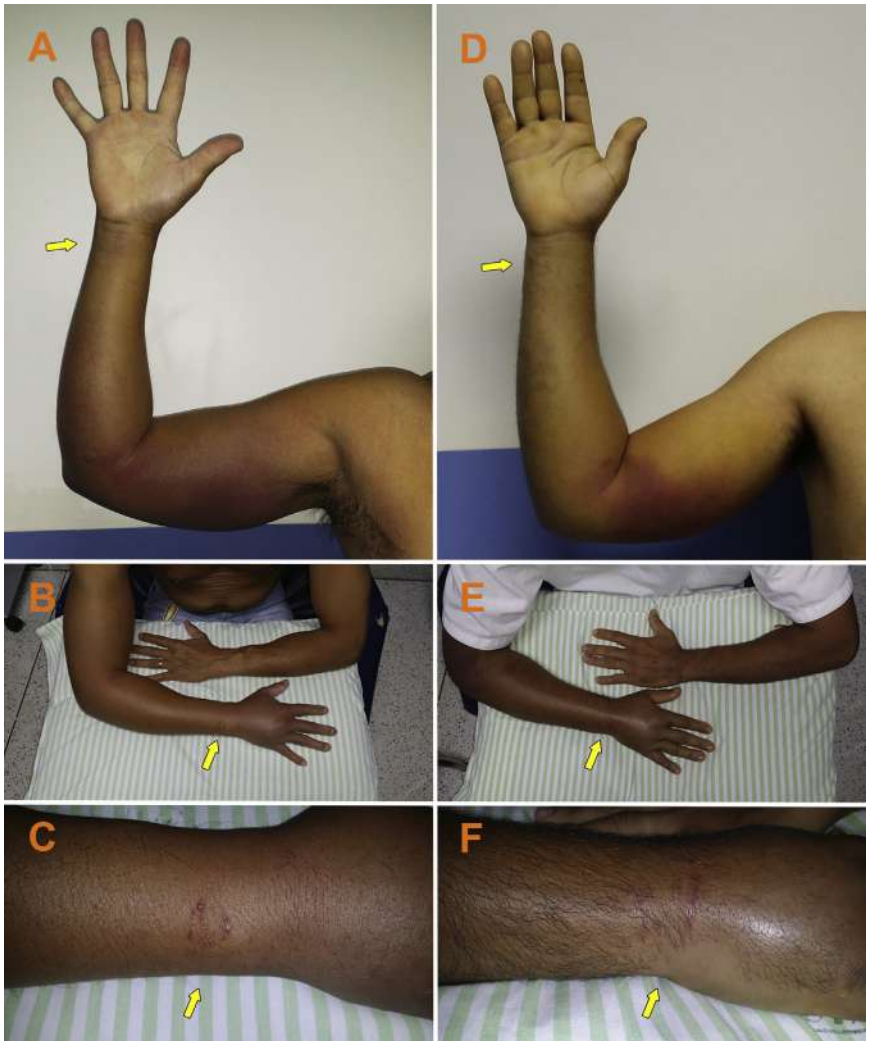

Figure 3. (A, B) Patient bitten by *Philodryas olfersii* on the dorsal surface of the distal third of his right forearm. Physical examination revealed severe edema, erythema, and widespread ecchymoses throughout his right upper limb. (C) Bite site detail showing semicircular bite marks. (D, E) Patient bitten by *Bothrops jararaca* on the dorsal surface of the distal third of his right forearm. Physical examination revealed severe edema, erythema, and widespread ecchymoses throughout his right upper limb. (F) Bite site detail showing excoriations caused by snake's dentition. (Photos by Carlos Roberto de Medeiros).

Eight vials of anti-*Bothrops* antivenom (BAV) were diluted and administered intravenously. BAV is a polyvalent antivenom containing equine-derived antibody fragments [(F(ab')2] against the venom of 5 *Bothrops* species (*B jararaca*, *B neuwiedi*, *B alternatus*, *B moojeni,* and *B jararacussu*), manufactured by Instituto Butantan, São Paulo, Brazil. According to the manufacturer, 1 mL neutralizes the lethality of 5 mg standard *B jararaca* venom. The patient's INR and fibrinogen values reached normal limits 19 h after antivenom administration. On Day 2 after the bite, the patient was afebrile, with erythema and increased temperature at the bite site and elevated CRP. He was treated with chloramphenicol owing to a secondary infection at the bite site. No microbial culture was performed. He was discharged after 4 d with a normal thrombocyte count, but with edema that persisted for approximately 2 wk.

Discussion

Snakebite victims often bring snakes to the hospital or take pictures of the snake. When this occurs, provided that correct identification by an experienced professional takes place, a differential diagnosis is facilitated. Otherwise, because local clinical presentations may be

Table 1. Laboratory test results of patients bitten by *Philodryas olfersii* (Case 1) and *Bothrops jararaca* (Case 2)

Test	*Case 1 Philodryas olfersii bite*			*Case 2 Bothops jararaca bite*				*Reference range*
	Admission	*First day*	*Second day*	*Admission*	*First day*	*Second day*	*Fourth day*	
Hemoglobin ($mg \cdot dL^{-1}$)	16.6	14.1	13.5	15.6	14.8	13.6	13.4	13.5–17.5
Leukocyte ($\cdot \mu L^{-1}$)	17,450	13,100	11,290	18,690	13,230	11,200	6000	4500–11,000
Platelets ($\cdot \mu L^{-1}$)	238,000	204,000	206,000	15,000	72,000	83,000	207,000	150,000–400,000
Sodium ($mEq \cdot L^{-1}$)	-	140	141	142	142	140	–	136–145
Potassium ($mEq \cdot L^{-1}$)	-	4.6	3.7	3.2	4.3	4.7	–	3.5–4.5
Creatinine ($mg \cdot dL^{-1}$)	1.37	1.11	1.02	0.95	0.92	0.92	–	0.70–1.20
Urea ($mg \cdot dL^{-1}$)	65	52	43	25	20	19	–	17–49
AST ($U \cdot L^{-1}$)	21	–	22	22	–	26	37	≤40
ALT ($U \cdot L^{-1}$)	39	–	33	24	–	21	45	≤41
LDH ($IU \cdot L^{-1}$)	–	166	216	199	146	284	154	≤250
Creatinine kinase ($IU \cdot L^{-1}$)	149	–	213	145	132	245	120	≤190
C reactive protein ($mg \cdot dL^{-1}$)	1.4	1.4	0.5	<0.5	2.1	5.0	4.9	<0.5
Fibrinogen ($mg \cdot dL^{-1}$)	278	284	253	103	255	413	–	238–498
D-dimer ($mg \cdot L^{-1}$)	1.943	2.683	2.740	7.530	–	1.975	–	<0.500
PT (s)	13.4	–	12.5	17.2	15.7	14.8	–	11.5–14.5
aPTT (s)	25.7	–	25.2	30.8	29.5	29.3	–	25.4–36.9
INR	1.00	–	1.00	1.32	1.18	1.10	–	0.80–1.20
WBCT20 (s)	Coagulable	–	–	Coagulable	Coagulable	–	–	Coagulable

ALT, alanine aminotransferase; aPTT, activated partial thromboplastin time; AST, aspartate aminotransferase; INR, international normalized ratio; LDH, lactate dehydrogenase; PT, prothrombin time; WBCT20, 20-minute whole blood clotting test.

similar, a differential diagnosis between venomous and "nonvenomous" snakebites can be difficult.[2]

Figure 3 shows that in both cases, the patients were bitten in the same anatomic region and had edema throughout the upper limb, associated with erythema and widespread ecchymoses. These symptoms are common in bites from *Bothrops* spp[3,4] and have been frequently reported in bites from *P olfersii*.[5-7] In Case 1, the bite marks were semicircular and suggestive of a "nonvenomous" snakebite (Figure 3C). In Case 2, the patient displayed excoriations at the bite site rather than the classic 2 perforations of a bite from a pit viper (Figure 3F). Although some authors have considered examination of the bite marks useful in the differential diagnosis between venomous and "nonvenomous" snakebites, its isolated use cannot be considered reliable owing to its low specificity.[8] In a recent study, semicircular bite marks were observed in less than a quarter of 141 cases of bites by *P olfersii*.[5]

Regarding the laboratory tests, in Case 1, the patient did not present with venom-induced consumption coagulopathy (VICC), only leukocytosis and high CRP and D-dimer levels, without consumption of fibrinogen and platelets or changes in prothrombin time, INR, or activated partial thromboplastin time. In the following days, D-dimer levels continued to increase (Table 1), accompanied only by a small increase in creatinine kinase, with no other changes suggestive of VICC. It is possible that these laboratory findings may have been due to extensive local inflammation. It has been shown that the principal effects of *P olfersii* venom in mice are local

edema, inflammatory cell infiltration, and myonecrosis, probably mediated by metalloproteinases, serine proteinases, cysteine-rich secretory proteins, and other components present in *P olfersii* venom.[9] The patient was also seen only 10 h after the bite, which does not allow us to rule out the possibility of transient coagulopathy. However, no clinically documented or confirmed coagulopathy from envenomation by *P olfersii* has been reported in the literature,[5] and the continuous elevation of the D-dimer levels without fibrinogen or platelet consumption observed in subsequent days does not support this hypothesis. Nevertheless, further studies are needed to clarify this issue. However, the patient also showed acute kidney injury, which resolved after hydration. Because there was no substantial increase in lactate dehydrogenase and thrombocytopenia, it is unlikely to have been due to microangiopathic hemolytic anemia previously described in envenomation by other snakes.[10] The drop in hematocrit may have been attributed to hemodilution caused by hydration and, at least in part, to extensive ecchymosis.

Bothrops spp are the most common cause of human snakebites in South and Central America. *Bothrops* envenomation can manifest with local inflammatory signs and hemostatic disorders.[3,4] Common signs and symptoms include pain, swelling, and bleeding, occurring within the first 6 h after envenomation.[3] In Brazil, *Bothrops* envenomation severity is classified as mild, moderate, or severe and treated with 3 to 4, 6 to 8, or 12 vials of BAV, respectively. Mild cases are defined as the presence of mild local signs, such as edema. Moderate cases include regional edema, and severe cases include swelling around the full length of the affected limb, acute kidney injury, shock, and/or severe hemorrhage.[3] These definitions do not take into account the presence or absence of coagulopathy.[3] In Case 2, the patient was admitted 2 h after the bite, with extensive edema, erythema, and sparse ecchymosis throughout his right upper limb. In addition to leukocytosis and elevated D-dimer levels, he presented with active VICC as shown by the consumption of fibrinogen, as well as thrombocytopenia. WBCT20 coagulability was not sensitive enough to detect slight changes in coagulation.[10] These are the classical alterations present in patients bitten by *Bothrops* spp in Latin America,[3,4] which facilitated diagnosis in association with identification of the snake. VICC and thrombocytopenia regressed after the use of the antivenom (Table 1), and the secondary infection was effectively treated with antibiotics.

Differentiating between the 2 types of envenomation is extremely important because treatment involves the use of antivenom. Misuse of antivenom, despite identification of the snake, has been observed in cases of bites by *P olfersii* reported in the literature.[2] Although *P olfersii* venom exhibits immunologic cross-reactivities to polyvalent BAV,[11] its use in the treatment of *P olfersii* bites does not seem to be justified because no controlled clinical study has used the antivenom in the treatment of these patients. Thus, the problem is not restricted to the identification of snakes, but includes misinformation about the differences between the medical risks posed by front-fanged venomous snakes and NFFC snakes, thus contributing to the inappropriate treatment of these bites.[2,5]

In many snakebite cases, the snake responsible remains unidentified, which frequently results in difficulties in deciding which antivenom to administer. However, the diagnosis of a venomous snakebite or determination of the snake possibly responsible for envenomation can be made using clinical criteria.[12] Clinical diagnosis depends on the recognition of envenomation symptoms observed in the patient. Localized symptoms of swelling, ecchymosis, blistering, and necrosis, or systemic symptoms such as hemorrhage, incoagulable blood, hypovolemic shock, neurotoxic signs, and rhabdomyolysis, may be observed depending on the genus of the envenoming snake (in Brazil the genera *Bothrops*, *Crotalus*, *Lachesis*, *Leptomicrurus*, or *Micrurus*).[3,12] In Case 2, the presence of coagulopathy and the correct identification of the snake (*B jararaca*) allowed for safe prescription of antivenom. However, because both factors are not always present, despite the presence of local symptoms, an alternative is the use of immunologic tests,[12] which are unfortunately not available in Brazil. Furthermore, in Case 2, the WBCT20 was not sensitive enough to detect mild VICC, which can be a problem in low-income settings where most snakebites occur and where bedside clotting tests, such as the WBCT20, play a major role in diagnosing coagulopathy.[10] In Case 2, if we could not identify the snake and only used the WBCT20 for the diagnosis of VICC, it would have been dangerous to wait for the progression of symptoms or the diagnosis of coagulopathy; studies have shown that delayed treatment increases the risk of serious complications, such as necrosis and amputation.[3]

In the absence of snake identification, differentiating between bites by *P olfersii* and *Bothrops* spp may be especially difficult in some regions of Brazil, where there are other crotaline species that, although relatively rare, can cause coagulopathy and local symptomology similar to envenomation from *Bothrops* spp, *Lachesis muta* (the only Bushmaster species in the Amazon rainforest and Atlantic Forest of Northeast and Southeast Brazil),[13] and *Crotalus durrissus ruruima* (present in the state of Roraima in Northern Brazil).[14] Envenomation from other subspecies of Brazilian *Crotalus durrissus* (species *cascavella*, *collilineatus*, *dryinas*, *marajoensis*, and *terrificus*) typically results in localized paresthesia at the bite site, with edema and erythema being rare or discrete,

confined to the bite site with ascending edema infrequently observed.[14] Primary systemic symptoms after envenomation by these subspecies are neurotoxicity and myotoxicity (myasthenic facies, prostration, drowsiness, myalgia, hematuria, and kidney failure).[14]

Conclusion

Even if the health professional knows the difference in the medical risks posed by these 2 species, patients bitten by *P olfersii* may present with local symptoms resembling mild-to-moderate *B jararaca* envenomation. This situation can lead to a misdiagnosis in the absence of precise identification of the snake or sensitive tests to detect mild VCCI. The unavailability of immunologic tests worsens the situation. This combination of factors makes it almost impossible to establish a safe differential diagnosis of *Bothrops* spp envenomation and discard the need for antivenom. In this context, a reasonable alternative is to take the risk of prescribing antivenom for a possible *P olfersii* bite, because failure to do so for a real *Bothrops* bite can result in severe complications and sequelae if treatment is delayed.

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REVIEW ARTICLE

The Disease Ecology, Epidemiology, Clinical Manifestations, Management, Prevention, and Control of Increasing Human Infections with Animal Orthopoxviruses

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Zoonotic orthopoxvirus outbreaks have occurred repeatedly worldwide, including monkeypox in Africa and the United States, cowpox in Europe, camelpox in the Middle East and India, buffalopox in India, vaccinia in South America, and novel emerging orthopoxvirus infections in the United States, Europe, Asia, and South America. Waning smallpox immunity may increase the potential for animal-to-human transmission followed by further community transmission person-to-person (as demonstrated by monkeypox and buffalopox outbreaks) and by contact with fomites (as demonstrated by camelpox, cowpox, and, possibly, Alaskapox). The objectives of this review are to describe the disease ecology, epidemiology, clinical manifestations, prevention, and control of human infections with animal orthopoxviruses and to discuss the association with diminished population herd immunity formerly induced by vaccinia vaccination against smallpox. Internet search engines were queried with key words, and case reports, case series, seroprevalence studies, and epidemiologic investigations were found for review.

Keywords: zoonoses, buffalopox, camelpox, cowpox, monkeypox, vaccinia, smallpox, variola

Introduction

After more than 150 y of successful vaccination against smallpox, begun by Edward Jenner in 1798, the World Health Organization declared smallpox eradicated in 1980, and smallpox vaccination with the vaccinia virus-based vaccine ceased.[1-4] In addition to smallpox protection, the vaccinia vaccine provided cross-protective immunity against other related orthopoxviruses, such as cowpox and monkeypox, and now can be used for postexposure prophylaxis.[5,6] Zoonotic orthopoxvirus outbreaks have occurred repeatedly worldwide, including monkeypox in Africa and the United States, cowpox in Europe, camelpox in the Middle East and India, buffalopox in India, vaccinia in South America, and novel emerging orthopoxvirus infections in the United States, Europe, Asia, and South America. These recent outbreaks demonstrate that waning smallpox immunity likely increases the potential for animal-to-human transmission of orthopoxviruses, which can be followed by further community transmission. Moreover, an absence of immunity to smallpox virus creates a risk of greater morbidity from orthopoxvirus infections, as demonstrated by an outbreak of monkeypox in the Democratic Republic of the Congo in 2017 that resulted in 88 cases (63 confirmed and 6 deaths).[5] Young persons never vaccinated for smallpox experienced the greatest morbidity and mortality during these outbreaks.[6]

In addition to waning smallpox immunity, other factors have played a role in the increasing prevalence of orthopoxvirus infections in humans.[7] Human behavior is believed to play the greatest role for 2 reasons.[7] First, the current enthusiasm for ownership of exotic animals and livestock has created an opportunity for the international movement of poxviruses, such as monkeypox, cowpox, and novel vaccinia virus, into close contact with

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nonimmune native animals and humans.[7] Second, the transportation and abandonment of infected companion and hobby animals can release zoonotic orthopoxviruses into naïve environments and precipitate outbreaks in nonimmune animals and humans.[7]

Methods

The objectives of this review are to describe the disease ecology, epidemiology, clinical manifestations, prevention, and control of human infections with animal orthopoxviruses and to discuss the association with diminished population herd immunity formerly induced by vaccinia vaccination against smallpox. To meet these objectives, Internet search engines (Google, Google Scholar, PubMed, Medline, and Ovid) were queried with the keywords as search terms to examine peer-reviewed scientific articles on the most common human infections with animal orthopoxviruses worldwide. The study period was 1970 to 2020. The articles reviewed included disease surveillance studies, seroprevalence studies, review articles, case reports and series, and disease outbreak investigations. Articles excluded from review included non–English-language articles, letters to the editor, dispatches, opinion-editorial articles, clinical-pathological case conferences, and abstracts of posters and presentations at conferences and scientific meetings. The selected methodology met all recommended criteria for narrative reviews, including several keywords, use of 2 or more Internet search engines, a defined study period, and article inclusion and exclusion criteria.[8]

Results

VIROLOGY AND TAXONOMY OF ORTHOPOXVIRUSES

The orthopoxviruses are a group of zoonotic, phylogenetically related, double-stranded DNA viruses with large animal reservoirs in vertebrates, especially in wild and domestic mammals, food animals, and rodents (Table 1). Although most orthopoxviruses (family Poxviridae, subfamily Chordopoxviridae, genus Orthopoxvirus) are host-specific in animals and do not cause disease in humans, several may be transmitted to humans by respiratory droplets or by direct contact with skin lesions or contaminated fomites. There are 17 known species of orthopoxviruses, including human smallpox (variola), with new emerging species frequently reported (Table 1).[9-11]

DISEASE ECOLOGY AND EPIDEMIOLOGY OF HUMAN INFECTIONS WITH ANIMAL ORTHOPOXVIRUSES

Monkeypox in Africa and the United States

Monkeypox has emerged as the most common cause of human orthopoxvirus infections, with most cases reported from Central and West Africa.[5,6,12,13] Monkeypox is transmitted from rodents to humans by rodent bites and close contact with infected live or dead animals or their bodily fluids, often through hunting, skinning, or butchering of bushmeat as food.[5,6,12,13] Human-to-human transmission occurs in approximately 10% of cases by respiratory droplets and by close contact with infected lesions or bodily fluids.[5,6,12,13] The exact host reservoir species for monkeypox is unknown, but monkeys, small mammals, rabbits, squirrels, prairie dogs, and other rodents are suspected.[5,6,12,14,15]

Diagnostic specimens may be obtained from skin lesion or lymph node biopsies.[5,14,15] Treatment is primarily supportive. Close contacts of infected persons may receive the smallpox vaccine prophylactically to prevent transmission postexposure.[5,15,16] Other recommended prevention and control strategies include avoiding physical contact with infected persons; avoiding contact with wild animals, especially those found dead; cooking all animal food products, including bushmeat, thoroughly before consumption; frequent handwashing; and early medical evaluation of all persons with signs or symptoms of monkeypox.[16]

Between May and July 2003, 71 cases of monkeypox were reported from 5 Midwestern US states, including Wisconsin (39), Indiana (16), Illinois (12), Kansas (1), Missouri (2), and Ohio (1).[14,15] The outbreak was traced to imported, infected Gambian pouched rats (*Cricetomys* spp.) that were shipped from Texas to an exotic animal distributor in Illinois, who housed them with prairie dogs destined for retail sale.[14,15] Of the 71 cases, 39 (55%) were in female patients; the median age of case patients was 28 y (range 1–51 y); 18 patients (26%) were hospitalized; and 2 patients, both children, developed severe, complicated illnesses.[14,15] No deaths were reported.[14,15] The median incubation period was 12 d (range 1–31 d).[14,15] Of 35 laboratory-confirmed cases, 32 (91%) tested positive for monkeypox by polymerase chain reaction (PCR) assay, immunohistochemical testing, and/or electron microscopy of lesion biopsies.[14,15] To halt transmission, 30 contacts in 6 states were vaccinated with the vaccinia smallpox vaccine.[14,15] This outbreak was the first time monkeypox, an Old World animal orthopoxvirus, was reported in the United States in native rodents and in humans.[14,15]

Table 1. Orthopoxviruses (family Poxviridae, subfamily Chordopoxviridae): animal reservoirs and transmission mechanisms

Orthopoxviruses (WHO abbreviation)	*Animal reservoirs*	*Geographic distribution*	*Animal-to-human transmission*	*Person-to-person transmission*	*Nosocomial transmission*	*Unique features in addition to a poxvirus syndrome*[a]
Abatino macapox virus	Monkeys	Italy, Europe (unconformed)	NR	NR	NR	NA
Akhmeta virus	Small mammals, cattle	Georgia (former USSR)	+	NR	NR	NA
Alaskapox (AK2015-poxvirus)	Small mammals	Alaska	+	NR	NR	NA
Buffalopox (BPXV)	Buffalo, cattle	India, Pakistan (unconfirmed)	+	+	+	Lesions usually confined to hands, axillary and inguinal lymphadenopathy lesions in oral mucosa after consumption of milk from infected animals
Camelpox (CMLV)	Dromedary camels	North Africa, Middle East, Afghanistan Pakistan, Southern Russia, India	+	NR	NR	Lesions usually confined to hands
Cowpox (CPXV)	Rarely cattle, horses, cats, rodents	Worldwide	+	+	NR	Used for early smallpox vaccination
Cowpox (atypical) France Amiens 2016	Unknown, probably rodents and cats	France	NR	NR	NR	Wound eschar, spreading cellulitis with subcutaneous abscesses, regional lymphangitis and lymphadenopathy, prolonged course
Ectromelia (mousepox) virus (ECTV)	Mice, other rodents	Worldwide	NR	NR	NR	NA
Horsepox	Originally horses and then cattle, probably extinct	Mongolia (last case)	+	NR	NR	Used for early smallpox vaccination
Monkeypox (MPXV)	Reservoir unknown, probably rodents, especially squirrels, and small mammals hunted for bush meat	Central and West Africa, introduced into United States	+	+	+	Cervical, submandibular, and inguinal lymphadenopathy
Rabbitpox	Rabbits	Worldwide	NR	NR	NR	NA
Raccoonpox	Raccoons	Worldwide	NR	NR	NR	NA

(continued on next page)

Table 1 (*continued*)

Orthopoxviruses (WHO abbreviation)	*Animal reservoirs*	*Geographic distribution*	*Animal-to-human transmission*	*Person-to-person transmission*	*Nosocomial transmission*	*Unique features in addition to a poxvirus syndrome*[a]
Skunkpox	Skunks	Worldwide	NR	NR	NR	NA
Taterapox	Rodents, especially gerbils, mice, and voles	Worldwide	NR	NR	NR	NA
Vaccinia virus (VACV)	Cattle, rodents, humans	Worldwide, new pathogenic strains in Brazil and Colombia	+	+	+	Used for smallpox vaccination until eradication by 1980
Variola virus (smallpox)	Humans	Biological weapons stockpiles worldwide	NA	+	+	NA
Volepox	Voles and other rodents	Worldwide	NR	NR	NR	NA

NR, not reported; NA, not applicable; WHO, World Health Organization.

[a]The poxvirus syndrome is characterized by an initial prodrome of fever, malaise, headache, myalgia, and, rarely, nausea and vomiting. A progressive pox stage begins after an incubation period of 10 to 14 d with successive crops of macules, papules, vesicles, pustules, ulcers, dry crusts, and depigmented scars over weeks to months. Neurologic complications include mental status changes, encephalitis, transverse myelitis, neurogenic bladder and bowel, and orbital infection with ophthalmoplegia.

Although uncommon in the United States, a diagnosis of monkeypox should be considered in any person who presents with a febrile prodrome followed by a pustular rash after travel to a country with endemic monkeypox, such as Nigeria and the Democratic Republic of Congo. In laboratory-confirmed cases, the index patient should be isolated immediately, and local and state health departments and the US Centers for Disease Control and Prevention (CDC) should be notified to initiate control investigations and conduct tracing of close contacts exposed to the index case either during travel or after arrival in the United States.

Cowpox in Europe

The cowpox virus is an orthopoxvirus with a reservoir in wild rodents and is no longer found in cows.[17,18] Cowpox is endemic in rats and feral cats throughout Europe and Asia and is typically transmitted to humans via close contact with accidental animal hosts, especially pet rats and cats that acquire infections from mice and other wild rodents.[18-23] In 2002, Dutch investigators reported the first case of rat-to-human transmission of cowpox in a 14-y-old girl who cared for a wild Norway rat (*Rattus norvegicus*) for 6 d and later developed ulcerated nodules on her face.[19]

Although cowpox lesions may be secondarily infected and heal with scarring, the disease is typically self-limited in the immunocompetent but can be generalized and severe in the immunocompromised.[24,25] Fatal, disseminated cowpox infection has been reported in an adolescent renal transplant recipient immunosuppressed by antirejection therapy.[24,25] Antibiotic treatment of secondary infections should be based on wound culture and antibiotic sensitivity testing; tetanus prophylaxis should be administered if indicated.

In early 2009, German investigators described a cluster outbreak of cowpox transmitted to 5 humans by infected Norway rats purchased from the same breeder and pet shop owner near Munich.[22] The investigators observed that the onset and severity of the lesions were associated with the case-patients' vaccinia virus vaccination (VVV) status, with shorter incubation periods of 3 to 5 d, multiple lesions, fever, and lymphadenopathy in patients with no history of VVV, and longer incubation periods of greater than 1 wk with single, smaller lesions and no fever or lymphadenopathy in patients with a positive history of VVV (Figure 1).[22]

In a 2013 molecular analysis of a cowpox outbreak after exposure to infected pet rats in non–smallpox-vaccinated patients in France, investigators detected the presence of cowpox DNA in lesion crusts by PCR and

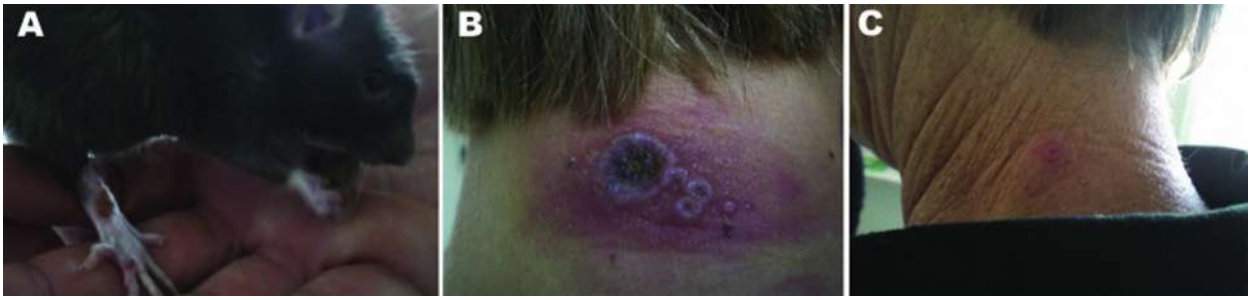

Figure 1. Cowpox lesions on a pet rat (*Rattus norvegicus*) and its owners during an outbreak in Germany in 2009.[21] A, Pet rat with a cowpox lesion on its right forepaw. B, Cowpox lesions on the neck of a pet rat owner without previous vaccinia virus vaccination (VVV) for smallpox 13 d after direct contact with her cowpox-infected pet rat. Regional lymphadenopathy was also described. C, Milder, nearly asymptomatic lesions on the neck of the patient's grandmother, who had a history of VVV for smallpox, 13 d after direct contact with the family's pet rat.

then extracted DNA from cell culture preparations from patient lesions to amplify viral genomes.[23] The results indicated that 3 closely associated patients (2 sisters and their sick pets' veterinarian) were infected with the same the cowpox strain.[23]

In 2019, French investigators reported an atypical case of cowpox in a 45-y-old previously vaccinia vaccinated healthy electrician who sustained a superficial puncture wound to the left lateral thorax from a metal guardrail stuck in the ground.[26] The wound did not heal and developed a black eschar with painful surrounding cellulitis and subcutaneous abscesses that spread to the left anterior chest with regional lymphadenopathy over a 4-wk period (Figure 2A).[26] All abscesses drained spontaneously, the cellulitis resolved by 4 mo, and the patient was discharged from follow-up care at 9 mo (Figure 2B).[26] Lesion samples submitted for electron microscopy, PCR assays, genomic sequencing, and phylogenetic analysis suggested the presence of an atypical novel orthopoxvirus related to cowpox clade E3 and designated Cowpox France Amiens 2016.[26] The investigators concluded that the patient's atypical cowpox infection was most likely transmitted by the initial puncture wound made by the metal guardrail, which was contaminated by ground contact with rodent or cat urine or feces.[26] Rodents and cats that eat infected rodents are well-known reservoir hosts of cowpox virus and can contract infection without clinical disease.[21,26] This case challenged the immunologic capability of prior smallpox-vaccinated persons to maintain prolonged cross-protective immunity against other orthopoxviruses, especially atypical and novel ones.

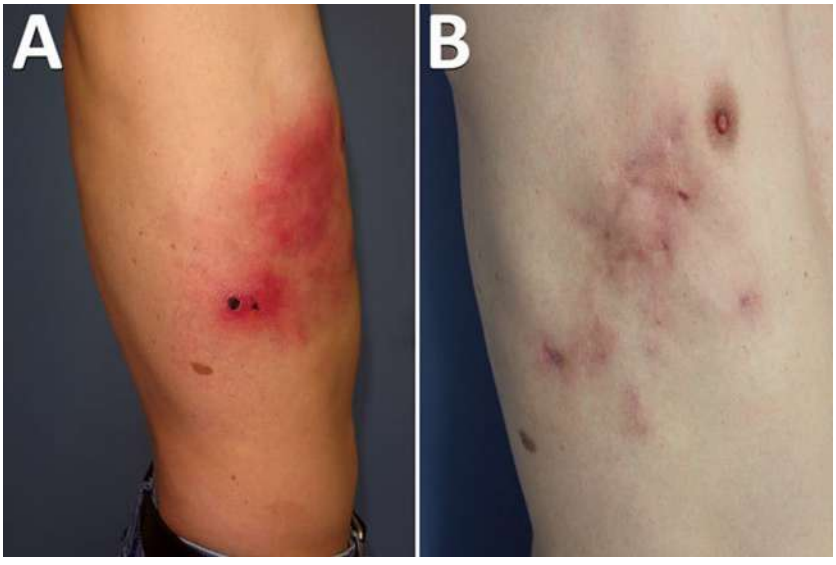

Figure 2. Atypical cowpox virus infection in a smallpox-vaccinated patient in France in 2016.[23] A, Profile appearance of the patient's torso 1 mo after the initial traumatic injury. B, Appearance 9 mo after the initial trauma.

Novel Vaccinia Virus in South America

Since 1999, 2 clades of vaccinia virus phylogenetically related to, but distinct from, the prototype vaccinia viruses used in smallpox vaccinations have caused repeated outbreaks of poxvirus diseases in dairy cows and in dairy and farming industry workers in Brazil and Colombia.[27-30] In 2015, Brazilian investigators described an outbreak of atypically severe vaccinia virus infection in 26 rural dairy farm workers, in which 12 workers who were not vaccinated against smallpox were hospitalized for severe systemic manifestations.[28] All 26 patients were infected after milking dairy cows with active lesions on their udders and teats.[28] All 12 hospitalized patients had high fever, prostration, regional lymphadenopathy, and painful vesicular lesions on the upper and lower extremities.[28] Three hospitalized patients had nausea, vomiting, diarrhea, mental confusion, and seizures.[28]All 14 nonhospitalized patients had been vaccinated for smallpox in childhood and manifested fewer symptoms and fewer lesions, which were confined to the hands and arms, when compared with the 12 unvaccinated, hospitalized patients.[28] All patients recovered with supportive therapy.[28] The causative vaccinia virus was identified by PCR in many cases, and the authors concluded that a new vaccinia virus isolate had caused the outbreak of very severe, exanthematous vaccinia infection.[28]

Buffalopox in India

Buffalopox affecting domestic buffalo, cattle, and humans has occurred in sporadic and epidemic outbreaks in small villages and commercial farms throughout India.[31,32] Despite its host specificity for buffalo and cattle, buffalopox is closer phylogenetically to vaccinia than to cowpox and causes similar clinical manifestations.[31] Investigators reported an attack rate of 12% for buffalo and 7% for villagers in a 2011 descriptive epidemiologic analysis of a large outbreak of buffalopox in 351 patients from 22 villages in western India in 2009.[32] The index case was a villager with a history of milking infected buffalo with lesions on the udders and teats.[32] Direct contact with infected animals was the main mode of transmission, and human-to-animal spread by infected dairy workers was suspected as the main mode of spread in animals.[32]

Most infected patients presented with fever, malaise, and painful pox-like lesions, more often on the fingers and hands than on the face and feet, that were associated with painful axillary and inguinal lymphadenopathy.[32] Children without contact with infected animals also presented with similar constitutional symptoms and painful pox-like lesions on the hands.[32] In some cases, pox lesions occurred in the mouth, suggesting transmission via consumption of contaminated raw milk.[32] Buffalopox was detected by electron microscopy and confirmed by virus isolation in culture and PCR identification in specimens from buffalo and humans.[32]

Camelpox in India

Camelpox is a host-specific orthopoxvirus infection in camels that does not infect other animal species as do monkeypox, cowpox, vaccinia, and buffalopox.[33] It is transmitted to humans by direct contact with contaminated lesions on camels or fomites.[33] Camelpox is closely related phylogenetically to the smallpox virus (variola).[33] Since the 1990s, sporadic outbreaks of camelpox have been reported from camel-rearing areas of the world, from North Africa throughout the Middle East and including India, Pakistan, Afghanistan, and Turkmenistan.[33] Clinical manifestations in camels include pox-like lesions distributed throughout hairless areas, preferentially on the head, neck, and inguinal regions.[33] Similar lesions occur on the mucous membranes of the mouth and digestive and respiratory tracts.[33]

In a 2011 descriptive epidemiologic analysis of the first human cases associated with a prolonged outbreak of camelpox in northwestern India in 2008 to 2009, investigators reported 3 cases of human infection in camel attendants, as confirmed by PCR testing of patient scabs and isolation of camelpox virus in cultures from infected camels.[33] All affected individuals had been exposed to infected camels and presented with fever and pox-like lesions confined to their hands.[33] The lesions began as raised vesicles that burst within 7 to 10 d, leaving deep ulcers that crusted over with scabs that sloughed off and left scars by 15 d.[33]

Novel orthopoxvirus infection, Alaska, 2015

In July 2015, a non-smallpox-vaccinated female patient living near Fairbanks, Alaska, sought urgent medical care for a suspected ulcerating spider bite on her left shoulder accompanied by 5 d of fever, malaise, fatigue, and painful regional lymphadenopathy.[10] A superficial ulcer 1 cm in diameter with 2 adjacent vesicles and a linear streak extending over the shoulder were confirmed by a physician, who unroofed and swabbed the vesicles for testing.[10] Although the samples were negative for herpes simplex, varicella zoster, and variola viruses, a generic orthopoxvirus PCR test was positive.[10] The patient's household contacts had no serologic evidence of orthopoxvirus exposure. Swab

samples from household surfaces and items and 31 small mammals collected from the house perimeter tested negative for orthopoxviruses by PCR.[10] Phylogenetic analysis identified the orthopoxvirus as genetically distinct from, but related to, an Old World clade of orthopoxviruses.[10] The patient's lesions took 6 mo to resolve fully.[10] The investigators concluded that the patient's lesions represented a novel orthopoxvirus infection that resulted from exposure to unidentified, infected wild small mammals or to fomites contaminated by their excreta.[10]

CLINICAL MANIFESTATIONS AND LABORATORY CONFIRMATION OF ANIMAL ORTHOPOXVIRUS INFECTIONS

Although some orthopoxvirus infections are characterized by unique clinical manifestations, all share similar incubation periods and clinical manifestations. The poxvirus syndrome is characterized by an initial prodrome of fever, malaise, headache, myalgia, regional lymphadenopathy, and, rarely, nausea and vomiting. A progressive pox stage begins after an incubation period of 10 to 14 d, with successive macules, papules, vesicles, pustules, ulcers, dry crusts, and depigmented, depressed scars over 3 to 4 wk.[14,15] In addition to viral bronchopneumonias and ocular inoculation, neurologic complications may include mental status changes, seizures, encephalitis, transverse myelitis, and neurogenic bladder and bowel.[14,15] In addition to smallpox, the differential diagnosis of all orthopoxvirus infections should include chicken pox (varicella zoster), herpes simplex, shingles (herpes zoster), cutaneous anthrax, bubonic plague, *Bartonella* spp. infection, ulceroglandular tularemia, and the tickborne rickettsial diseases.[19]

Laboratory confirmation of orthopoxvirus infections is critical to rule out other febrile illnesses with similar cutaneous manifestations. After consultation with state health departments, specimens should be taken directly from skin lesions before smallpox vaccination and submitted to state health departments for analysis by state laboratories equipped for rapid viral identification or sent by unequipped state health departments directly to CDC labs.[14,15] The best specimens are swabs of lesions or entire crusts from healing sores submitted for rapid viral identification by electron microscopy, immunohistochemical techniques, molecular confirmation by PCR, or viral culture in active cases.[14,15] Serologic tests are less dependable and may be complicated by prior smallpox vaccinations or prior orthopoxvirus infections with cross-reacting antibodies.

MANAGEMENT, PREVENTION, AND CONTROL OF ANIMAL ORTHOPOXVIRUS INFECTIONS AND OUTBREAKS

Multiple case reports and series provide evidence supporting cross-reactive immunity to animal orthopoxvirus infections in humans with less severe disease or protection from disease. During repeated monkeypox outbreaks in the Democratic Republic of Congo from 1981 to 1986, prior smallpox vaccination conferred 85% protection against monkeypox.[34] Prior smallpox vaccination was associated with fewer and less severe lesions in cowpox, as noted in a household outbreak in Germany in 2009.[22]

In addition to vaccinia vaccination, other management strategies for human orthopoxvirus infections include intravenous vaccinia immunoglobulin (VIG), intravenous cidofovir, and oral tecovirimat. Intravenous VIG has been used to treat the complications of smallpox vaccination, such as generalized vaccinia, eczema vaccinatum, ocular vaccinia, and postvaccinial central nervous system complications.[14,15] There were no requests for VIG in the US monkeypox outbreak in 2003.[14,15]

Although intravenous cidofovir has proven effective in the postexposure prophylaxis of monkeypox in monkeys, no data exist on the use of either VIG or cidofovir for prophylaxis or treatment of monkeypox.[35,36] The CDC has issued guidance recommendations that cidofovir only be used for the treatment of life-threatening monkeypox and not for prophylaxis.[36] In 2018, the US Food and Drug Administration (FDA) approved the first oral antiviral medication, tecovirimat, for the treatment of smallpox (FDA News, July 2018). In 2019, investigators reported the first use of oral tecovirimat in conjunction with intravenous VIG in the management of a case of laboratory-acquired vaccinia virus infection after an accidental needlestick on the finger.[37] Within 48 h of initiating therapy, local pain and edema at the needlestick site decreased, and fever and axillary lymphadenopathy resolved.[36]

In 2021, ophthalmologists in London reported the successful use of oral tecovirimat in the management of a case of orbital cowpox in a 28-y-old female patient who presented with conjunctival necrosis, orbital swelling, and ophthalmoplegia after failed treatment with topical and intravenous antibiotics and steroids.[38] Two weeks before the patient first presented with symptoms, her pet cat developed lesions on its head and paws that were scraped by a veterinarian and tested positive for orthopoxvirus by PCR.[38] A PCR test on a conjunctival swab from the patient also tested positive for orthopoxvirus, and genome sequencing documented a diagnosis of cowpox in the patient and her cat.[38] In addition to orbital decompression and debridement, the patient received a

prolonged course of oral tecovirimat for several months.[38] At 6-mo follow-up, visual acuity in the right eye was normal, but residual ptosis and restriction of extraocular movements remained.[38] This case demonstrated another example of human infection with animal orthopoxvirus and confirmed the effectiveness of tecovirimat in treating cowpox virus infection. Tecovirimat is now maintained in the US Strategic National Stockpile (SNS), the nation's largest supply of essential pharmaceuticals and medical supplies for use in public health emergencies, including a biological weapons attack with smallpox or another orthopoxvirus.[36,37,39]

On September 24, 2019, the FDA announced the approval of the Jynneos smallpox and monkeypox vaccine, a live, nonreplicating vaccine for the prevention of smallpox and monkeypox (FDA News, September 2019). The Jynneos vaccine does not contain live smallpox or monkeypox virus but does contains a modified nonreplicating form of the vaccinia virus, vaccinia Ankara, which does not cause human disease. The Jynneos vaccine is also maintained in the SNS.[36,39]

It is unknown how many people born before 1980 were vaccinated for smallpox and remain relatively protected against smallpox and related orthopoxviruses. Because the world population has doubled from about 4 billion in 1979 to about 8 billion in 2020, as many as 4 billion people may be unvaccinated and susceptible to smallpox and closely related orthopoxviruses. Although military personnel were routinely vaccinated for smallpox in the past, only military and civilian laboratory personnel who work directly with smallpox or related animal orthopoxviruses should be vaccinated today.[36] Smallpox vaccine is no longer available to the general public in the United States.[36,39] However, in the event of a smallpox outbreak, the SNS maintains enough smallpox vaccine to provide postexposure prophylaxis.[36,39]

Conclusions

Since the eradication of smallpox in 1980, orthopoxvirus outbreaks have occurred repeatedly worldwide. Young persons never vaccinated against smallpox experienced the greatest morbidity and mortality during these outbreaks. As population herd immunity formerly induced by vaccinia vaccination against smallpox wanes, there is increased potential for animal-to-human transmission of zoonotic orthopoxviruses, followed by fomite and person-to-person community transmission of animal orthopoxviruses. Only early recognition and management of orthopoxvirus infections in animals by isolation, quarantine, and vaccination, if available, can limit transmission of zoonotic orthopoxviruses to other animals and their human caregivers.[7]

Case isolation and administration of the recently approved, nonreplicating modified Ankara vaccinia vaccine and new antivirals, such as tecovirimat, may limit community contact and person-to-person transmission of orthopoxviruses after outbreaks.[39] Clinicians should consider a diagnosis of orthopoxvirus infection in patients with ulcerating vesicular lesions after urban or rural domestic, wild, or exotic animal exposures, especially close contacts with rodents, feral cats, insectivores, cows, buffalo, and camels.

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REVIEW ARTICLE

Nephrotoxic Mushroom Poisoning: Global Epidemiology, Clinical Manifestations, and Management

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Because mushroom poisonings are increasing worldwide after ingestions of known, newly described, and formerly considered edible species, the objectives of this review are to describe the global epidemiology of nephrotoxic mushroom poisonings, to identify nephrotoxic mushrooms, to present a toxidromic approach to earlier diagnoses of nephrotoxic mushroom poisonings based on the onset of acute renal failure, and to compare the outcomes of renal replacement management strategies. Internet search engines were queried with the keywords to identify scientific articles on nephrotoxic mushroom poisonings and their management during the period of 1957 to the present. Although hepatotoxic, amatoxin-containing mushrooms cause most mushroom poisonings and fatalities, nephrotoxic mushrooms, most commonly *Cortinarius* species, can cause acute renal insufficiency and failure. Several new species of nephrotoxic mushrooms have been identified, including *Amanita proxima* and *Tricholoma equestre* in Europe and *Amanita smithiana* in the United States and Canada. In addition, the edible, hallucinogenic mushroom *Psilocybe cubensis* has been noted recently via mass spectrometry as a rare cause of acute renal insufficiency. Renal replacement therapies including hemodialysis are often indicated in the management of nephrotoxic mushroom poisonings, with renal transplantation reserved for extracorporeal treatment failures.

Keywords: orellanine, *orellanus* syndrome, allenic norleucine, rhabdomyolysis, mushroom-induced

Introduction

INCREASING MUSHROOM POISONINGS

Mushroom poisonings are increasing worldwide today for many reasons, including novices mistaking poisonous species for edible ones, recent immigrants mistaking poisonous species for edible ones back home, and adolescents mistaking poisonous species for hallucinogenic ones.[1–6] The American Mycological Association and the US Centers for Disease Control and Prevention have warned clinicians that mushroom intoxications continue to occur among newly arrived migrant groups accustomed to foraging for edible mushrooms in their home countries who are not familiar with local mushroom ecology.[2,3]

In addition to the risk factors for mushroom poisoning noted among novices and recent immigrants, the recreational ingestion of hallucinogenic mushrooms has become a popular form of substance abuse among adolescents and young adults that can also result in mistaking poisonous mushrooms for hallucinogenic ones.[4,5] Among 174 adolescents previously identified as substance abusers, 45 (26%) reported having ingested psilocybin-containing hallucinogenic mushrooms, often in conjunction with alcohol and other drugs.[4] In a 2012 survey study of 882 randomly selected college students, 30% of the 409 responders reported having ingested hallucinogenic mushrooms (mean number of ingestions 3.4; mode 1) on several occasions.[5]

NEPHROTOXIC MUSHROOM POISONINGS

Recently, several new species of *Amanita* mushrooms have been identified as nephrotoxic, including *A proxima* in Europe, *A smithiana* in Canada and the United States, *A pseudoporphyria* in Japan, and *A punctata* in Korea.[7–13] Prior case reports have also suggested, without any laboratory confirmation, that acute renal failure could result from the consumption of hallucinogenic *Psilocybe* species mushrooms.[14,15] In 2019, a case of acute kidney

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injury was reported after consumption of edible, hallucinogenic *Psilocybe cubensis* mushrooms from a psychedelic mushroom "grow kit" purchased online.[16] A sample from the crop of *P cubensis* mushrooms consumed in this case was noted via mass spectrometry to contain the hallucinogenic toxin psilocin.[16] In addition to directly nephrotoxic mushrooms, newly recognized myotoxic mushrooms can cause rhabdomyolysis with massive myoglobin release that may indirectly cause acute kidney injury, including the formerly considered edible *Tricholoma equestre* in Europe and *Russula subnigricans* in China.[17–19]

Because mushroom poisonings are increasing worldwide after ingestions of known, newly described, and formerly considered edible species, the objectives of this review are to describe the global epidemiology of nephrotoxic mushroom poisonings, to identify nephrotoxic mushrooms, to present a toxidromic approach to earlier diagnoses of nephrotoxic mushroom poisonings based on the onset of acute renal failure, and to compare the frequencies and outcomes of renal replacement management strategies.

SEARCH STRATEGY

Internet search engines and databases including PubMed, Google Scholar, Google, and Ovid Medline were queried with the keywords as medical subject headings to identify scientific articles on nephrotoxic mushroom poisonings and their treatments and outcomes during the search period, 1957 to present. The keywords included mushrooms, poisonous, nephrotoxic; *Amanita*, poisonous, nephrotoxic; *Cortinarius*, poisonous, nephrotoxic; orellanus syndrome, orellanine; and allenic norleucine.

The articles selected to meet the first objective to identify nephrotoxic mushroom species included case reports, case series, and review articles on nephrotoxic mushroom poisonings. The articles selected to meet the second and third objectives to present a toxidromic approach to earlier diagnoses and to compare the frequencies and outcomes of renal replacement treatment strategies included observational studies, retrospective descriptive and analytical epidemiologic studies, and toxicologic investigations. Articles excluded from review included letters to the editor, dispatches, opinion-editorial articles, clinical-pathological case conferences, and abstracts of posters presented at conferences and scientific meetings. These selection methodologies met all recommended criteria for narrative reviews, including use of several keywords, use of 2 or more Internet search engines, a defined study period, and article inclusion and exclusion criteria.[20]

Figure 1. *Cortinarius orellanus* has a large orange to rusty brown cap with gills underneath connected to a thick stem without a ring or annulus. It contains highly nephrotoxic orellanine compounds. Source: Wikimedia Commons (public domain). Photographer: Michael 11. Available at https://en.wikipedia.org/wiki/Orellani#/media/File:Corellanus.jpg.

THE GLOBAL EPIDEMIOLOGY OF NEPHROTOXIC MUSHROOM POISONINGS AND THE INITIAL IDENTIFICATION OF NEPHROTOXIC MUSHROOMS

Nephrotoxic Cortinarius Mushrooms

Nephrotoxicity and mortality after ingestion of *Cortinarius* species mushrooms was initially described in Poland in 1957 in a series of 102 cases of acute renal failure with 11 fatalities in patients who had consumed cooked *Cortinarius orellanus* mushrooms.[21] By 1962, Polish investigators had isolated a crude extract from *C orellanus*, which was named orellanine and which caused renal toxicity when administered orally to experimental animals.[22]

In 1990, French investigators reported a case series of 26 healthy young men who developed acute renal failure after ingesting a mushroom soup made with *C orellanus*.[23] All patients were hospitalized within 10 to 12 d of ingestion, with 12 patients presenting in acute renal failure with acute tubulointerstitial nephritis on renal biopsy.[23] Among these 12 patients, 8 required hemodialysis and recovered rapidly.[23] The remaining 4 patients developed chronic renal failure lasting for months and ultimately required kidney transplants.[23] Of the remaining 14 patients, 12 developed leukocyturia, and all 12 had normal renal function at 1-y follow-up.[23] Another group of investigators later followed 12 of the 26 men who had developed renal failure for a period of 13 y after ingestion of the *C orellanus* mushroom soup.[24] Of these 12 patients, 7 recovered normal renal function, 4 underwent kidney transplant for chronic renal failure, and 1 patient on hemodialysis died in a car accident.[24] Investigators estimated the incidence of acute renal failure after *C orellanus* ingestion to range from 30 to 46% depending on individual sensitivity, pre-existing nephropathy, and the cumulated dose of nephrotoxin ingested.[24]

Figure 2. *Amanita smithiana* is native to the Pacific Northwest of the United States and Canada, where it has been mistaken for the edible pine or matsutake mushroom, *Tricholoma magnivalere*. It has a large white convex cap with unattached free gills and a thick, shaggy white stem with a torn or absent ring. Source: Wikimedia Commons (public domain). Photographer: Sava Krstic. Available at https://upload.wikimedia.org/wikipedia/commons/8/89/Amanita_smithiana_283102.jpg.

Investigators also noted that renal failure regressed progressively over several months in 60% of cases.[24]

Although most *Cortinarius* poisonings to date have been reported from Poland and France, *Cortinarius* species mushrooms are widely distributed in the coniferous forests of the Scandinavian countries, Britain, the United States, Canada, and Australia.[25] In 1995, Swedish investigators reported a case series of 22 patients who were poisoned after consuming cooked *C speciosissimus* mushrooms during the period of 1979 to 1993 (Figure 1).[25] Nine patients developed chronic renal failure, and 5 patients required kidney transplants.[25] Three of these patients underwent transplantation after up to 6 mo of hemodialysis.[25] Two patients who did regain some renal function restarted hemodialysis 24 and 30 mo later and received kidney transplants.[25] The investigators concluded that delayed renal failure could follow partial renal recovery by many years and that renal transplantation could guarantee successful outcomes even years after mushroom poisonings.[25]

NEPHROTOXIC AMANITA MUSHROOMS

In 1994, French investigators were the first to report 5 cases of acute renal insufficiency after consumption of cooked *A proxima* mushrooms that were most likely mistaken for the edible *Amanita* species *A ovoidae*, which shares the same habitat.[7] Temporary hemodialysis was required in 4 of the 5 cases, and all patients recovered quickly, with normal renal and hepatic function restored by 3 wk.[7]

In 1998, Canadian investigators in British Columbia reported 4 cases of renal failure in patients who had consumed cooked *A smithiana* mushrooms and developed gastrointestinal symptoms 5 to 8 h after ingestion (Figure 2).[10] One of the patients, an elderly patient with diabetes, presented to an emergency department with renal failure the day after a mushroom meal and required hemodialysis.[10] The remaining 3 patients presented to local emergency departments 5 to 6 d after mushroom ingestions and also received supportive care with hemodialysis.[10] All patients subsequently regained normal renal function.[10]

In 2009, clinicians in Portland, Oregon, reported 4 additional cases of acute renal failure after consumption of cooked *A smithiana* mushrooms in patients who mistook *A smithiana* for edible matsutake (*Tricholoma matsutake*) mushrooms.[11] All patients presented with gastrointestinal symptoms from 20 min to 12 h after mushroom consumption.[11] All patients subsequently developed acute renal failure 4 to 6 d post-ingestion, and all received temporary hemodialysis for several weeks before regaining normal renal function.[11] The laboratory ranges of the presenting serum biomarkers of acute renal failure in these cases included a blood urea nitrogen of 72 to 91 $mg \cdot dL^{-1}$ and a creatinine level of 12 to 14 $mg \cdot dL^{-1}$.[11] In addition to *A proxima* in Europe and *A smithiana* in North America, delayed onset of acute renal failure was reported from Asia after consumption of other species of *Amanita* mushrooms,

Figure 3. *Psilocybe cubensis* hallucinogenic mushrooms cultivated in a home "grow kit" or "grow box" easily purchased online. Source: Wikimedia Commons (public domain). Photographer: Lord Toran. Available at https://commons.wikimedia.iorg/wiki/File:growbox-cubensis.jpg.

Figure 4. *Tricholoma equestre* (synonym *T flavovirens*), also known as the yellow knight mushroom, has a large flat yellow cap with gills underneath connected to a long thick stem without a ring. *T equestre* has caused delayed and fatal rhabdomyolysis with acute renal failure. Source: Wikimedia Commons (public domain). Photographer: Matthias Renner. Available at. https://upload.wikimedia.org/wikipedia/commons/8/82/Tricholoma_equestre.jpg.

specifically *A pseudoporphyria* in Japan (2003) and *A punctata* in Korea (2015).[12,13]

PSILOCYBE CUBENSIS

In 2019, the first case of acute kidney injury was reported in a 15-y-old Canadian male who consumed several hallucinogenic *P cubensis* mushrooms from a psychedelic mushroom "grow kit" purchased online along with 3 of his friends (Figure 3).[16] The suspect mushrooms were identified morphologically by a mycologist, and the serotonergic hallucinogen psilocin was identified by liquid chromatography-mass spectrometry in a sample of the mushroom meal consumed.[16]

Before identification of the ingested mushrooms, the patient's initial presentation was consistent with orellanine toxicity after ingestion of *Cortinarius* mushrooms.[16] However, orellanine was not detected in the mushroom meal sample, and the 3 other boys who ingested mushrooms from the patient's mushroom meal remained asymptomatic and had normal renal function.[16] In this case, no other cause of acute renal injury was suspected by history or detected by laboratory analyses, including rhabdomyolysis, which was ruled out by normal serum creatine kinase (CK) levels.[16] Hallucinogenic *P cubensis* mushrooms are frequently consumed recreationally without adverse effects other than negative sensory experiences.[4,5,16] Future case reports and toxicologic studies will be required to document the nephrotoxicity of *P cubensis* mushrooms.

RHABDOMYOLYSIS-CAUSING MUSHROOMS

Finally, there are 2 species of myotoxic mushrooms: *Tricholoma equestre*, first reported as toxic in France in 2001, and *Russula subnigricans*, first reported as toxic in China in 2015. Both mycotoxins can cause potentially fatal rhabdomyolysis resulting in acute renal failure after consumption.[17–19]

In 2001, French investigators reported 12 cases of delayed rhabdomyolysis with 3 fatalities in patients who had consumed consecutive meals of the edible wild mushroom *Tricholoma equestre*, harvested from pine forests in coastal southwestern France (Figure 4).[17] After a prodrome of afebrile fatigue and myalgia 24 to 72 h after the last mushroom meal, most (n=8) patients described worsening weakness and stiffness of their legs, accompanied by facial erythema, mild nausea without vomiting, profuse sweating, and darkening urine color over 3 to 4 d.[17] Rhabdomyolysis was later confirmed by significantly elevated CK levels without laboratory evidence of cardiac or hepatic injury.[17] Muscle biopsy in 6 patients demonstrated histopathologic evidence of acute myopathy.[17] In all but 3 of the patients, serum CK levels normalized and most symptoms resolved, but muscular weakness persisted for weeks.[17]

In the 3 fatal cases, serum CK levels continued to rise, and all patients developed hyperthermia up to 42°C,

Figure 5. *Russula subnigricans* mushrooms are native to Asia, with poisonings reported from China, Taiwan, and Japan, where they have been mistaken for edible *Russula nigricans* mushrooms. *R subnigricans* has caused delayed and fatal rhabdomyolysis with acute renal failure. Source: Lin et al.[18]

cardiac arrhythmias, renal dysfunction (elevated serum creatinine, blood urea nitrogen, and potassium), and cardiovascular collapse.[17] Autopsy revealed myocardial lesions identical to the muscle biopsy lesions in 1 patient, renal lesions in 1 patient, and no histopathologic evidence of hepatic damage.[17]

In 2015, Chinese investigators reported 7 cases of delayed rhabdomyolysis with 1 fatality in a family (age range 18–58 y) who had consumed 1 meal of cooked *Russula subnigricans* mushrooms harvested from the forests of Guizhou Province in southern China (Figure 5).[18] Twenty hours later, all 7 family members were hospitalized with nausea, vomiting, diarrhea, dizziness, fatigue, and muscle weakness.[18] Five patients presented with myalgias in the upper legs.[18] No patient was febrile on admission.[18] Although serum creatinine and coagulation tests were normal in all patients, serum CK levels were elevated in 6 patients, and all 7 had moderate elevations in serum alanine aminotransferase and aspartate aminotransferase.[18] In 4 of the 6 patients with elevated serum CK levels, the CK continued to rise, weakness worsened, and urine color darkened, consistent with rhabdomyolysis.[18] All 4 patients received hemodialysis to prevent acute kidney injury, and their serum CK levels began to decline to normal ranges by the third day of hemodialysis.[18] In the single fatality, a 50-y-old male, weakness and myalgia with dark urine worsened during the first 12 h after admission, hemodialysis was initiated on the second day after admission, and hyperthermia up to 40°C developed on the third day after admission.[18] Cardiac arrhythmias with QRS widening and cardiovascular collapse ensued as the serum CK levels rose to a maximum of 228,750 UL^{-1} (laboratory normal range 38–174 UL^{-1}), and the patient died 4 d after admission.[18] Autopsy was not performed.[18]

In summary, retrospective epidemiologic analyses conducted worldwide have confirmed that nephrotoxic mushrooms can cause both reversible and irreversible acute and delayed-onset renal failure, with some cases progressing to chronic renal failure, and a few species can cause potentially fatal rhabdomyolysis with its indirectly associated risks of renal damage and failure.

CLINICAL MANIFESTATIONS OF NEPHROTOXIC MUSHROOM POISONING

The currently known and suspected nephrotoxic mushroom species stratified by genera are listed in Table 1. The ecologic and morphologic features of nephrotoxic mushrooms and the clinical manifestations of nephrotoxic mushroom poisonings are described in Table 2.

IDENTIFYING NEPHROTOXINS IN MUSHROOMS AND IN POISONED PATIENTS

Today, heat-stable nephrotoxins such as orellanine (a tetrahydroxylated-N-oxide bipyridine) in *Cortinarius* species mushrooms, allenic norleucine (2-amino-4,5-hexadienoic acid) in *Amanita* species mushrooms, and psilocin in *P cubensis* mushrooms can be most accurately measured directly in the serum and urine of poisoned patients using immunologic and chromatographic techniques, such as enzyme-linked immunosorbent assay, radioimmunoassay, thin-layer chromatography, high-performance liquid chromatography, and liquid chromatography-mass spectrometry.[26,27]

The mechanisms of orellanine's toxicity in *Cortinarius* species mushrooms are unknown but may be the result of an unidentified nephrotoxic metabolite.[27] Toxicologists have demonstrated that oxidated orellanine can generate orthosemiquinone anion radicals in vitro that produce oxygen free radicals and deplete glutathione.[27] Investigators have postulated that the oxidation of orellanine in the kidneys may result in an accumulation of quinone metabolites that covalently bind to renal tissues and cause cellular damage.[27]

Portuguese toxicologists used thin-layer chromatography and *A smithiana* toxin to test several other *Amanita* species for a suspected nephrotoxin.[9] The *A smithiana* nephrotoxin, now known as allenic norleucine, was detected in samples of *A boudieri*, *A gracilor*, and *A echinocephala*.[9] The investigators concluded that the intoxications produced by these *Amanita* mushrooms would resemble the toxidrome that followed the consumption of *A smithiana*, with a delayed onset of acute, reversible renal failure and mild hepatitis with

Table 1. Nephrotoxic mushroom species

Cortinarius	*Amanita*	*Tricholoma*	*Russula*	*Psilocybe*
C bruneofulvus	*A boudieri*	*T equestre* (synonym *T flavovirens*)	*R subnigricans*	*P cubensis*
C brunneoincarnata	*A echinocephala*			
C callisteus (suspected)	*A gracilor*			
C cinnamomeus (suspected)	*A neoovoidea*			
C henrici	*A pseudo-porphyria*			
C limonius (suspected)	*A proxima*			
C ranierensis	*A punctata*			
C sanguineus (suspected)	*A smithiana*			
C speciosissimus (synonym *C rubellus*)				
C splendens (suspected)				

Table 2. Ecology and morphology of nephrotoxic mushroom species and clinical manifestations of poisonings

	Amanita mushrooms	*Cortinarius mushrooms*	*Russula subnigricans*	*Tricholoma equestre*	*Psilocybe cubensis*
Family	Amanitaceae	Cortinariaceae	Russulaceae	Tricholomataceae	Hymenogastraceae
General descriptions	Large off-white mushrooms; about 600 species; at least 7 are nephrotoxic; many are edible	Largest mushroom family with 2000–3000 species; at least 8 are nephrotoxic; none are recommended as edible worldwide; more nephrotoxic species likely exist	A creamy white mushroom native to eastern Asia, where ingestions have resulted in outbreaks of rhabdomyolysis associated with ARF	A yellow mushroom also known as the yellow knight; formerly considered edible; rhabdomyolysis-associated nephrotoxicity may follow several mushroom meals; many *Tricholoma* species are edible	Frequently cultivated for its hallucinogenic effects shortly after ingestion, caused by its primary active serotonergic compound, psilocybin
Geographic distribution	Worldwide	Worldwide	China, Japan, Korea, Taiwan	Worldwide	Worldwide in a semitropical band above and below the equator
Preferred habitat	Leaf and needle litter and decaying wood of coniferous and oak woodlands with chalky (limestone) soil	Needle litter of coniferous woodlands with chalky (limestone) soil	Leaf litter of deciduous oak woodlands in mountainous regions	Needle litter of pine forests with sandy soil	Moist, sunny, grassy fields and meadows used for cattle grazing; requires meadow habitats containing cow dung
Color (adult)	Large, white caps that can flatten out from convex to plano-convex	Large orange to rusty brown caps that flatten from convex to flat with age	Large dull creamy white caps that flatten from convex to flat to concave with age	Large yellow to yellow-green cap with yellow gills	Golden brown conical to convex cap that flattens with age; gills darken with age
Color (spore)	Rust brown	Brown	White	White	Dark purple brown
Smell	Described as unpleasant	Slight radish smell	Not distinctive	Not distinctive	Distinctive smell of moist cucumber or watermelon rind
Distinctive mycologic features	Cap convex to flat with age; free gills; stem (stipe) is shaggy with an easily torn annulus (ring) and a prominent volva (base)	Large flat orange to rusty brown caps with gills connected to stems without rings; top of the cap may have a darker umbo (protuberance)	Large dull creamy white caps that flatten from convex to flat to an everted umbrella shape with age; gills attached to thick, stems; no annulus	Large yellow to yellow-green cap with yellow gills attached to an even diameter stem without a ring	Small golden brown conical caps that become more convex to flat to slightly everted umbrella shape; dark gills are attached to long, thin stems with a white annulus
Dimensions (adult)	*C orellanus*	*A smithiana*	*R subnigricans*	*T equestre*	*P cubensis*
Height, cm	6–12	6–18	4–10	7–10	4–15
Diameter (cap), cm	3–8	5–17	5–18	5–10	2–8
Edible species most commonly mistaken for	*A proxima* has been mistaken for edible *A ovoidae. A smithiana* has been mistaken for edible *Tricholoma magnivalere.*	*Cortinarius* species mushrooms have been mistaken for edible chanterelles. *C speciosissimus* (synonym *C rubellus*) has been mistaken for edible *Cratellus tubaeformis* and *Hygrophorus* species.	*R subnigricans* has been mistaken for *R nigricans*, a less poisonous species that may be consumed.	*T equestre* was formerly considered edible. It has been mistaken for other edible *Tricholoma* species, such as *T aestuans*, *T auratum*, and *T sulphureum*.	Often confused with other *Psilocybe* species that contain different amounts of psilocybin.
Clinical toxidromes after ingestion	Rapid-onset GI distress in 6–12 h. Lab evidence ARF in <2–4 d.	Rapid-onset GI distress in 6–12 h. Lab evidence of ARF in >2–14 d.	Delayed-onset GI distress in >24–72 h. Delayed lab evidence of rhabdomyolysis in 3–5 d.	Delayed-onset GI distress in >24–72 h. Delayed lab evidence of rhabdomyolysis in 3–5 d.	Hallucinations within 20–30 min with facial flushing, mydriasis, tachycardia. Resolves in 4–6 h.

(continued on next page)

Table 2 *(continued)*

	Amanita mushrooms	*Cortinarius mushrooms*	*Russula subnigricans*	*Tricholoma equestre*	*Psilocybe cubensis*
Associated presenting clinical features	Fever, chills, headache, anorexia, fatigue, abdominal and flank pain	Fever uncommon, oliguria progressing to anuria, abdominal and flank pain	Fever uncommon, dizziness, fatigue, weakness, myalgias in upper legs, vomiting uncommon, darkening urine	Fever uncommon, weakness and stiffness in lower extremities, facial erythema and edema, profuse sweating, vomiting uncommon, darkening urine	Potential for delayed onset of ARF within 36 h that resolves with supportive therapy
Risk of CRF	40%	40%	20%	20%	NR

ARF, acute renal failure; CFR, case fatality rate; CRF, chronic renal failure; GI distress, gastrointestinal distress (nausea, vomiting, diarrhea); NR, not reported.

transaminitis.[9] The mechanisms of allenic norleucine's toxicity are unknown, but it is a suspected direct nephrotoxin.[9] It causes renal epithelial cell necrosis when cultured with renal tubular epithelium in vitro and does not deplete glutathione as does orellanine.[9]

The hallucinogenic nephrotoxin in *P cubensis* is the tryptamine psilocybin, which is dephosphorylated by hepatic alkaline phosphatase to its active metabolite, psilocin.[16] Both psilocybin and psilocin are multiple serotonin receptor agonists, and their agonism at the 5-HT2A serotonin receptor accounts for most of their hallucinogenic properties.[16]

French investigators administered boiled extracts of *T equestre* to mice, which developed significant increases in CK levels and postmortem histopathologic evidence of striated muscle damage compared to control mice, which received boiled extracts from another species of mushroom and maintained normal CK levels and muscle biopsies.[17] The investigators concluded that because most patients with toxic rhabdomyolysis survived, a genetic muscular susceptibility may have triggered the fatal dose-related myotoxic effects after an ingestion threshold of mushrooms was exceeded.[17]

In 2016, Japanese investigators isolated a unique compound from *R subnigricans* mushrooms, cyclopropylacetyl-(R)-carnitine, which caused elevated CK levels in mice and was suspected to be the human myotoxin.[19]

Visual and microscopic identification of poisonous mushrooms and their spores by experts may offer a more rapid means of identifying mushrooms as potentially nephrotoxic in the field than immunologic and chromatographic techniques in the laboratory. In 2012, Boston-based investigators reported 2 cases of hepatotoxic *Amanita* mushroom poisoning in Ukrainian immigrants in the Boston area; the authors attributed their successful supportive management to early identification of the ingested mushrooms as containing amatoxin via an initial cellphone image transmitted to a consulting poison control center mycologist.[28] Nevertheless, there are no specific antidotes for nephrotoxic mushroom poisoning, all treatments are supportive, and precise identification of the causative species is unnecessary in the short term but recommended for epidemiologic and educational purposes in the long term.

MANAGEMENT OF NEPHROTOXIC MUSHROOM POISONINGS

The general management of nephrotoxic mushroom poisonings should include fluid resuscitation and oral activated charcoal (1 $g \cdot kg^{-1}$) within the first 1 to 4 h after ingestion. A baseline laboratory assessment should include complete blood count; peripheral blood smear; serum glucose and electrolytes, including calcium; liver and renal function tests; and serum CK. Hepatic transaminases, coagulation studies, serum bilirubin, serum glucose, serum creatinine, blood urea nitrogen, and CK will serve as baseline comparative laboratory values over time. Liver and renal function tests should be repeated at least every 12 to 24 h after toxic mushroom ingestion and followed periodically to exclude late-onset hepatotoxicity from cyclopeptide-containing mushroom co-ingestions (*Amanita*, *Galerina*, and *Lepiota* species) and delayed-onset nephrotoxicity from nephrotoxic *A proxima* and *A smithiana* mushrooms. Finally, serum CK should be measured every 12 to 24 h in the first 5 to 10 d and every 36 h for 10 to 14 d in delayed-onset myotoxicity with rhabdomyolysis from *T equestre* or *R subnigricans* ingestion. All patients with any potential for mushroom nephrotoxin–induced acute renal injury should be referred to medical centers equipped and staffed for hemodialysis and kidney transplantation in the event that conservative supportive care measures fail and severe renal failure ensues.

Conclusions

Nephrotoxic mushrooms, most commonly *Cortinarius* species, can cause acute renal damage and kidney failure. Recently, several new species of nephrotoxic mushrooms have been identified, including *A proxima* and *T equestre* in Europe, *A smithiana* in the Pacific Northwest of the

United States and Canada, *A pseudoporphyria* in Japan, and *A punctata* in Korea. In rare cases, consumption of the edible, hallucinogenic mushroom *P cubensis* has caused acute, reversible renal failure. In addition, 2 newly recognized myotoxic mushrooms can cause rhabdomyolysis with massive myoglobin release that indirectly causes acute kidney injury, *T equestre* in Europe and *R subnigricans* in China. The management of nephrotoxic mushroom poisonings often requires renal replacement treatments, with renal transplantation reserved for extracorporeal treatment failures.

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CLINICAL IMAGES

The Boy With the Jellyfish Tattoo and Facial Swelling

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Introduction

The Maltese Islands are a small archipelago of islands at the center of the Mediterranean where seaside activities are popular year-round among locals and tourists alike. We frequently treat injuries caused by sea creatures, jellyfish stings being the most common. Similar reports are seen in countries with similar climates and seascapes.[1,2] We present a case of a child who sustained a marine envenomation caused by an unknown tentacled organism. In such cases, it is often difficult to find the causative organism, and the healthcare practitioner has to rely on the patient's description of events and clinical signs and symptoms.

Case Report

A 4-y-old boy presented to the pediatric emergency department (PED) with significant upper lip swelling, acquired while he was swimming in the sea off the coast of Malta. The boy had been swimming along the rocky shore when he returned to the shore crying after a tentacled creature had stung him. He immediately developed facial swelling. His mother took him to a clinic, where he was given 5 mg promethazine orally and transferred to PED by private vehicle.

On examination at the PED, the boy had a facial urticarial rash and a severely swollen and erythematous upper lip with tentacle marks on his left cheek, chin, and upper lip (Figures 1 and 2). He also had thin, elongated white patches on the tongue consistent with tentacle marks. The patient was crying and anxious but alert and communicative, with no apparent evidence of airway compromise or systemic symptoms. Vital signs were stable on primary assessment: heart rate of 106 beats·min^{-1}, capillary refill time of <2 s, SpO_2 98% on room air, and afebrile; however, we were unable to record blood pressure because the patient was poorly compliant.

In the PED, the patient was given 60 mg intravenous hydrocortisone, a dose of 4 mg·kg^{-1}. He was admitted to a general pediatric ward for observation and further treatment in view of the significant facial and perioral swelling. During the inpatient stay, the patient received regular hydrocortisone (60 mg) and chlorphenamine (2.5 mg), both intravenous 4 times daily. The patient experienced no respiratory distress and remained hemodynamically stable throughout his admission. He was discharged after 48 h of observation, with improvement in facial swelling.

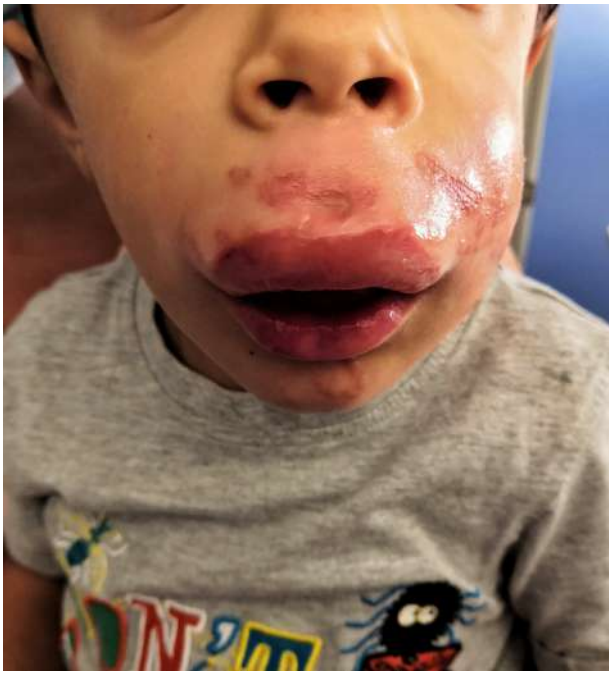

Figure 1. Reaction to jellyfish sting illustrating localized swelling and erythema extending from the upper lip to left cheek and the erythematous tentacle marks.

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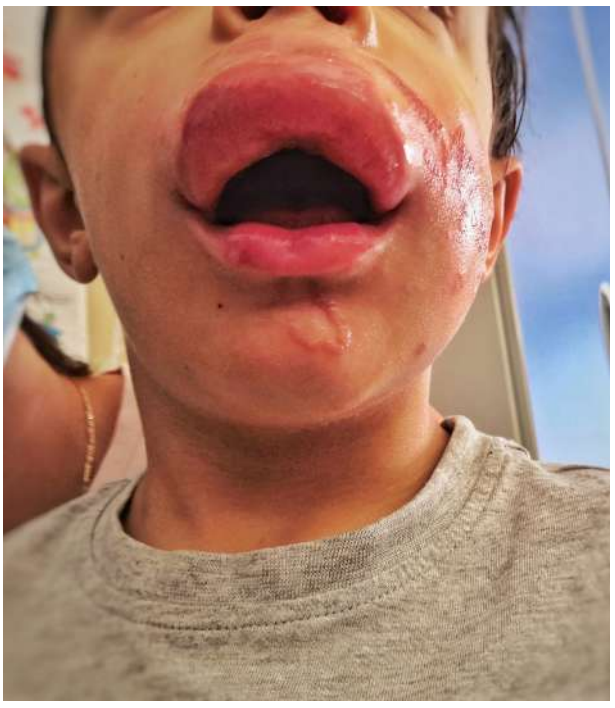

Figure 2. Reaction to jellyfish sting illustrating localized swelling and erythema of the upper lip, particularly when compared to the lower lip. Tentacle marks seen on the chin and left cheek.

Discharge medications were cetirizine 2.5 mg twice daily and prednisolone 30 mg daily for 3 d.

Discussion

Jellyfish are pervasive across many regions and are responsible for a large range of aquatic human envenomations. Environmental changes in sea temperatures, pH, and salinity have resulted in changes in the distribution of jellyfish and lengthening of reproductive periods, resulting in a greater number of invasive species causing greater numbers of human envenomations.

The effects of cnidaria envenomation largely depend on the species responsible; however, they can be associated with clinical features ranging from local pain, vesicular or urticarial eruptions, itching, erythema, edema, and lymphadenopathy to widespread systemic features such as anaphylaxis and cardiac arrest.[3] Cardiovascular collapse can occur secondary to a phenomenon known as Irukandji syndrome, whereby systemic manifestations of muscle spasm, severe sweating, hypotension, and cardiac failure occur 20 to 30 min after envenomation. This is thought to result from systemic catecholamine excess, but the exact mechanism is poorly understood.[3]

Nematocysts stored in cnidocytes found in the tentacles of jellyfish are responsible for the toxic effects seen after contact with venomous species. There can be up to several billion nematocysts,[4] which, upon mechanical stimulation of the tentacle, uncoil and penetrate dermal and epidermal skin layers within a fraction of a second. The nematocyst thread is immersed in a collection of antigenic proteins and enzymes that are responsible for the variety of clinical features after exposure to toxic jellyfish tentacles.[4] Multiple variables, relating to both the victim and the jellyfish, affect the extent of envenomation. The victim's body weight, the site of injury, and surface area of exposed skin, combined with the individual's reactivity to the varying molecules released by a diverse range of nematocysts, determine the response to and extent of injury.[5]

Initial contact is often associated with pain at the time of inoculation. The subsequent reaction may vary, with some individuals showing evidence of delayed type IV hypersensitivity reaction with delayed cutaneous manifestations and histologic features similar to those seen in allergen-mediated contact dermatitis. Anaphylaxis with subsequent shock is an uncommon yet well-described manifestation of jellyfish envenomation in previously sensitized individuals.[5] However, cross reactivity between sea nettle antigens and certain jellyfish such as *Physalia physalis* can result in anaphylactic reactions without previous exposure.[5]

Sea anemones are closely related to jellyfish. Both belong to the phylum Cnidaria, with the most common species of the former in the Mediterranean being *Amaranthus viridis*. Anemones are more prevalent close to rocky shores and are frequently encountered in areas with swimmers. They are sedentary creatures that typically have iridescent yellow-green tentacles with purple tips. This makes anemones particularly attractive to swimmers, especially unsuspecting young children who end up being stung.[2] Similar to jellyfish, anemone tentacles possess nematocysts, which may release venom on contact. Sea anemone venom is typically less potent than jellyfish venom, but anemone tentacles are more numerous and may still cause severe reactions.[2]

Management of both jellyfish and sea anemone stings is largely limited to seaside management and rarely requires hospital admission. The victim should be removed from the water to avoid complications while immersed. Airway, breathing, and circulation should be stabilized if necessary. Vinegar, or 5% acetic acid, should be used liberally for more than 30 s over the injured area. This deactivates the nematocysts, preventing further envenomation. Gentle removal of tentacles can be achieved by plucking remnants off the victim while taking care to minimize manipulation and further nematocyst discharge. The affected areas should be immersed in water of about 45°C to denature active enzymes found in venom. Appropriate analgesia

should be used. Topical or systemic antihistamines and glucocorticoids can be used according to the extent of injury.[6] The use of promethazine as the initial antihistamine may have been a cause for concern because of the risk of respiratory depression, particularly in a patient at risk of respiratory failure.[7]

Perioral cnidarian envenomation in the pediatric population has not been reported frequently. A retrospective review of all children presenting to a PED after jellyfish envenomation between 2010 and 2015 reported only 3 patients sustaining facial envenomations. One of these patients required hospitalization for 3 d for buccal swelling, dyspnea, and trismus.[8] Despite our patient's uncomplicated inpatient course, prudence in such cases is necessary because of the risk of delayed cutaneous eruptions, placing the patient at risk of airway compromise.[3]

In cases similar to ours when the envenomating species is unknown, one must rely on the patient's description. However, the shape and type of skin lesion may provide a hint to the identity of the offending creature. Knowledge of the exact location, time of day, time of year, and depth, as well and the local distribution of dangerous organisms, is also helpful.[1]

Conclusion

Seaside first aid could have mitigated the extent of injury, possibly preventing the need for hospitalization in this case. The use of promethazine at the primary healthcare facility in the setting of perioral edema increased the risk of respiratory depression. This, together with the risk of delayed cutaneous eruption, further emphasizes the need for observation and hospitalization.

Young children tend to be particularly prone to envenomation injuries because they have thinner skin than adults, particularly in sensitive areas such as the face, genitalia, and axillae. Education aimed at providing current information about first aid seaside management might improve treatment at the initial medical contact, possibly mitigating effects after envenomation.

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LESSONS FROM HISTORY

Resuscitation of an Unconscious Victim of Accidental Hypothermia in 1805

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In 1805, W.D., a 16-y-old boy, became hypothermic after he was left alone on a grounded boat in Leith Harbour, near Edinburgh, Scotland. He was brought to his own house and resuscitated with warm blankets, smelling salts, and massage by Dr. George Kellie. W.D. made an uneventful recovery. We discuss the pathophysiology and treatment of accidental hypothermia, contrasting treatment in 1805 with treatment today. W.D. was hypothermic when found by passersby. Although he appeared dead, he was rewarmed with help from Dr. Kellie and his assistants over 200 y ago using simple methods. One concept that has not changed is the critical importance of attempting resuscitation, even if it seems to be futile. Don't give up!

Keywords: emergencies, resuscitation, rewarming, unconsciousness, prehospital

Introduction

On May 20, 1805, Dr. George Kellie, president of the Royal Medical Society and a Fellow of the Royal College of Surgeons (Edinburgh), reported a case of "Torpor from Cold," describing the successful resuscitation from accidental hypothermia of W.D., a 16-y-old boy (see online Supplemental Material).[1] Although the methods of resuscitation from deep accidental hypothermia were less developed in 1805 than today, W.D. made a full recovery. We summarize the case and Dr. Kellie's analysis and compare differences in resuscitation and knowledge of hypothermia between1805 and today.

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Case Summary

On February 26, 1805, W.D., a 16-y-old boy, had been sailing in the vicinity of Leith Harbour, near Edinburgh, with companions, when their boat ran aground just short of the pier at about 1630 (Table 1). The companions managed to get themselves ashore, leaving W.D. alone on the boat, after promising to return shortly. Three hours later, at about 1930, 2 seamen passing by noticed W.D. lying in the stern of the boat. At first, they thought he was asleep, but then they realized that he was cold and apparently lifeless. They transported him to a nearby house and placed him near the fire. Dr. Kellie was called and arrived at about 2000 with his assistants. He described the scene:

> When we arrived, [W.D.] was stretched out, before the fire, on his back, with very little appearance of life; the whole

Table 1. Timeline of patient W.D. on February 26, 1805

~1300	Boat left harbor.
1630	Boat grounded on return.
~1930	W.D. was found unconscious.
2000	Dr. Kellie arrived and examined W.D.: "[R]espiration was obscure and insensible; but the pulse was quite distinct, even at the wrist."
2030	Treatment was started. (At 2330, rewarming had been applied for 3 h.)
2130	Jaw tone was increased.
2145	Breathing was clearly visible; pulse was regular and full (heart rate 100 beats·min^{-1}).
2330	Body temperature seemed normal.
0000	W.D. felt warm, was breathing normally, and had a full bounding pulse.
0300	W.D. awoke, spoke normally, complained of thirst, drank a basin of tea, and fell asleep.
0900	Dr. Kellie returned, just after W.D. had finished his usual breakfast; W.D. complained of thirst and abrasions from the application of smelling salts (HR 90 beats·min^{-1}).

Reported times are in bold. Estimated times are in regular text.

> body, with the exception of the face, which was well coloured, was of a deadly pale appearance, and very cold. The powers of sensation, and of muscular motion, were completely suspended. The head and limbs, perfectly flexible, fell lifeless to the ground, from whatever position they were raised to; the mouth was half open, and the jaw, obedient only to the hand, could be moved upwards and downwards, but returned to the half-closed position; the respiration was obscure and insensible; but the pulse was quite distinct even at the wrist, though irregular and slow. The organs of sense were equally inexcitable; a candle held close to the exposed eyes made no impression, the eye-balls remained fixed and motionless; the pupils, though dilated, contracted irregularly, while yet exposed to the light, in the way I have sometimes observed them to do in the recently dead.

Starting at about 2030, Dr. Kellie and his assistants attempted to stimulate breathing, support the circulation, and rewarm W.D. with vigorous rubbing of the body and extremities and the application of external heat using warm flannels (blankets). Dr. Kellie's assistants, "little satisfied with the slow effects," requested that venesection (bloodletting) be performed to see if W.D. would bleed. About 120 mL (4 ounces) of blood was drained from an arm vein. Following this, ammonium water (smelling salts) was used to stimulate breathing. The first application of smelling salts to the nose caused contraction of the facial muscles but did not stimulate breathing. However, when the assistants poured a small quantity of smelling salts on the chest and upper abdomen and rubbed it in vigorously, W.D.'s breathing increased dramatically. The assistants repeated these procedures at short intervals with the same effects. They continued applying warm blankets and rubbing the body and extremities. Soon, W.D.'s breathing was accompanied by movements of the neck and shoulders. The assistants were then able to stimulate breathing and movement further by applying more smelling salts to the nose. The pulse became more regular and more rapid. At about 2130, an hour after the start of treatment, W.D.'s jaw was more closed and resisted attempts to open the mouth**.** The rescuers thought that he might be able to swallow, so they tried, initially unsuccessfully, to give him fluid. After several attempts with stimulation of the lips and tongue, he managed to swallow a few teaspoons of warm brandy and water. The process was repeated several times.

About 2 h after Dr Kellie had been called, W.D.'s life "seemed secure." Breathing was clearly visible and the pulse was regular and full, at 100 beats·min^{-1}. The face and chest were warm, although the extremities were still cold. The assistants applied warm bricks to W.D.'s feet, covered him with blankets, and continued to massage his limbs. At about 2330, after 3 h of resuscitation, the body temperature seemed normal.

At midnight, when Dr Kellie left, W.D. felt warm and was breathing normally, with a full, bounding pulse. W.D. did not regain consciousness until 0300, when he awoke as if from sleep, spoke to his father, complained of thirst, drank a bowl of tea, and went back to sleep again for a few hours. Dr Kellie returned at 0900, just after W.D. had finished breakfast, which he had eaten enthusiastically. He complained of thirst and about the abrasions from the application of the smelling salts. His pulse was normal at a rate of 90 beats·min^{-1}. He remembered feeling cold, tired, and sleepy but had no further recollection of events.

Discussion

PHYSIOLOGY

Dr. Kellie discussed "caloric" (heat).[1] In the early 19th century, caloric was thought to be a fluid that flows from warmer to cooler bodies. This concept anticipated the laws of thermodynamics, formalized later in the century.

Dr. Kellie concluded that "[O]f the general exciting or stimulant power of heat, there can be no doubt." He dismissed the illusion that cold is "a positive and active energy," correctly stating that cold is just a decrease in temperature: "[F]or any degree of temperature… cold is still heat."

Dr. Kellie observed that the effects of temperature depend on the organism. Some living beings "perish or become torpid from a diminution of temperature, which is yet sufficient for the life and activity of a great many others."

Dr. Kellie stated that changes in temperature could have stimulant or sedative effects: "[A]s the temperature descends, it stimulates less and less." This is an oversimplification. In humans, cooling of the skin stimulates shivering, increasing heat production as much as sixfold compared to resting, nonshivering metabolism.[2-4] If core temperature continues to drop, shivering continues at high intensity until the core temperature decreases to about 32°C. Shivering then begins to diminish, ceasing at a core temperature of about 30°C.[5] Metabolism, minute ventilation, heart rate, blood pressure, and cardiac output increase as core temperature falls to about 32°C, then progressively decrease at lower core temperatures.[6] In contrast to shivering and metabolism, central nervous system function only decreases with cooling. Sedative effects become noticeable below a core temperature of about 33°C, progressing to coma at lower temperatures.[7]

PATHOPHYSIOLOGY

During prolonged exposure to cold, if heat loss exceeds heat gain, core temperature decreases. "Torpor" can mean unresponsiveness, as in the title of the case report. Torpor can also describe the lethargy of animals in hibernation or the inability of poikilothermic animals, such as amphibians or reptiles, to move when they are cold. In animals that hibernate, torpor is controlled and the body is allowed to cool, in contrast to accidental hypothermia as the result of excessive cold exposure. Although people who are unconscious because of hypothermia may die, Dr. Kellie observed that survival is sometimes possible: "[T]he capability of resuscitation is sooner lost in humans [than in animals]; the torpor terminates rapidly in death, though there are not wanting examples of recovery, after long-continued torpor, even in men."

Dr. Kellie explained the progression to torpor:

> In passing into this state, the irritability of the sanguiferous [circulatory] system seems first affected; the pulsations of the heart and arteries are languid and less frequent; the respiration, next affected, is performed more slowly; the internal heat falls below the natural standard; the functions of digestion are suspended; the powers of voluntary motion and of sensation gradually sink, and at length the torpor becomes complete.

This clinical description is mostly accurate, although the declines in neurologic and cognitive function accompany the continued decreases in cardiac output, heart rate, and respiratory rate.

Dr. Kellie thought that rewarming presents a mirror image of cooling: "In tracing the progress of resuscitation, we remark that the functions are revived nearly in the order in which they became torpid; the circulation and respiration first, and, gradually after, the activity of muscular motion, is restored." This is not strictly true. One exception is that during cooling, there is a cold diuresis that may cause or exacerbate hypovolemia.[8] W.D. was likely hypovolemic from decreased food and fluid intake and may also have had diuresis caused by cold. His tachycardia during rewarming may have been exacerbated by hypovolemia.

CENTRAL NERVOUS SYSTEM AND RESPIRATION

Dr. Kellie expressed the opinion that, if torpor were the direct result of cold, it would "in no case take place till, the heat of the brain and nerves was reduced beneath the natural standard." Instead, he thought that torpor must be caused by "changes produced on the blood during the pulmonary circulation. Whatever impedes the respiratory changes of the circulating fluid debilitates or destroys the powers of muscular motion."

Cerebral function starts decreasing at brain temperatures below about 33°C and continues to decrease with further cooling.[7] The electroencephalogram shows only minor changes until core temperature drops below 33.5°C.[9] Clinical manifestations include irritability, confusion, apathy, dysarthria, ataxia, and decreased mental status progressing to coma, usually at a core temperature between 30 and 28°C. These changes are caused by the direct effect of brain tissue cooling. They can generally be reversed with rewarming, except when hypothermia has been associated with asphyxia. Cerebral metabolism decreases by 6% for every decrease in brain temperature of 1°C or about 50% for each decrease of 10°C.[10] Decreased metabolism protects the brain from ischemia, allowing tolerance to prolonged periods of decreased or absent blood flow during hypothermia.

CARDIOVASCULAR SYSTEM

Although decreasing core temperature initially causes tachycardia and increased blood pressure, continued cooling below about 33°C causes pulses to become progressively weak and slow.[11] Based on Dr. Kellie's

examination, W.D. was initially bradycardic with an irregular pulse. The irregularity was likely caused by atrial fibrillation, a common occurrence during cooling.

When Dr. Kellie arrived, W.D.'s body was pale except for the face. Dr. Kellie thought that "[T]he terminations of the sanguiferous system on the surface of the body have their action immediately diminished by the local abstraction of caloric [loss of heat]." Although the circulatory system is a circuit that does not have terminations, Dr. Kellie's understanding was consistent with modern knowledge that hypothermia causes peripheral vasoconstriction with centralization of blood flow.

Dr. Kellie believed that "though the temperature of the heart itself be no way affected its irritability is instantly diminished by abstraction of caloric from the extreme branches of the system." He was incorrect that the temperature of the heart was not affected, but correct that loss of heat from the periphery causes cooling of blood returning to the heart. The word "irritability," as used by Dr. Kellie, refers to the automaticity of the electrical system of the heart. Modern usage is that the heart becomes more irritable, meaning "unstable," with cooling. Dysrhythmias are more likely at lower core temperatures.[12]

As core temperature falls below 33°C, cardiac function decreases. Hypothermia interferes with electrical activity, causing bradycardia and other dysrhythmias, with decreased automaticity of the sinus node, a decrease in transmembrane resting potential, and prolongation of the action potential.[13] As the heart cools to 28°C, the risk of life-threatening dysrhythmias, especially ventricular fibrillation, increases.[14] Ventricular fibrillation arrest can be caused by cooling alone but can also be provoked by movement or rough handling.[15] Cooling can also cause asystole rather than ventricular fibrillation.[12]

RESPIRATORY SYSTEM

Dr. Kellie stated that respiration is "completely suspended" in hibernating amphibians and markedly diminished by cold in "higher orders" of animals. He used the example of people "remaining long torpid, deeply buried under snow" as proof that decreased respiration causes decreased cerebral function. We now know that respiratory rate and tidal volume decrease in response to diminished brainstem activity. Cold is a direct cause of decreased ventilatory drive.[16] Dr. Kellie observed that cold causes a greater decrease in respiration than in cardiac activity. W.D.'s "respiration was obscure and insensible, though the pulse was distinctly perceptible, even at the wrist."

DIAGNOSIS AND STAGING OF HYPOTHERMIA

Dr. Kellie knew that W.D. had been exposed to cold. The differential diagnosis of unconsciousness includes intoxication and injury. Dr. Kellie excluded intoxication by history and by smelling W.D.'s breath. He excluded injury with a thorough physical examination. He concluded that "the symptoms had arisen from the operation of the cold" after hours of exposure to "the diminished temperature of the atmosphere … assisted by moisture" from the rain that had fallen. Dr. Kellie added that W.D. had "suffered from fatigue" and lack of nourishment, both contributing factors to accidental hypothermia.[17]

If there are no coexisting conditions, such as intoxication, injury, exhaustion, or decreased reserves of energy caused by inadequate nutrition, the severity of hypothermia can be estimated by clinical signs using the Wilderness Medical Society system or the Swiss system. In the Wilderness Medical Society system, unconsciousness indicates severe or profound hypothermia with an estimated core temperature below 28°C.[18] In the Swiss system, unconsciousness without apparent death (HT III) correlates with a core temperature of 28 to 24°C.[19] If exhaustion and lack of nourishment contribute to unconsciousness, estimates of core temperature using the Wilderness Medical Society or Swiss system may not be accurate.

TREATMENT

It is likely that W.D.'s wet clothes were removed before Dr. Kellie arrived because Dr. Kellie describes the appearance of the trunk and extremities as well as that of the face. Removal of the wet clothes would have limited heat loss (Table 2). Evaporation from wet clothes increases the rate of heat loss fivefold, compared to dry clothes.[20]

Dr. Kellie's goals of treatment were to provide exogenous heat to warm the tissues and stimulate cardiorespiratory activity. Rewarming with exogenous heat increases core temperature, causing stimulation of ventilation and circulation. W.D. was placed supine near a fire and wrapped in warm blankets. The fire would have provided radiant heat to the side of the body facing the fire, decreasing heat loss from the blankets, and would have also decreased heat loss by warming the room (Table 2). The small heat content of the blankets not facing the fire would have dissipated quickly, much of it to the surrounding air, at least from the outermost blanket, but these blankets would have provided insulation limiting further heat loss. Massage of the extremities had the potential to be harmful by causing peripheral vasodilation of the skin with increased return of cold blood to the heart, lowering core temperature after removal from the cold (afterdrop).

Dr. Kellie advocated rapid rewarming for accidental hypothermia. He contrasted rapid rewarming with

Table 2. Treatment of WD in 1805 with current concepts in 2021

Treatment	*Current concepts*
W.D. evacuated from the scene to a warm environment	Effective first step
Wet clothes removed in a warm environment	Effective at decreasing heat loss; if transport time is short, wet clothes should not be removed until the patient is in a warm environment
W.D. placed near fire	Radiant heat source for parts of body facing the fire; decreases heat loss from warm blankets by providing radiant heat and by warming the room
Warm bricks applied to feet	Appropriate attempt to provide heat, but likely ineffective; no core warming; may cause burns to feet if bricks are too hot
Massage with warm flannels (blankets)	Limited heat transfer; risk of tissue damage if tissue frozen (not applicable in this case); risk of ventricular fibrillation because of movement, if core temperature <28°C
Warm blankets	Appropriate attempt to provide heat, but relatively ineffective; low heat content, but would provide insulation, protecting against further heat loss
Smelling salts (ammonia)	Only useful to evaluate unresponsiveness; likely no effect on hypothermia
Teaspoons of brandy	Alcohol is generally contraindicated because it can produce vasodilation, leading to increased heat loss; in this case, the amount of alcohol was likely too small to cause significant vasodilation

gradual rewarming of a frostbitten limb that was thought to be necessary to prevent "a violent reaction" or injury. He observed that in spite of the means employed to rewarm W.D., "the temperature was very slowly restored, the surface and limbs long felt as cold as ever." The slow pace of rewarming is not surprising. The amount of active heating was likely less than that provided by modern methods of active warming, such as forced-air warming and chemical, electric, or water-filled heating pads. Regardless of the amount of heat donated by the fire and warm blankets, transfer of heat to the core would have been limited, at least initially, because of vasoconstriction. The pallor and cold skin of the body, except for the face, suggests peripheral vasoconstriction.

Dr. Kellie and his assistants used venesection (bloodletting) diagnostically rather than therapeutically. Dr. Kellie wrote, "As to blood-letting, I regard the practice as useless, while the body is cold, and the circulation and respiration languid." The drainage of a small amount of blood is unlikely to have had any appreciable effect on resuscitation. Bloodletting, to see if W.D. would bleed, was superfluous in the presence of vital signs, even though they were diminished.

W.D.'s response to inhaled smelling salts was to grimace. This suggests that he was not deeply comatose. Fatigue and nutritional depletion may have contributed to the decreased level of consciousness. Inhaled smelling salts may stimulate ventilation.[21] Irritation of the chest and epigastrium when Dr. Kellie applied ammonium water by "rubbing it forcibly in" with his hand seemed also to stimulate W.D.'s breathing. This maneuver was similar to a sternal rub, a noxious stimulus in which the examiner's knuckles are rubbed against the sternum. A sternal rub is very uncomfortable for a conscious patient. Topical application of ammonium water to the chest and upper abdomen likely had no direct effect on ventilation. It is likely that forcible rubbing acted as a general stimulant to increase the level of consciousness, stimulating respiration.

Shortly afterward, rubbing ammonium water in the epigastric area seemed to cause not only "the usual full inspirations, but a writhing motion of the neck and shoulders, which could now also be reproduced by [smelling salts] applied to the nose." The pulse became stronger, more regular, and more rapid. W.D. seemed more alert, although he still had a decreased level of consciousness.

Eventually, W.D. was able to swallow a few teaspoons of warm brandy and water. Although giving fluids or food to a patient with a decreased level of consciousness risks aspiration, the amounts that Dr. Kellie administered were likely quite small. Stimulation of the lips and tongue may have allowed reflex swallowing.

Modern methods of external rewarming include chemical or electrical heat packs and heated forced air. Unlike warm bricks, forced-air warming and heat packs provide significant heat to the chest, the back, and the axillae, where heat can be effectively transferred to the core.[18] Massage with warm blankets was likely of little benefit. The only evidence-based medical use of smelling salts is to place them near the nose to see if a patient is feigning unconsciousness.[21] The composition of modern smelling salts can vary, but they still usually consist of ammonium carbonate dissolved in water. It is very difficult for a conscious patient to suppress a reaction to this noxious stimulus. The fact that WD winced when ammonia was placed near his nose most

likely indicated that he was not deeply comatose. The practice of venesection has survived but is now mostly limited to withdrawing small samples of blood for laboratory analysis, a purpose that is still diagnostic rather than therapeutic. Administration of small amounts of warm brandy and water were unlikely to have been helpful or harmful. Current practice is to administer isotonic fluids, usually intravenously.

Conclusions

W.D. was hypothermic when found by passersby. Although he appeared dead, he was rewarmed by Dr. Kellie and his assistants over 200 y ago, using simple methods. Some of the methods, such as vigorous rubbing, use of smelling salts, and administration of brandy, are obsolete. Other methods have survived as fundamental elements in the treatment of accidental hypothermia. These include keeping the victim horizontal, gentle handling, removal from the cold environment, providing insulation, and rewarming. The methods of rewarming included heat from a fire, warm blankets, and applying warm bricks to the feet. Only radiant heat from a fire may have been as effective as modern methods, such as heat packs and forced air warming.

Some of Dr. Kellie's understanding of pathophysiology and thermodynamics has long since been disproved, but many of his observations were surprisingly accurate. One concept that has not changed is the critical importance of attempting resuscitation, even if it seems to be futile. Don't give up!

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Supplemental Material(s)

Supplementary material associated with this article can be found in the online version at https://doi.org/10.1016/j.wem.2021.08.007.

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Letters to the Editor

Regarding "Median Cut-Off Membrane Can Be a New Treatment Tool in *Amanita phalloides* Poisoning"

To the Editor:

We found the description of 2 cases of severe *Amanita phalloides* (AP) toxicity interesting and are grateful that physicians continue to strive to improve care for such patients.[1] However, we are concerned that the description of success attributed to hemodialysis (HD) with a medium cut-off membrane may prompt unnecessary intervention or transfer of patients to receive HD based on very low quality and incomplete evidence of benefit. Broad supportive care measures are the mainstay of treatment for AP toxicity, including intravenous fluid administration and maintenance of normal electrolyte concentrations. Studies of other treatments directed specifically at AP toxicity, such as N-acetylcysteine and silibinin, yielded mixed results.[2-5]

Similarly, studies of HD in patients with AP toxicity report conflicting results and are limited by confounding treatments, uncontrolled observations, and lack of data to demonstrate amatoxin clearance.[6,7] In the current study, HD was performed for renal replacement and thus served a supportive care role for the patient.

The authors' conclusion that the use of an HD membrane with amatoxin filtering possibly reduced toxicity is unsupported in their study because no data demonstrating amatoxin removal are reported. In fact, prior studies reported little if any circulating amatoxin at the time patients can reasonably be started on HD.[6,8] Future studies aiming to demonstrate a benefit of HD in AP toxicity must measure some form of amatoxin clearance. Without direct evidence of removal of toxin, HD seems to provide useful but ultimately only supportive care.

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In Reply to Dr Connors et al

To the Editor:

We are very grateful to read the comments from Connors et al[1] on our article.[2] Amatoxin poisoning is a medical emergency characterized by hepatoxicity, encephalopathy, coma, and death. The main amatoxins are α, β, and γ amanitins. They inhibit eukaryotic RNA polymerase II, causing transcriptional arrest and affecting metabolically highly active cells such as hepatocytes and kidney cells. Amanita toxin has a molecular weight of 373 to 990 Da, is water soluble with 0.3 $L \cdot kg^{-1}$ *Vd*, is 0.3% protein binding, and has 2.7 to 6.2 $mL \cdot min^{-1} \cdot kg^{-1}$ endogenous clearance.[1]

Although oral decontamination with activated charcoal, intravenous hydration, N-acetyl cysteine, silibinin, and penicillin are recommended in the standard treatment approach, these treatments are often insufficient because symptoms occur after a relatively long and obscure incubation period. Extracorporeal treatments (eg, conventional hemodialysis [HD], continuous renal replacement therapies [CRRT], plasmapheresis, hemoperfusion [HP], extracorporeal albumin dialysis) can be used to remove toxic compounds from the body for supportive treatment in clinical emergency situations when specific treatment is insufficient.[3,4] The lack of well-designed studies on the optimal method of extracorporeal removal of toxic compounds, thus staying at a lower level of evidence, is a major problem.

The more expeditiously toxins are removed, the less chance major toxicity will ensue[5]; thus, extracorporeal treatment may be an efficacious alternative treatment modality. In general, clearance of dialysable substances is lower with CRRT than with conventional HD[6] owing to slower flow rates, and although HD is readily available in most hospitals, many are not capable of delivering CRRT, HP, plasmapheresis, or extracorporeal albumin dialysis. One of the problems with toxin removal by dialyzers and HP devices is that their effects on pharmacokinetic parameters other than simple clearance measurements are largely unknown. This problem can also be experienced in newly developed dialyzers (mid-cutoff, high-cutoff). However, new studies can be instructive.

Owing to the urgent nature of amatoxin poisoning treatment and uncertainties in resolving potential complications, we may never be able to find well-designed, evidence-based studies to guide us. In dealing with these controversial issues, we must continue to use less than ideal evidence and our own experience to guide our decision-making.

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More or Less? Wilderness Education in Emergency Medicine Residencies

To the Editor:

In "A National Survey of Wilderness Medicine Curricula in United States Emergency Medicine

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Residencies," the authors state that 63% of emergency medicine (EM) residencies that responded to their survey had some form of wilderness medicine (WM) training as part of their residency curriculum.[1] Yet, the data they put forth are less than convincing that this is an accurate assessment. Using data from a survey in which only 57% (136 of 240) of EM programs responded is an unreliable surrogate for what the reality of national WM education entails. The authors did address this as a limitation, stating that if the 104 nonresponding programs were assumed not to offer any WM education, then only 35% of programs would. Although we applaud their self-awareness relating to this flaw of the study, 35% compared to 63% is a massive difference that changes the conversation from *most* to *few* programs teaching WM.

It is unclear from the article whether the authors were attempting to determine WM education *within* or *outside* the educational recommendations set forth by the Core Content Task Force II. In 2016, that group published an updated Model of the Clinical Practice of Emergency Medicine, which outlines the topics necessary for a complete resident education.[2] (Of note, a more recent model was published in 2019, but this would not have been available to the study authors at the time of their survey.[3]) In that model, topics such as *high altitude illness* and *decompression syndrome* are listed under "environmental disorders," but they easily qualify as WM content.[4] With this in mind, every program should be teaching WM to a degree. Any program stating that it does not may be unaware that it does or may have interpreted the opening survey question ("Does your program teach WM?") as meaning teaching WM other than what is listed in the core content. Depending on the authors' true intent, this could lead to either a huge underreporting or overreporting of WM education.

It should also be noted that a similar study was undertaken only 3 y earlier, which in itself makes us question the utility of this updated study given that only a short time has passed. The authors in that study, however, attempted to parse the difference between *required* and *optional* WM education activities within EM residencies.[5] The most recent study did not, and that failure adds to the confusion surrounding the data and interpretation of the results. For example, at our institution we offer our residents additional training in tactical and combat medicine, but we would never say that we teach tactical or combat medicine as part of our residency curriculum.

The authors also chose not to include or analyze data related to the geographic location of the responding institutions. In the presence of a 56% response rate, performing a subanalysis of the geographic locations of respondents would go a long way to support the data being a true representation of the national WM curricula. Seeing as 144 of the 240 EM residencies at the time of the survey were located in the Northeast, Southeast, or Midwest, if all 136 responders were from these regions, it would obviously not be a true national representation.[6] In this hypothetical scenario, the entire West, Southwest, and Northwest would have been absent.

Although a regular assessment of EM residency WM education is useful, this particular study does little to expand our insight. We openly acknowledge that getting high survey return rates is very difficult and respect the authors' attempts while acknowledging their own study's limitations. Unfortunately, the low return rates and vagueness of the study's intent leaves us with more questions than answers. In the future, however, the lessons learned from this study can potentially aid the subspecialty in truly assessing WM educational offerings within EM residencies.

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We thank Dr Hill for his letter to the editor[1] regarding our recent publication, "A National Survey of Wilderness Medicine Curricula in the United States Emergency Medicine Residencies"[2] and appreciate the opportunity to comment.

Indeed, a study evaluating wilderness medicine (WM) education was published in 2015,[3] 3 y prior to our data collection. We reference that paper here not to draw it into any critique, but as a foundation for our work. Although the 2015 study does not specify the timing of actual data collection, the listing of 187 residencies used was based on a Society for Academic Emergency Medicine directory accessed in March of 2014. In the interim, emergency medicine residency program numbers grew by almost 30%. We believe this rapid growth in residencies, rather than a specific timeframe, should be the indicator for reappraisal.

Although we too would have preferred a higher response rate, our response rate of 57% is comparable to that of the 2015 paper (56%). No analysis of non-responding programs was given in that study, so we cannot compare these points. Similarly, neither paper specifically defined WM educational content, and the model of the clinical practice of emergency medicine has not changed significantly between the 2013 and 2016 versions.[4,5] Although indeed "every program should be teaching WM to a degree,"[1] both papers reported that programs indicated that they did not teach any WM, so the interpretation of the terminology by respondents may have been similarly limited. As such, although our approaches did differ, we believe that our paper can be viewed as complementary to the 2015 study.

Lastly, we concur in our paper that categorizing programs by geography could have yielded additional insights, and we encourage the inclusion of these data in future studies.

We appreciate Dr Hill's statement that our study poses "more questions than answers" as an opportunity for further inquiry and look forward to contributing to the effort to cement excellent WM education in our residencies on a national level.

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A Case of Splenic Injury Caused by a Blow from a Dolphin's Tail Fin

To the Editor:

The common bottlenose dolphin, *Tursiops truncatus*, has a global distribution. The mammal weighs an average of 150 to 200 kg and reaches 2 to 4 m in length.[1] Dolphins are characterized as high-speed swimmers, propelled by a powerful horizontal lunate tail fin consisting of 2 flukes, which play an important role in their swimming.[2] They can leap in the air or stand out of the water using this tail fin. They are also intelligent creatures and able to learn tricks, perform in shows, or interact with humans after appropriate training.[3] We herein report a case of splenic injury induced by a blow from a dolphin's tail fin.

A 12-y-old boy in a wetsuit was travelling through the water while holding the dorsal fin of a trained bottlenose dolphin in an aquarium. He had no remarkable personal or family medical history and was a tourist from Saitama, located near Tokyo. When he erroneously released the dorsal fin, he sustained a sharp blow to the left flank by the dolphin's tail fin. He acutely felt severe pain, but he returned to his hotel with his parents. He then experienced a generalized tonic clonic seizure of approximately 10 s in duration at 3 h after the injury. He vomited 5 times within 7 h after the seizure. These symptoms did not recur throughout the night or the following day. However, the flank pain did not improve. His parents called an ambulance, and he was transported to our hospital by a

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Table 1. Spleen organ injury scale—2018 revision[4]

AAST Grade	*Imaging criteria (computed tomography findings)*
I	Subcapsular hematoma <10% surface area Parenchymal laceration <1 cm depth Capsular tear
II	Subcapsular hematoma 10–50% surface area; intraparenchymal hematoma <5 cm Parenchymal laceration 1–3 cm
III	Subcapsular hematoma >50% surface area; ruptured subcapsular or intraparenchymal hematoma ≥5 cm Parenchymal laceration >3 cm depth
IV	Any injury in the presence of a splenic vascular injury or active bleeding confined within splenic capsule Parenchymal laceration involving segmental or hilar vessels producing >25% devascularization
V	Any injury in the presence of splenic vascular injury with active bleeding extending beyond the spleen into the peritoneum Shattered spleen

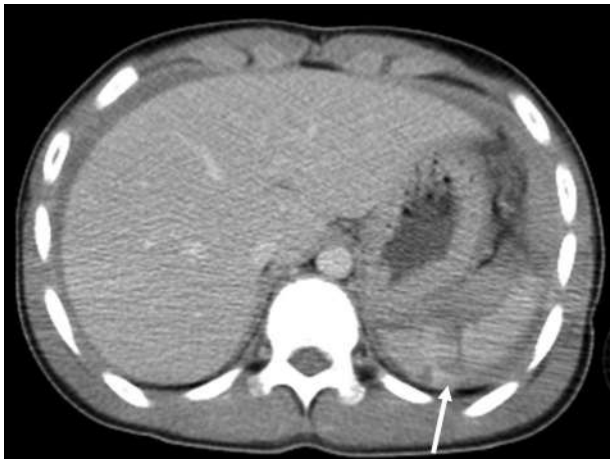

Figure 1. Findings of enhanced computed tomography on arrival. Computed tomography showed a grade of III (see arrow) according to the American Association for the Surgery of Trauma splenic injury scale.

physician-staffed helicopter. Helicopter transportation was arranged owing to the distance needed to travel to arrive at the hospital; ground ambulance would take more than 90 min, versus 15 min by helicopter.

On arrival, his level of alertness was Glasgow Coma Scale score of 14 (E3V5M6) with a blood pressure of 129/70 (normal range: 110–124/70–79) mm Hg, a heart rate of 109 (55–85) beats·min^{-1}, a respiratory rate of 30 (12–18) breaths·min^{-1}, and a percutaneous oxygen saturation of 97% on room air. The physical examination findings included only localized rebound tenderness in the right flank without skin lesions, contralateral to the side of the initial injury. The main results of a blood analysis were as follows: white blood cell count, 15,800·μL^{-1} (normal 4000–10,700·μL^{-1}); hemoglobin, 10.5 g·dL^{-1} (normal 12.7–15.7 g·dL^{-1}); platelets, 31.0×10^4 μL^{-1} (normal $18–44 \times 10^4$ μL^{-1}); aspartate aminotransferase, 29 IU·L^{-1} (normal 15–31 IU·L^{-1}); alanine aminotransferase, 10 IU·L^{-1} (normal 9–32 IU·L^{-1}); blood urea nitrogen, 21.1 mg·dL^{-1} (normal 6.8–19.2 mg·dL^{-1}); and creatinine, 0.63 mg·dL^{-1} (normal 0.39–0.62 mg·dL^{-1}). An abdominal ultrasound revealed hyperechoic areas at Morrison's pouch, around the spleen, and at the rectovesical pouch. Enhanced computed tomography showed a grade III splenic laceration, according to the American Association for the Surgery of Trauma splenic injury scale (Table 1),[4] with no findings of extravasation or pseudoaneurysm (Figure 1), as well as fluid retention of the small intestine in the right abdomen, probably due to the subileus state after splenic injury. He was diagnosed as having an isolated splenic injury with a large amount of hemorrhaging in the intra-abdominal space, and he underwent angiography for embolization. However, the radiologists were unable to insert a catheter into the splenic artery, and the patient was admitted under observational status.

On Day 2, the patient's hemoglobin level dropped to 8.0 g·dL^{-1}, and he received a transfusion of 2 units of red blood cells for fatigue. The patient tolerated the transfusion well. On Day 3, his hemoglobin level increased to 9.0 g·dL^{-1}, and he began to eat and was initiated on an iron agent and acetaminophen. Subsequent contrast-enhanced computed tomography on Days 4 and 7 revealed no significant changes, and the posttransfusion course was uneventful. An electroencephalogram was normal, and he was free from seizures during hospitalization, without the need for an anticonvulsant. The precise mechanism was unclear, but the cause of the convulsions might have been transient hypotension or some cranial damage. After the final computed tomography examination, his bedrest was finished. His hemoglobin level returned to 11.2 g·dL^{-1}. On Day 12, he was transported to a local hospital in Saitama for evaluation of late-presenting complications after splenic trauma, with a plan to return to school.

The patient in the present case underwent angiography for embolization of the splenic artery even though he had stable vital signs. The American Pediatric Surgical Association guidelines state that embolization should be performed only in patients with evidence of ongoing hemorrhaging.[5] However, the results of a study comparing prophylactic splenic arterial embolization (pSAE) with surveillance and then embolization only if

necessary (SURV) in a randomized clinical trial revealed significantly fewer splenic pseudoaneurysms and a shorter duration of hospitalization for adult patients in the pSAE group than for those in the SURV group.[6] Accordingly, pSAE was performed in the present case.

Several newspaper articles have reported human injuries induced by dolphins.[7-9] Dolphins can injure or kill humans by biting, attacking, hitting with their tails or snouts, or drowning by dragging humans underwater. During the literature review, the authors found a news report involving an encounter between 2 intoxicated tourists and a dolphin. The tourists grabbed the dolphin, rode it like a horse, and even attempted to insert an ice cream stick into its blowhole. The dolphin attacked the tourists. One of the tourists died as a result of internal organ damage sustained during the attack, and the second tourist sustained a broken rib and injuries to the head caused by blows from the animal's tail fin.[9] Such potential risks based on historical accounts of human–dolphin interactions are described in a previous paper.[10]

The present case was indeed an accident. However, dolphins can be dangerous animals, and caution may be necessary when humans interact with these creatures. There are many regulations protecting dolphins. We could find no regulations, guidelines, or protective measures in place to ensure the safety of humans when interacting with dolphins, suggesting no such measures have yet been established.[10] In addition, there were few medical reports concerning dolphin-related injury. A further analysis with the accumulation of reports concerning dolphin-related injury will be necessary; such findings will function as the basis for establishing relevant regulations.

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Medics Need the Wilderness as Much as It Needs Medics: A Letter in Reply to "Can't See the Wood for the Trees"

To the Editor:

I write in reply to the fascinating letter by Robert Cussen published in this journal[1] extolling the unique place of woods and trees within medicine and the ways in which we interpret this phenomenon. Although I enjoyed the whole piece, I found myself drawn particularly to his final statement. As a medical student revising for examinations myself, the assertion that we would be better off returning to the trees (or else losing ourselves in the wilderness in general) is one that resonates deeply.

The benefits of this reimmersion are multiple. Although the superiority of outdoor exercise over equivalent indoor exertion is disputed,[2] exercise is certainly beneficial to mood, health, and focus[3] and is something that is implicitly linked to the very act of finding oneself in a wilderness environment, far from easy comforts and motorized transportation. Furthermore, research into the effects of nature itself on our ability to focus profound directed attention, and on our mood, highlights a link between the range of soft-hard fascination[4] evoked within these wild environments

necessary (SURV) in a randomized clinical trial revealed significantly fewer splenic pseudoaneurysms and a shorter duration of hospitalization for adult patients in the pSAE group than for those in the SURV group.[6] Accordingly, pSAE was performed in the present case.

Several newspaper articles have reported human injuries induced by dolphins.[7-9] Dolphins can injure or kill humans by biting, attacking, hitting with their tails or snouts, or drowning by dragging humans underwater. During the literature review, the authors found a news report involving an encounter between 2 intoxicated tourists and a dolphin. The tourists grabbed the dolphin, rode it like a horse, and even attempted to insert an ice cream stick into its blowhole. The dolphin attacked the tourists. One of the tourists died as a result of internal organ damage sustained during the attack, and the second tourist sustained a broken rib and injuries to the head caused by blows from the animal's tail fin.[9] Such potential risks based on historical accounts of human–dolphin interactions are described in a previous paper.[10]

The present case was indeed an accident. However, dolphins can be dangerous animals, and caution may be necessary when humans interact with these creatures. There are many regulations protecting dolphins. We could find no regulations, guidelines, or protective measures in place to ensure the safety of humans when interacting with dolphins, suggesting no such measures have yet been established.[10] In addition, there were few medical reports concerning dolphin-related injury. A further analysis with the accumulation of reports concerning dolphin-related injury will be necessary; such findings will function as the basis for establishing relevant regulations.

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and the maintenance/restoration[5] of the faculties we must possess to fulfill our roles as good students and good doctors. This research echoes Dr Cussen's choice to mention "forest bathing," a term that pleasantly evokes the mentally healing potential of such experiences.

Going even further, it is certainly possible to draw parallels between journeys into wilderness environments (where the conventions and rubric of normal life are defied by challenging surroundings) and entering liminal spaces that anthropologists suggest is a defining feature in the structure of rituals.[6] It is not an unthinkable stretch to characterize such an expedition as an example of a 3-part rite of passage.[7] These acts pluck someone away from their ordinary life and take them through a period of transformative liminality until they are returned, with altered status, to the life they were leading before. Consider overworked medical students assembling their hiking and wild-camping gear, throwing themselves into a period of solitude in nature, stripping back the mental and physical burdens that come from long hours at a laptop screen, and emerging lightened and more resilient to re-enter their studies.

The prescribed schematic rites necessary to pursue hiking, mountaineering, camping, sailing, and climbing as pastimes are used by many as a way of allowing the space and time required to change their mental state. This could be hypothesized to act as a direct tonic to the stresses and strains of professional life, with ~60% of those who participate in outdoor exercise in the United States being college-educated.[8] Although clearly not a tool that is uniquely limited to those working within medicine, the relevance of this kind of contextualizing and grounding practice to the self-reflections we are required to perform in medical education in the United Kingdom[9] is immediately obvious. This seems especially evident in the process of engaging with past experiences from different perspectives and reframing them,[9] something that the addition of physical and contextual distance can only aid. Indeed, we see iterations of these same ideas accessed by many through the lens of mindfulness,[10] which is increasingly taught in medical schools[11] and has scope for helping practitioners in areas including coming to terms with failure, adapting to changing roles, or preparing to embark on a new professional venture entirely.

Speaking from personal experience, I have found nothing to be quite as useful in helping to adapt to the demands of my medical degree as spending time in mountains, forests, and tents. These experiences are challenging, cathartic, and ultimately an expression of kindness that is sorely needed in the avoidance of burnout.

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Book Review

Ward, Milledge & West's High Altitude Medicine and Physiology, Sixth Edition
Andrew M. Luks, Philip N. Ainslie, Justin S. Lawley, Robert C. Roach, Tatum S. Simonson
Boca Raton, FL: CRC Press, 2021.
US $200, 544 pages, hardcover or digital

Ward, Milledge & West's High Altitude Medicine and Physiology, sixth edition, is the latest edition of this venerable, but hardly stodgy, book. As with the second through the fifth editions, it continues to improve. The book has come of age with the addition to the title of Michael Ward, James Milledge, and John West, authors of the first 3 editions. Milledge and West continued to the fifth edition and have now passed the baton to new, younger authors. Andrew Luks, the lead author of the sixth edition, joined as an author in the fifth edition. The other authors are all new to the book. Like Dr Luks, all of them are well-respected experts in the subject.

The style is less sparse than in previous editions, but still clear and consistent. The authors have rewritten most of the material, rather than just revising the chapters, and have added a wealth of new material. New material includes updates throughout the book, based on new studies published since the fifth edition in 2013, and expansion of many areas, such as genetics and geography. The number of pages and the type size are about the same as in the fifth edition. The format of the sixth edition is larger to accommodate the additional material.

The book is now divided into 3 sections: The Environment and Its People, Physiologic Responses to Hypoxia, and Clinical High Altitude Medicine. The chapters have been reorganized and rearranged. The 27 chapters, decreased from 29 in the fifth edition, are divided into many more subsections, making it easier to find information on specific topics. Another welcome change is that the references are now at the end of each chapter rather than at the back of the book. Every chapter is thoroughly referenced.

Although any new textbook is already out of date by the time it is printed, this edition is as up to date as possible, with references as recent as 2020. The authors have once again anticipated future trends, pointing the way forward to help readers remain current between editions. Examples include genomic studies of high altitude adaptation and susceptibility to altitude illness, which continue to proliferate, and altitude preacclimatization. New studies of preacclimatization are likely after recently reported ascents of Mount Everest with climbers flying directly to base camp after arriving in Nepal. The authors mention that future editions are likely to include significant new information in these areas as well as others.

My worst, but still minor, disappointment is that the iconic frontispiece photo of the original authors on skis during the 1960 Silver Hut expedition that graced the first 3 editions is still missing from the sixth edition.

Ward, Milledge & West's High Altitude Medicine and Physiology continues to be the foremost reference work on its subjects. It was a great pleasure to review the latest edition. I congratulate the authors on freshening and expanding the text. I recommend it to both experts and neophytes with clinical or research interests in high altitude. Before you put it on your bookshelf, preferably in easy reach so that you can refer to it often, you will probably want to read some, or even all, of the chapters.

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