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EDITOR'S NOTE

The Nature of the Editorial

Editorials are, by definition, opinions on topical issues. Journal articles sometimes need them, but often not. Informative titles help readers decide if abstracts are worth reading, and informative abstracts do the same for the full piece. Well-developed articles may stand firmly on their own, delivering appropriate content with clarity and balance.

A recent comment pointed out that the editorials in this journal espouse standards that are not always met by the articles within the pages. That is true, and for good reason. Editorials are often aspirational or educational. They can promote best practice even when it is hard to achieve.

Many who read or submit manuscripts to this and other medical journals are not primarily trained in research or research communication. The peer review and revision process can play an important role in helping authors refine their skills.

There are issues that threaten the integrity of peer review. Several have been discussed in this space in recent years, many related, directly or indirectly, to the rise of predatory journals that will publish anything for a price. Societally, we need to promote critical thinking and reading skills both to aid in the intake and assessment of information and to demand high levels of credibility from sources. The structure of scientific writing can help readers navigate. It is easier to see what is missing when expectations exist that can rise above enthusiasm or bombast.

We need to be more active in training our authors, reviewers, and editors on best practice. Bias can never be eliminated, but it can be controlled. The limitations section of reports is one of the best places to see if authors have objectively developed their work. This will be evident in what is included, what is lacking, and how interpretations and conclusions are tempered. The limitations text should not be viewed as a fundamental weakness in a scientific report, but as a fundamental strength.

Peer review should start with no assumptions about the propriety of a manuscript being accepted, revised, or rejected. Author and institution names are given more weight than they deserve by some. It is really only the content of a report that matters. There is a necessary reliance on the good faith efforts of researchers and authors to deliver valid content, but outcome decisions must be based on a careful evaluation of what is presented in every section and the logic, clarity, and temperance in crafting and interpretations.

There are ongoing debates over the strengths of fully unblinded, author blinded, reviewer blinded, and fully blinded peer review. There are arguments for and against each that will not be resolved here. The critical point for me is that every review should be written as though it is unblinded. As social media has made clear, anonymity can breed a lack of civility that does not aid in promoting thoughtful exchange. Relentless diplomacy should be the fundamental goal, with a healthy dose of forgiveness for those who suffer from inevitable lapses.

Scientific thought advances as experience and understanding evolve. Insight and opinion influence manuscripts and the reviews of them. The plan is to get things right, but it is also important to appreciate that incorrect reports can still play an important role if they drive others to look further into questions. The problem to avoid is publishing reports that are invalid at the point of inception. Open communication helps to make this possible.

Credible peer reviewed journals have both power and responsibility in developing and protecting the literature record. Sensitivity to concerns is often aided by simple conversations. Editorials are one of the tools used to encourage them.

Neal W. Pollock, PhD
Editor-in-Chief

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REVIEWER THANK YOU

2021 *Wilderness & Environmental Medicine* Peer Reviewers

The editors express their sincere appreciation to the following individuals who completed peer reviews of articles submitted to *Wilderness & Environmental Medicine* in 2021. WEM serves an important role in bringing literature to the scientific community. Our peer reviewers play an essential role in ensuring the merit and quality of the manuscripts we publish. Many of these individuals reviewed multiple papers, and some also serve on the editorial board and maintain section editor or associate editor duties.

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A TRIBUTE

Blair Dillard Erb, MD: 1930-2021

"There are strange things done in the midnight sun by the men who moil for gold." When I hear those words, I see Blair Erb standing at the presidential podium at a Wilderness Medical Society (WMS) meeting with a copy of Robert Service's poetry in his hand. Blair was the epitome of a Renaissance man who always brought a sense of decorum and class to any function. Along with being an astute clinical cardiologist, he was renowned for his storytelling and great love of the outdoors.

Blair was born in the hills of Virginia but moved to the flatlands of Memphis, Tennessee, where he was raised along with his older brother. He learned to love the outdoors at an early age and achieved the rank of Eagle Scout, no mean feat during the 1940s. As another sign of an outstanding work ethic and intellect, Blair completed undergraduate and medical school in 5 years at The University of Tennessee. The Blue Ridge Mountains must have been in his blood, however, for he developed a great love for the "high places," and as an Air Force flight surgeon stationed in Colorado, he was smitten by the mountains.

He was also smitten around this time by Sally Smith, to whom he was happily married for 45 years. Sally and Blair had 3 children, who accompanied them on many camping, climbing, and other adventures. I had the pleasure of making Sally's acquaintance on many occasions, and she was the perfect match for Blair.

Blair's accomplishments in the field of medicine over 30-plus years were legend. His expertise in exercise physiology, cardiology, and cardiac rehabilitation made him an international authority and led him to the WMS, where he became a long-time board member and president. He never lost his love for the mountains, and he combined his medical expertise with high altitude and mountaineering.

I remember Blair not so much for his medical knowledge but for his love of the liberal arts. He could expatiate on the history of the national parks, the Civil War, types of aircraft, the poetry of Longfellow, or the essays of Emerson. He could tell stories that would make you laugh out loud or cry inside. Most importantly, he did everything with Class, with a capital "C." I learned a great deal from Blair, and I can see why his patients loved him. He showed me not just how to do things right, but how to do the right things in the correct manner. Having dinner with Blair and Sally was always the high point of a meeting, and I will sorely miss him.

Edward J. (Mel) Otten, MD

Blair Dillard Erb, MD, in 2015. Photo courtesy of Mary Lou Layton.

Gracious and humble, Blair was the consummate Southern gentleman/physician. I will never forget his assistance in obtaining independent accreditation to sponsor our own continuing medical education events. We partnered with the University of California San Diego for our first couple of WMS meetings, and they served as our sponsor for continuing medical education accreditation. The WMS was growing, and even picking up some

international members, so we knew it was time to pursue our own accreditation status. The paperwork was mind-boggling and a formidable hurdle for the founders, who were already juggling busy work schedules. Blair had just gotten involved with the WMS, and I discussed with him the challenge of working through the accreditation process. Without hesitation, he jumped right in to assist, saying, "I know the hell out of these organizations. Let me help." Indeed he did, and we got our accreditation. That was Blair—a wonderful combination of experience, enthusiasm, and willingness to roll up his sleeves and get the job done.

Edward Geehr, MD

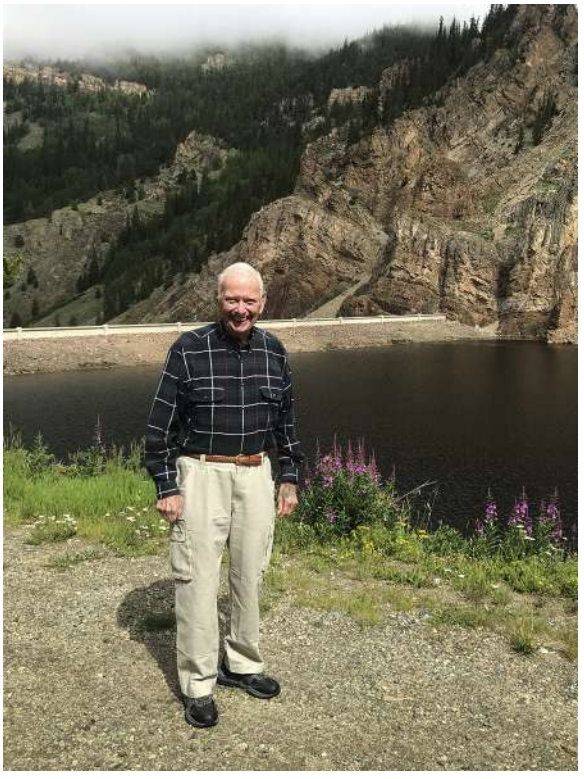
Though I never got to work with Blair during his time on the WMS board or during his presidency, I became acquainted with him as an elegant presence at society functions. He had the voice and embodiment of a stately uncle or grandfather; when Blair showed up, everyone stood up straight and checked their diction and politeness. WMS meetings are informal (and some have been known to take that to the extreme), but even the most disheveled among us took care to comb our hair and tuck in our shirttails when Dr. Erb was nearby. Blair was a traditional Southern gentleman; the first female WMS president threw him a curveball when he wanted to continue the "green jacket" tradition. We chuckled over how it was going to work for me and future presidents who didn't fit into, for example, a 42L suit coat. I found a suitable lady's jacket and he mailed me the custom-made WMS buttons to sew on to the jacket. (It was ugly, but it made him smile to see all of us in our green jackets at the next meeting.) His grace and elegance will be missed.

Luanne Freer, MD

There will always be a critical moment in the development of a new organization as it transitions from its founders into a mature program. Blair Erb, MD, will be recalled by the many who knew him, both by the WMS founders and those members who followed, as a true Southern gentleman. He had the ability to calmly lead this fledgling organization into its maturity through, at times, turbulent waters.

Under his guidance, the WMS expanded its scope of membership, integrated multiple competing wilderness medicine programs into cooperation, oversaw attempts at standardization of curricula, and championed the editing of comprehensive practice guidelines.

He established the evening annual dinner and elevated its stature into the grace of a semiformal affair, resplendent with an increasing number of awards honoring members' contributions; initiated green jackets for past presidents; established the first international wilderness medicine conference as one of our annual meetings every 4 years; and planned a triptych of paintings memorializing wilderness medicine themes to be displayed at the annual meetings—all efforts to imbue the WMS with both social respect and meaningful purpose.

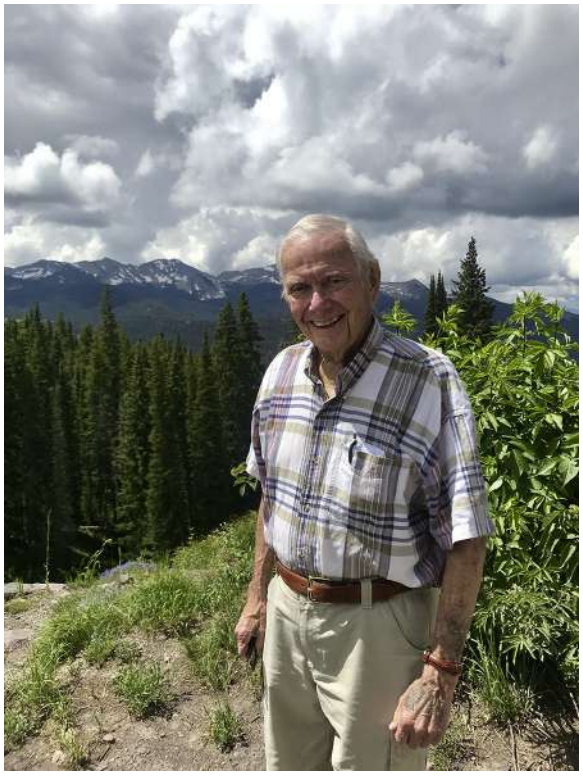


Near Crested Butte, Colorado (July 2019). Photo courtesy of Mary Lou Layton.

He was my mentor and my friend. My membership in the WMS has given me great privileges, among which it has introduced me to many heroes. Blair Erb ranks at the top of that list.

William W. Forgey, MD

Blair Erb was an early and constant supporter of the WMS, serving as its fourth president. In remembrances among the other past presidents, the description used repeatedly is "Southern gentleman." He was a lifelong son of the South, but the term "gentleman" is no longer in frequent use and warrants explanation. Blair was attentive, compassionate, and had a calm and thoughtful demeanor. He did not have any hint of prejudice or arrogance. Although he had a certain formality, as evidenced by the purchase (at his own expense) of a green sports coat for all the WMS presidents, he was not stiff or inflexible, and he had a good sense of humor. In the early days of the WMS, he added his experience with other medical societies and a vision of a society based on a



Near Crested Butte, Colorado (July 2019). Photo courtesy of Mary Lou Layton.

common passion as well as medical science. While president, he fully endorsed and developed the first World Congress on Wilderness Medicine in partnership with other organizations. Blair was able to add a historical perspective to wilderness medicine and provide depth with his specialty of cardiology and keen interest in exercise physiology and cardiac rehabilitation. For example, I learned from Blair that for the purpose of predicting cardiac risk for wilderness activities, participants could be divided into 5 categories, from demonstrated high performance to significant limitations from chronic illness, but the ones who were most concerning were those who were formerly athletic but now deconditioned with risk factors, because they overestimated their ability. Blair Erb's love of the outdoors and wild places as well as medicine was palpable. He imparted that to the WMS, and we will miss both his enthusiasm and his gentleman's demeanor.

Howard Backer, MD, MPH

As many have reminisced, Blair Erb was a Southern gentleman, but he was so much more. Although he was from Tennessee, he never tired of reciting a poem by Robert Service, "The Cremation of Sam McGee." Sam McGee was also from Tennessee, but the similarity may have ended there. Sam was always cold in the Yukon, a condition that only resolved when he died, presumably of hypothermia, on the day after Christmas on the Dawson Trail. Sam McGee was cremated, at his own last request, several days later. Blair often referred to Sam's cremation as a case of therapeutic excess.

As president of the WMS, Blair came to Alaska to speak at a wilderness medicine conference organized by Peter Hackett. Blair suggested that we start an Alaska chapter of the WMS. Peter and I started the chapter and had an initial meeting before we found out that chapters were not mentioned in the WMS bylaws. Also not mentioned in the bylaws was the fact that there was no way to amend the bylaws.

I came to Blair with the idea that the WMS should start a library of photographs. In those days, before PowerPoint, photographs for lectures were on slides, so I suggested a slide bank from which members of the WMS could obtain photographs that they might not have themselves for use in lectures. Blair thought for a moment and suggested instead that the WMS could develop educational slide sets, so that a speaker could give an entire prepared talk using the slide set. Although he hardly knew me, he appointed me to run the project. That was the start of the WMS educational presentations.

There was always more to Blair than met the eye. He was 1 of the 4 American members of the Austrian Society for Mountain and High Altitude Medicine (ÖGAHM). He may have joined partly to support his friend Franz Berghold, the president of the ÖGAHM. The other 3 members were Blair's 2 sons and me.

Blair was always a welcome presence at WMS meetings, usually accompanied by his wife, Sally. He encouraged younger members like me to contribute to the society. He was instrumental in making the WMS what it is today and he did it in a typically understated style. In his later years, he willingly took on an assignment to speak about elderly people in the wilderness at a WMS meeting. The first slide, he said, illustrated his own condition. He had seen it in the airport on his way to the meeting, a sign that said "Terminal." He found a way to stop by WMS meetings for many years, even after he was no longer active in the society. I will always remember Blair's genial and inspiring presence.

Ken Zafren, MD

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ORIGINAL RESEARCH

Arterial Blood Gas and Rotational Thromboelastometry Parameters in Healthy Rescuers Incidentally Exposed to Nitroglycerin, Nitrogen Compounds, and Combustion Products

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Introduction—Acute exposure to nitrogen compounds combined with a massive inhalation of air pollutants can influence respiratory and cardiovascular symptoms and coagulation abnormalities in accidentally exposed healthy adults during cave detonation operations.

Methods—Italian alpine and cave rescuers widened a cave in the Abisso Luca Kralj in Trieste, Italy. Volunteers inside the cave were accidentally exposed to the fumes from an uncontrolled detonation of blasting gelatin microcharges. We performed a retrospective cohort study on the clinical data, arterial blood gas analysis, and rotational thromboelastometry parameters from the rescuers involved in the accident.

Results—Ninety-three healthy rescuers were involved in the uncontrolled detonation: 47 volunteers handled a mixture of nitrogen compounds (blaster group), and 46 volunteers did not (nonblaster group). After the accident, statistically significant differences ($P<0.05$) in arterial blood gas values were observed between the groups, with a pattern of mild respiratory acidosis with hypercapnia in the nonblaster group and severe mixed acid-base disorder with hypoxia and hypercapnia in the blaster group. Mild hyperfibrinolysis was observed in 44 volunteers in the blaster group, as were associated bleeding symptoms in 34 volunteers; no significant coagulation modifications were recorded in the nonblaster group.

Conclusions—Respiratory acidosis with hypoxia, hypercapnia, a compensatory metabolic response, and mild hyperfibrinolysis were probably related to the combined effect of nitrogen compounds and the inhaled toxic products of detonation. Therefore, each element exerts a determinant effect on promoting the biological toxicity of the others.

Keywords: cave rescue, air pollutants, pulmonary gas exchange, coagulation, acid-base disorders, hyperfibrinolysis

Introduction

The cave widening group (CWG), a part of the Italian alpine and cave rescue (IACR), uses blasting gelatin (a mixture of nitroglycerin, nitrogen compounds, and chalk) to enlarge narrow portions of the caves, facilitating patient evacuation if an accident occurs. The physicians on the medical commission of the IACR must be present at procedures. An evaluation of volunteer vital signs and parameters (heart rate [HR], arterial blood pressure, and oxygen saturation measured with pulse oximetry [S_pO_2]), arterial blood gas exchange function, and coagulation function is required before starting an enlargement procedure, in accordance with our protocol.

During cave widening, the CWG volunteers handle explosive materials that can permeate the human body by inhalation or skin absorption, despite the use of personal protective equipment. Thus, the medical commission

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equips each volunteer with a large, full-face respirator with eye protection against dust, organic vapors, odors, fumes, sawdust, and asbestos, as well as full-body suits and gloves to protect against chemicals and gases. In addition, detonation gases remain in the air because of the narrow space in the cave, despite the use of portable air purifiers and toxic gas detectors during enlargement procedures. Thus, IACR volunteers may be subjected to prolonged inhalation of combustion products.

Detonating microcharges lead to the release of various combustion products, particularly carbon monoxide (CO), resulting in concentration-dependent symptoms ranging from headache, tachycardia, dyspnea, and seizures to death. The binding affinity of CO for hemoglobin (Hb) is greater than that of oxygen (O_2), resulting in the formation of carboxyhemoglobin (COHb). This alteration decreases the oxygen-carrying capacity of blood and impairs the release of O_2 from Hb. During microcharge detonation, high concentrations of carbon dioxide (CO_2) can also be released into the air, leading to tachypnea, tachycardia, fatigue, nausea and vomiting, syncope, convulsions, coma, and death. Nitric oxide (NO) can also be present. High doses of inhaled NO (>100 parts per million [ppm]) appear to promote lung injury, potentially owing to concurrent nitric dioxide (NO_2) formation. The toxicologic effects of high-dose NO_2 inhalation in humans are related to airway reactivity with diffuse inflammation that presents clinically as dyspnea, followed by pulmonary edema and death.[1]

Vasodilatation is the primary consequence of nitroglycerin and nitrogen compound toxicity, and it results in hypotension with tachycardia, headache, dyspnea, coma, and death. Methemoglobinemia is a consequence of nitrate overdose,[2] and clinical manifestations can include grayish pigmentation of the skin, the presence of chocolate-colored blood, central cyanosis, dizziness, headache, anxiety, dyspnea, symptoms of low cardiac output, somnolence, seizures, reduced consciousness, respiratory depression, shock, and death.[2]

Other toxic effects from nitrogen compounds caused by nitrous oxide exposure have been shown to be associated with neurologic symptoms including headache, dizziness, euphoria due to hypoxia, vitamin B_{12} deficiency, and megaloblastic anemia. In addition to regulating vascular tone, nitric oxide inhibits platelet adherence to endothelial cells and aggregation.[3]

The Abisso Luca Kralj cave is a sinkhole in the dolomite rocks of the karst near Trieste, Italy. Airborne dust produced from the explosion of dolomite rocks contains high concentrations of calcium ions, magnesium ions, and hydroxyl ions that increase the pH of the biofilm on the exposed surface of the cornea and the oral and airway mucosa, resulting in ophthalmic damage, airway and skin irritation, and ulcerative lesions of the mouth and esophagus.

We investigated whether nitroglycerin and nitrogen compounds influence coagulation changes in response to hypoxia[4] resulting from the toxic effects of the combustion products from blasting gelatin. Subsequently, data from the widening of the Abisso Luca Kralj and its effects on the rescuers were investigated.

Methods

This retrospective cohort study analyzed clinical data from CWG rescuers from IACR who were accidentally exposed to fumes from an uncontrolled detonation of blasting gelatin microcharges during the widening of the cave Abisso Luca Kralj.

This study was approved by the cave ethics committee of the IACR (ClinicalTrials.gov identifier: NCT04201860). It adhered to the STROBE statement and the Declaration of Helsinki for ethical principles for medical research involving human subjects. Written informed consent was obtained from all rescuers.

Before starting the operations for cave enlargement, the rescuers were divided into 10 teams, 9 teams with 9 volunteers (5 explosive specialists and 4 speleologists) and 1 team with 12 volunteers (2 explosive specialists and 10 speleologists) at each point of microcharge placement. The speleologists assisted the explosive specialists during the progression through the cave. The volunteers did not have any exposure to nitroglycerin, nitrogen compounds, or combustion products in the 6 mo before the study.

Forty-seven healthy volunteers handled, manufactured, and blasted the explosive microcharges (blaster group, BG), and 46 healthy volunteers did not have contact with the explosives (nonblaster group, NBG). Three hundred microcharges were packed and blasted. Each rescuer in the BG packed 6 microcharges and handled blasting gelatin for an average of 85 min. The composition of the blasting gelatin was 50% nitroglycerin, 41% diglycerin tetranitrate, 8% nitrocellulose, and 1% chalk.

All rescuers were equipped with a large, full-face respirator with eye protection (3M full facepiece reusable respirator 6000 series, Milan, Italy) and wore full-body suits (DuPont C3122T TN Tychem 5000 coveralls, Wilmington, DE) and gloves (Chemstop 30 cm [12 in] blue cut-resistant, impact-resistant PVC-coated gloves, Superior Glove, Ontario, Canada) that protected them from chemicals and gases. An air purification system was activated; it consisted of a series of portable air purifiers (Trotec, TAC 750 E, Trotec International GmbH & C. Sas, Verona, Italy) with filters for airborne dust, fumes,

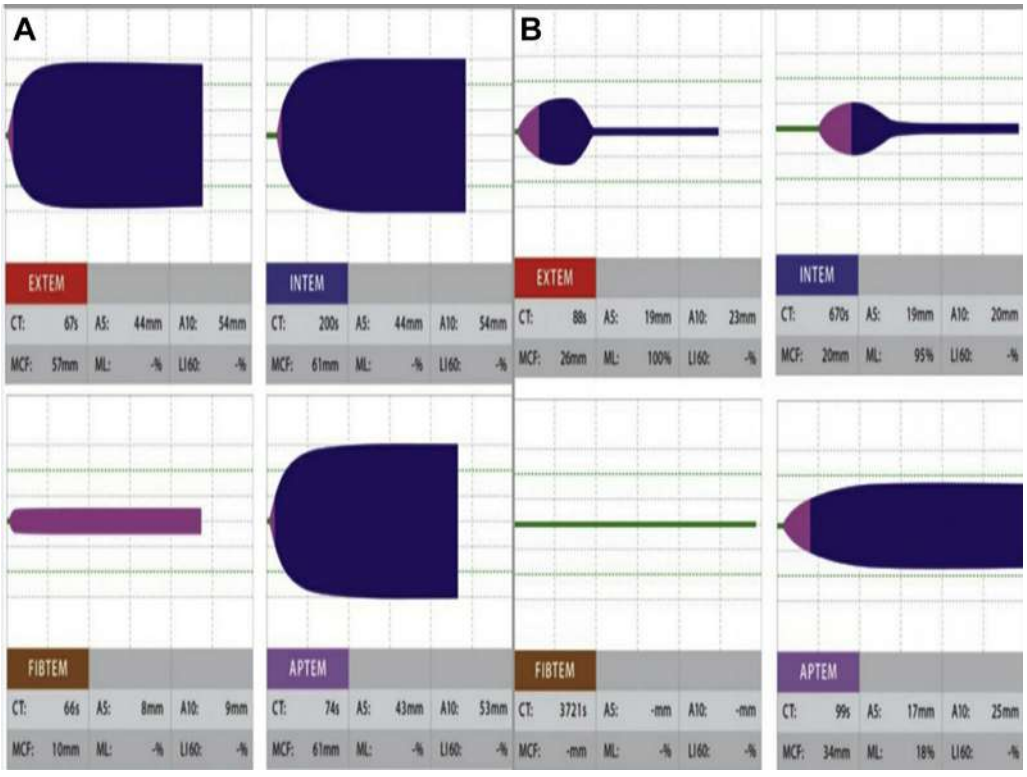

Figure 1. ROTEM variables. A, The results are presented in a normal graphical plot. B, The results indicate hyperfibrinolysis and reduced clot firmness.[12]

and vapors. One volunteer in each team used a handheld gas detector (MineARC, Aura-PT 4 gas detector, MineARC Systems, Perth, Australia) to monitor the gas levels within their immediate surroundings continuously. The handheld gas detectors were able to monitor the thresholds of O_2, CO, CO_2, and NO_2 gases instantaneously and provided visual and vibration alerts. The allowed thresholds of toxic gases were CO <10 ppm, CO_2 <800 ppm, and NO_2 <5 ppm.[5]

An advanced medical presidium (AMP) was placed outside the cave. The AMP is a light and pneumatic tent-type structure from which a voluntary staff of doctors and

Table 1. Characteristics of rescuers

	Groups		P *value*
	NBG (n=46) *n (%) or mean±SD*	*BG (n=47)* *n (%) or mean±SD*	
Age, y	37±9	40±10	0.887
Sex			
Female	10 (11)	5 (22)	0.169
Male	36 (89)	42 (78)	
Smoking			
No	35 (74)	30 (65)	0.331
Yes	12 (26)	16 (35)	
BMI ($kg{\cdot}m^2$)	23.8±2.7	24.9±3.3	0.099
HR ($beats{\cdot}min^{-1}$)	85±17	92±12	0.014
DP (mm Hg)	74±6	70±7	0.013
SP (mm Hg)	128±12	122±13	0.027
S_pO_2 (%)	95±19	98±1	0.193

BMI, body mass index; DP, diastolic pressure; HR, heart rate; SP, systolic pressure; S_pO_2, oxygen saturation.

Table 2. Parameters investigated at 2 follow-up times within the groups

Parameters	*NBG (n=46) Follow-up (mean±SD)*		*BG (n=47) Follow-up (mean±SD)*	
	T0	*T1*	*T0*	*T1*
pH	7.39±0.03	7.32±0.05	7.39±0.02	7.25±0.06
PO_2 (mm Hg)	106±19	75±9	96±13	65±5
PCO_2 (mm Hg)	37±4	50±7	38±4	58±5
HCO_3- (mmol·L^{-1})	24±3	22±2	23±2	20±1
BE (mmol·L^{-1})	1±2	−6±2	0±1	−11±4
Na (mmol·L^{-1})	138±4	137±4	140±4	140±4
K (mmol·L^{-1})	4.3±0.4	4.3±0.4	4.2±0.5	4.2±0.5
$_iCa$ (mg·dL^{-1})	4.5±0.3	4.7±0.3	4.8±0.4	4.2±0.3
Hct (%)	45±6	45±6	45±4	45±4
S_pO_2 (%)	95±19	92±3	98±1	87±2
eT (°C)	36.7±0.2	37.6±0.2	37.0±0.3	37.6±0.3
Hb (g·dL^{-1})	15±1	15±1	15±1	14±1
Glu (mg·dL^{-1})	97±12	95±9	77±7	73±8

BE, base excess; eT, epitympanic temperature; Glu, glucose; Hb, hemoglobin; HCO_3^-, bicarbonate; Hct, hematocrit; $_iCa$, ionized calcium; K, potassium; Na, sodium; PCO_2, partial pressure of carbon dioxide; pH, arterial blood; PO_2, partial pressure of oxygen; S_pO_2, oxygen saturation measured by pulse oximetry.

nurses operates.[6] The medical rescue team consisted of 2 anesthesiologists and 4 nurses with training in prehospital trauma life support and advanced cardiac life support protocols.[7] The anesthesiologists were also trained in the extended focused assessment with sonography for trauma (e-FAST) protocol. They performed the e-FAST assessment using double head convex (3.5-5.0 MHz) plus linear (7.5-10.0 MHz) color Doppler wireless transducers (wireless double head ATL linear/convex color Doppler echography probe, ATL s.r.l., Milan, Italy) connected to a tablet. The e-FAST protocol was performed to obtain scans of the abdominal, pleural, and pericardial space and bilateral lungs to detect free intraperitoneal, intrathoracic, and pericardial fluid and pneumothorax and to exclude the possibility of organ injuries. During the anterolateral chest scan, the number of B-lines was summed for a qualitative evaluation of lung congestion; up to 15 B-lines per single intercostal space was considered evocative for pulmonary congestion or edema.[8] The images from the e-FAST examination were captured and stored in the tablet.

The following data were collected before the rescuers entered the cave (T0): age, sex, body mass index (BMI), smoking status, HR, arterial blood pressure (diastolic pressure [DP] and systolic pressure [SP]), S_pO_2, epitympanic temperature (eT), arterial blood gas analysis (ABGA), and hemostatic measurements (HMs). The vital signs were recorded using a multiparametric monitor (ARGUS PRO LifeCare2, SCHILLER AG, Baar, Switzerland), and eT was measured using an epitympanic probe (Braun ThermoScan PRO 6000 ear thermometer, Kronberg im Taunus, Germany).

The following blood gas tests were performed using an iSTAT handheld blood analyzer (iSTAT handheld; Abbott Point of Care, Inc.; Princeton, NJ) with CG8+ cartridges with arterial reference ranges: sodium (Na 138−146 mmol·L^{-1}), potassium (K 3.5-4.9 mmol·L^{-1}), ionized calcium ($_iCa$ 4.5−5.3 mg·dL^{-1}), glucose (Glu 70−105 mg·dL^{-1}), hematocrit (Hct 38−51%), hemoglobin (Hb 12−17 g·dL^{-1}), pH (7.35−7.45), partial pressure of carbon dioxide (PCO_2 35−45 mm Hg), partial pressure of oxygen (PO_2 80−105 mm Hg), concentration of hydrogen carbonate (HCO_3 22−26 mmol·L^{-1}), base excess (BE [-2]- [+3] mmol·L^{-1}), and arterial oxygen saturation (SaO_2 95−98%).[9,10] A venous blood sample was obtained by venipuncture using a 21-gauge needle. Blood was collected in vacuum tubes (BD vacutainerVR citrate tubes 3.2%), and the first 3 mL of blood was always discarded.

The HMs were evaluated using thromboelastometry (ROTEM delta analyzer, Tem International GmbH, München, Germany). Rotational thromboelastometry (ROTEM) is easy to use as a point-of-care assay in an emergency setting; it produces rapid graphical and numerical results for hemostatic status and is able to detect and quantify the cause of coagulopathy.[11] ROTEM measurements were performed within 30 min after blood sample collection and at least 15 min after venipuncture. The device temperature was set to 37°C, and the maximum runtime was 120 min.

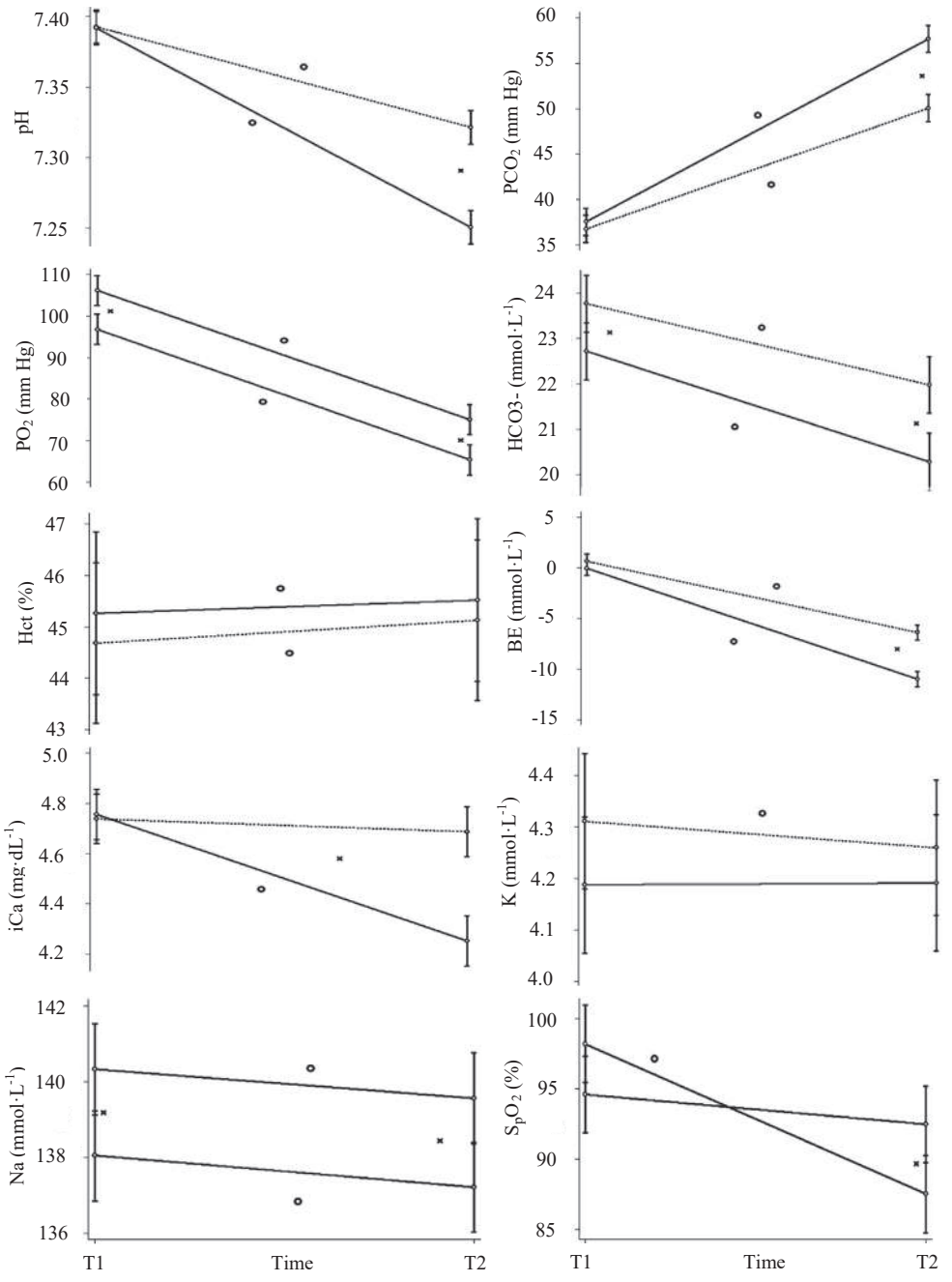

Figure 2. Changes in blood gas parameters within the 2 groups over time (predicted mean with 95% confidence interval). **o** and **x** indicate significant differences within and between groups, respectively, at $P<0.05$. The solid line indicates the blaster group (BG), and the dashed line indicates the nonblaster group (NBG). pH, arterial blood pH; PO_2, partial pressure of oxygen; PCO_2, partial pressure of carbon dioxide; HCO_3^-, bicarbonate; BE, base excess; Na, sodium; K, potassium; $_iCa$, ionized calcium; Hct, hematocrit; S_pO_2, arterial oxygen saturation measured by pulse oximetry.

The ROTEM delta analysis was performed in parallel on the 4 channels using the following reagents: EXTEM to evaluate the extrinsic coagulation pathway, INTEM for the intrinsic coagulation cascade, FIBTEM to evaluate the fibrinogen concentration, and APTEM for the detection of hyperfibrinolysis. These data resulted in a graphical plot (TEMogram) of clot firmness over time. Figure 1 shows normal and abnormal TEMograms.[12]

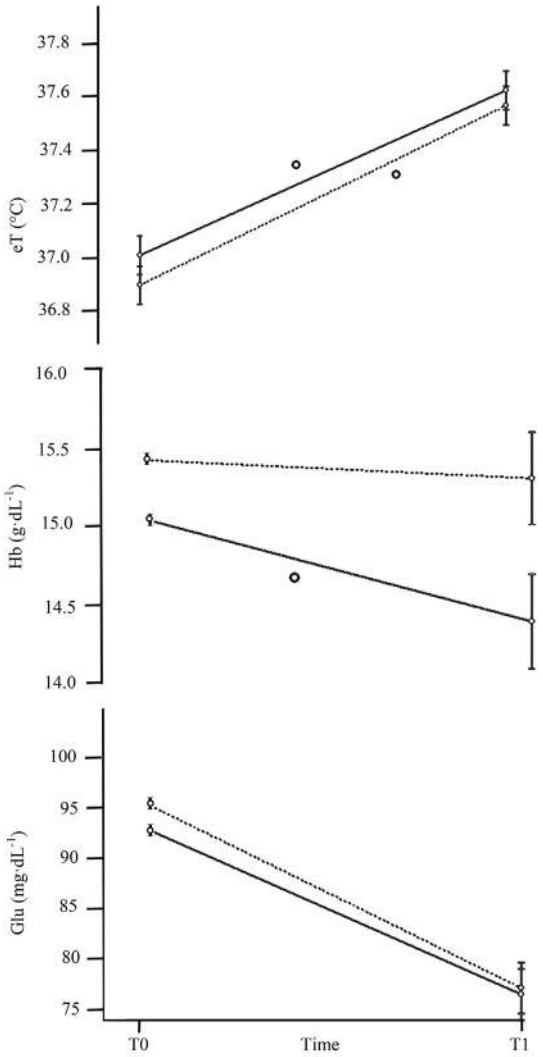

Figure 3. Changes over time for epitympanic temperature, glucose, and hemoglobin within the 2 groups (predicted mean with 95% confidence interval). **o** and **x** indicate significant differences within and between groups, respectively, at $P<0.05$. The solid line indicates the blaster group (BG), and the dashed line indicates the nonblaster group (NBG). eT, epitympanic temperature; Hb, hemoglobin; Glu, glucose.

The following parameters were considered in the present study: clotting time (CT) in seconds and amplitudes of clot firmness as measured in millimeters at 5 (A5), 10 (A10), and 20 (A20) min after the start of clot formation, including maximum clot firmness.

Under aseptic conditions, the radial artery was punctured, and 1 mL of blood was subjected to ABGA. When gas exchange disorders were detected using the ROTEM delta analyzer, the volunteer was forbidden to go into the cave.

The iSTAT and ROTEM delta analyzers were placed in the AMP outside of the cave. Each rescuer recorded the duration of blasting gelatin handling using a chronometer that was started at the first moment each volunteer handled the explosive. The detonation of microcharges at each point was remote-controlled, and the volunteers were in a safe place 50 m from the detonation area. Ten detonation sites were placed, with 30 microcharges at each site. One detonation was allowed at a time, and after each detonation, the purification devices were set to clean the air inside the cave until the gas detectors recorded a safe breathing level.[5]

After uncontrolled detonation, each volunteer exited the cave as soon as possible and immediately reached the AMP (T1); the following parameters were recorded: HR, DP, SP, S_pO_2, eT, ABGA, and HMs. Symptoms related to the toxic gas levels were also recorded. The e-FAST assessment was performed.

STATISTICAL ANALYSIS

Descriptive statistics were calculated for all the variables in the study and reported as the mean±SD or frequency, depending on the scale level and distribution. The χ^2 test or Fisher's exact test was used to analyze categorical variables. Continuous variables were tested for normality with the Shapiro-Wilk test and analyzed using the independent-samples t test to compare means or the Wilcoxon rank-sum test when adequate. A $P<0.05$ was considered statistically significant. Repeated-measures analysis of variance with the group (BG versus NBG) as the between-subjects factor and time as the within-subjects factor was used to assess the presence of significant differences in the blood parameters investigated between the groups. The statistical analysis was performed using STATA 14 software.[13]

Results

Ninety-three volunteers went inside the cave. Table 1 shows the baseline characteristics recorded before they entered the cave; significant differences in the HR, DP, and SP between the groups were reported.

During the enlargement procedure, the remote controller for the detonation malfunctioned, resulting in the uncontrolled blasting of all the microcharges simultaneously and a subsequent blackout of the cave electric network.

The purification devices stopped working, and acceptable toxic gas levels were likely exceeded within a few minutes. The atmosphere of the cave was saturated by fumes and vapors from the uncontrolled detonation. The thresholds of the detected toxic gases were impossible to obtain because all rescuers had to escape from the cave rapidly without concern for their protective equipment, which resulted in serious damage. In addition, they

lost the handheld gas detectors because of the dangerous environment of the cave; thus, the volunteers from the 2 groups were exposed to toxic levels of combustion products in the narrow portions of the cave. All volunteers reached the AMP outside the cave after an average of 110 min.

After the accident, significant differences were confirmed in the following parameters between the NBG and BG: HR (134±14 beats·min^{-1} vs 119±13 beats·min^{-1}, P<0.001), DP (92±10 mm Hg vs 85±9 mm Hg, P=0.003), and SP (171±15 mm Hg vs 124±24 mm Hg, P<0.001).

Table 2 summarizes the results of the ABGA at 2 follow-up visits within the groups. The changes in each blood gas parameter over time and the differences between the 2 groups investigated using the repeated measures analysis of variance model are shown in Figures 2 and 3.

As reported in Figure 2, no statistically significant differences in the pH, PCO_2, HCO_3-, BE, Hct, $_i$Ca, K, and S_pO_2 values were observed between the rescuers in the BG and NBG (P>0.05) before entering the cave, whereas statistically significant differences in PO_2, HCO_3-, and Na values were noted (P<0.05). After the accident, statistically significant differences (P<0.05) were observed in the following blood gas values within the groups: pH, PO_2, PCO_2, HCO_3-, BE, $_i$Ca, Na, and S_pO_2. Figure 2 shows the differences in the ABGA within the groups. The explosive specialists exhibited a more significant loss of bicarbonate, and hypocalcemia was recorded (Figure 2). No differences in eT and Glu were observed at T0 between the BG and NBG; Hb levels decreased over time, with a significant difference between the 2 groups observed at T1 (P<0.05), as reported in Figure 3.

No respiratory disturbances were reported in either group before entering the cave. After the uncontrolled detonation, respiratory symptoms such as cough, tachypnea, and respiratory distress were noted in 41 volunteers in the BG and 6 volunteers in the NBG, with a statistically significant difference between the 2 groups (P<0.05). Rescuers in the BG with respiratory symptoms had diffuse wheezing on chest evaluation.

AN e-FAST assessment of the volunteers in both groups was performed, but no free intraperitoneal, intrathoracic, or pericardial fluid, pneumothorax, or organ injuries were noted. Bleeding from the nose and gums and blood-stained oral secretions were present in 34 volunteers in the BG and 12 volunteers in the NBG (P<0.05).

All volunteers had a normal TEMogram at T0. After the accident, the following HMs were recorded on average in 44 volunteers in the BG: 270±10 s for CT (normal range: 100−240), 33±15 mm for maximum clot firmness (normal range: 50−72), alpha angle of 51±5°, A5 of 20±4 mm, A10 of 27±8 mm, and A20 of 34±13 mm. Complete clot lysis was reported after 60 min. In contrast, no significant TEMogram modifications were recorded in the NBG at T1.

In AMP, peripheral venous access (16 G) was obtained in 12 rescuers from the BG with tachypnea, wheezing, respiratory distress with hypoxia and hypercapnia, and TEMogram abnormalities. They received an intravenous injection of a total of 500 mL of crystalloid solution and noninvasive ventilatory support with oxygen via a Venturi mask (12 L·min^{-1}). Two hundred micrograms of nebulized salbutamol and 250 mg of intravenous hydrocortisone were also administered to these rescuers.[14] They were rapidly hospitalized.

Discussion

In this study, we investigated the short-term changes in arterial blood gas and ROTEM parameters in healthy rescuers who handled nitroglycerin and nitrogen compounds and were accidentally exposed to blasting gelatin fumes in an austere environment.

Potential confounders, such as age, sex, smoking status, and BMI, had no significant influence on the models. Before the rescuers entered the cave, significant differences in their HR, DP, and SP were likely due to the BG rescuers transporting the tools for microcharge packing without being exposed to the explosives before data collection.

Little scientific evidence is available to support the hypothesis that acid-base disorders in healthy adults are related to exposure to nitroglycerin[15] and the nitrogen compounds in blasting gelatin. Nitrogen compounds in blast form at airborne concentrations up to lethal inhalation levels (eg, 3000 ppm) have minimal skin penetration ability, but skin absorption and reservoir effects have been described.[16] Potential secondary inhalation should be avoided by removing the outer layer of protective clothing. Decontamination and movement into an area of enhanced ventilation are also advised.[16] In the austere environment of the cave, with poor ventilation, decontamination of the full-body suits was not possible. In addition, after uncontrolled detonation, the rescuers seriously damaged their protective equipment. Thus, during microcharge packing or detonation, molecules of nitroglycerin and nitrogen compounds remained on the suits and then were probably inhaled or remained on the skin of the explosive specialists during their escape from the cave, penetrating into the body.

Nitroglycerin reduces pulmonary artery pressure[17] because it is metabolized to NO, which is a potent vascular smooth muscle relaxant in vascular endothelial

cells.[18] Inhaled nitroglycerin and NO easily cross the alveolar-capillary barrier and stimulate soluble guanylate cyclase in the smooth muscle of the microcirculatory network near the alveoli,[19] producing pulmonary vasodilation with a reduction in pulmonary artery pressure, pulmonary vascular resistance, and right ventricular afterload while avoiding systemic hypotension.

Therefore, nitroglycerin and nitrogen compounds produce vasodilation with a reduction in pulmonary vascular resistance and an increase in blood flow to these areas, whereas acute exposure to toxic gases may rapidly induce inflammatory responses, oxidative injury, and the death of bronchial epithelial cells before ultimately impairing the respiratory membrane of the lungs.[20] Thus, after uncontrolled detonation, we speculate that the respiratory epithelium of the rescuers in the BG was more permeable to inhaled toxic molecules from the combustion pollutants, with a subsequent increase in absorption from the pulmonary to systemic circulation. Consequently, nitroglycerin and nitrogen compounds were not directly responsible for respiratory acidosis, but they enhanced the toxicity of the other gaseous pollutants. Under this condition, pulmonary gas exchange function was impaired, resulting in severe respiratory acidosis with hypoxia and hypercapnia and a compensatory metabolic response. This biological mechanism might also explain the differences in HR, SP, and DP between the groups at the 2 follow-up time points. After the accident, the increased absorption of air pollutants may also have influenced the systemic vasculature. In effect, the short-term inhalation of air particulate pollutants over the allowed threshold causes acute arterial vasoconstriction by generating systemic inflammation and cytokine production,[21] which are possibly related to the free radical activity of components in particulate matter[22] and to the impairment of large artery endothelial function.[23] In addition, exposure to acute and massive concentrations of nitrous oxide presumably present in the atmosphere of the cave after the accident might promote beta-adrenergic activation with increased cardiac output, HR, and blood pressure.

A possible explanation for the conflicting findings of our research is that vasodilatation is the primary consequence of nitroglycerin and nitrogen compound toxicity on the one hand, and arterial vasoconstriction with higher HR and blood pressure in the BG may be related to the inhalation of particulate air pollution and gaseous pollutants on the other hand.

Glycemia and Hb levels decreased in all rescuers, with significant differences in Hb levels between the 2 groups. We hypothesize that the decrease in Hb levels might be related to bleeding from the airways, or presumably due to hemodilution because rescuers with bleeding symptoms drank water because of thirst (1500 mL, on average) during the evacuation from the cave. However, lung inhalation injury, inhalation burn injury, or other bleeding sources were not excluded in rescuers who were bleeding, especially because their personal protection equipment was destroyed and they were exposed to the airborne dust produced from the explosion of dolomite rocks.

Primary blast injuries are a complex type of trauma resulting in damage to gas-containing organs such as the lungs.[24] The shock wave from detonations can lead to mild blast injury to the lungs owing to increased intrathoracic pressure or chemical/thermal damage, leading to acute inflammatory responses[25] with small artery reactive hyperemia and pulmonary capillary rupture.[26] Based on this information, we hypothesize that this lung injury may have resulted in the airway bleeding observed in the 2 groups, potentially because all rescuers were exposed to gases for 110 min, on average. A chest x-ray and bronchoscopy would be necessary but was impractical in the context. The literature lacks clear evidence for the use of lung ultrasonography as a suitable tool for diagnosing mild blast injury to the lung and concurrent pulmonary bleeding in adults[27–29]; thus, in this experience, lung ultrasonography could not confirm the diagnosis of mild blast injury to the lung because no organ injuries were noted.

A pattern of mild hyperfibrinolysis[30] was observed, especially in explosive specialists. Fibrinolysis related to shockwave trauma has a wide spectrum of activity with a complex multifactorial process that is often affected by the amount of tissue injury the individual sustains.[31] The ROTEM delta analysis provided a picture of hemostasis due to the ability to assess coagulation factors, platelet function, and fibrinolytic activity.[32] Thus, we hypothesize that inhaled NO-releasing agents may increase fibrinolytic capacity[33] and modulate clotting by decreasing coagulation protein function and platelet function.[34] In addition, a decreased pH combined with a low calcium level may also influence clot and platelet function, resulting in mild coagulopathy predisposing the individual to increased bleeding[35,36] such that the effect of one factor may enhance the effects of the others.

LIMITATIONS

This retrospective cohort study analyzed clinical data from rescuers involved in a cave accident. The incident was not planned, but it provided an opportunity to perform our research, which could be considered the principal limitation of the study.

After the accident, we were unable to clearly determine which agent(s) caused changes in arterial blood gas and ROTEM parameters because the threshold of the toxic gases detected was impossible to record. We

hypothesize that the rescuers were exposed to toxic gas levels over the allowed threshold for the entire time spent exiting the cave.

In addition, the rescuers were significantly exposed to many other toxic agents not measured by the detectors, such as dust, quartz, and other volatile compounds. We could not collect these data because of the harsh environment and the emergency situation, which was another limitation of this study. Another important drawback of this research was the lack of methemoglobinemia and lactate values in the arterial blood gas tests; the cartridges used for our iSTAT handheld blood analyzer did not include these parameters, and we were unable to collect these data. Although methemoglobin intoxication was suspected, the clinical signs required to confirm this diagnosis were not recorded. Sulfur dioxide and cyanide may also have been liberated in the blast and might have contributed to the clinical symptoms related to uncontrolled detonation. Unfortunately, we were unable to measure their levels in the atmosphere of the cave using handheld gas detectors, which were not able to detect these gases; in addition, all rescuers lost their handheld gas detectors during their escape from the cave. Last, a kappa test was not used for interrelater reliability for the clinical examination or e-FAST, which is another limitation of this research.

Conclusions

The higher blood pressure, respiratory acidosis with hypoxia, hypercapnia, a compensatory metabolic response, and mild hyperfibrinolysis were probably related to a combined effect of nitrogen compounds and the inhaled toxic products of the detonation. Each element exerts a determinant effect in promoting the biological toxicity of the others. This information could be useful for providing a strategy for future clinical interventions in individuals with acute exposure to nitrogen compounds combined with massive inhalation of air pollutants.

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ORIGINAL RESEARCH

Cardiovascular and Blood Oxidative Stress Responses to Exercise and Acute Woodsmoke Exposure in Recreationally Active Individuals

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Introduction—Those who work and recreate outdoors experience woodsmoke exposure during fire season. Exercise during woodsmoke exposure harms the cardiovascular system, but the acute physiologic and biochemical responses are understudied. The purpose of this pilot laboratory-based study was to examine the effect of exercise during woodsmoke exposure on acute indicators of cardiovascular function, including heart rate variability (HRV), pulse wave velocity (PWV), blood pressure (BP), augmentation index (AIx), and blood oxidative stress.

Methods—Ten participants performed 2 moderate-intensity exercise (70% $\dot{V}O_{2\ max}$) trials (clean air 0 $\mu g \cdot m^{-3}$, woodsmoke 250 $\mu g \cdot m^{-3}$) in a crossover design. HRV, PWV, BP, AIx, and blood oxidative stress were measured before, after, and 90 min after exercise for each trial. Blood oxidative stress was quantified through lipid damage (LOOH, 8-ISO), protein damage (3-NT, PC), and antioxidant capacity (TEAC).

Results—A 45-min woodsmoke exposure combined with moderate-intensity exercise did not result in a statistically significant difference in HRV, PWV, BP, AIx, or oxidative stress ($P>0.05$).

Conclusions—Despite the known deleterious effects of smoke inhalation, moderate-intensity aerobic exercise while exposed to woodsmoke particulate matter (250 $\mu g \cdot m^{-3}$) did not result in a statistically significant difference in HRV, PWV, or blood oxidative stress in this methodologic context. Although findings do not negate the negative impact of woodsmoke inhalation, additional research approaches are needed to better understand the acute effects of smoke exposure on the cardiovascular system.

Keywords: air pollution, free radicals, antioxidants, autonomic recovery, parasympathetic recovery, arterial stiffness

Introduction

Smoke inhalation from biomass combustion is a public health problem in the western United States owing to frequent and large-scale wildfires.[1] Health dangers are associated with biomass particulate matter (PM) inhalation, including airborne particles ≤2.5 microns in diameter ($PM_{2.5}$). Populations affected by $PM_{2.5}$ include wildland firefighters and those who exercise outside.[2] Exercise ventilatory rates elevate the inhaled $PM_{2.5}$ dose, and smoke inhalation dose is proportional to the $PM_{2.5}$ concentration, duration of exposure, and ventilatory rates associated with activity.[3] Accordingly, this approach for estimating woodsmoke inhalation doses is currently untested in laboratory assessments where the acute impacts on cardiovascular function can be determined.

Long-term smoke inhalation elevates the risk for cardiovascular mortality by 0.4 to 1.0% with an incremental increase of only 10 $\mu g \cdot m^{-3}$ above the mean 24-h $PM_{2.5}$ concentration.[4] Furthermore, chronic $PM_{2.5}$ inhalation is associated with reduced life expectancy and increased rates of cardiovascular disease.[4] How the long-term consequences of smoke inhalation are reflected by acute physiologic and biochemical responses to smoke

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inhalation is relatively understudied. Preliminary findings in this regard indicate that pathophysiologic mechanisms include perturbations in autonomic dysregulation, mitigated cardiovascular control, systemic inflammation, and oxidative stress.[4] Cardiovascular and autonomic control can be quantified by heart rate variability (HRV), pulse wave velocity (PWV), and resting blood pressure (BP). HRV is prognostic of cardiovascular health when HRV recovery after physiologic stressors is delayed.[5] In some experimental context, HRV is reduced by woodsmoke inhalation, suggesting a concomitant decline in autonomic tone.[6,7] PWV and blood oxidative stress also provide further insight into acute physiologic changes after exercise with woodsmoke exposure.

We examined the effect of exercise on acute woodsmoke inhalation using a laboratory-based pilot investigation. Key dependent variables included metrics of cardiovascular function (HRV, PWV) and a blood oxidative stress panel to gauge associations between woodsmoke inhalation and cardiovascular control. This approach was based on prior investigations that demonstrated physiologic perturbations at $PM_{2.5}$ inhalation dosages of 250 $\mu g \cdot m^{-3}$,[8,9] or "very unhealthy" according to the air quality index. We hypothesized that moderate-intensity exercise during a 45-min woodsmoke exposure at 250 $\mu g \cdot m^{-3}$ would decrease HRV, elevate PWV, and amplify blood oxidative stress.

Methods

University of Montana institutional review board and participant informed consent was obtained before data collection. Inclusion criteria dictated a $\dot{V}O_{2\ max}$ of ≥40.0 $mL \cdot kg^{-1} \cdot min^{-1}$. The presence of limiting orthopedic problems, smoking (previous 6 mo), or diagnosed cardiovascular, pulmonary, or metabolic diseases (determined by the physical activity readiness questionnaire) resulted in exclusion from the study.[10]

The study used a crossover design. Data collection started between 0600 and 0900 on 3 occasions with a minimum of 7 d separating visits to allow for a wash-out period after smoke exposure. Participants fasted for 10 h, abstained from alcohol and exercise for 24 h, and avoided caffeine for 12 h before testing. The first laboratory visit consisted of study orientation, body composition measurements, and a $\dot{V}O_{2\ max}$ cycling test. The 2 exposure trials were performed in filtered air (0 $\mu g \cdot m^{-3}$) and woodsmoke (250 $\mu g \cdot m^{-3}$) during 45 min of cycling at 70% $\dot{V}O_{2\ max}$. Measurements of HRV, PWV, augmentation index (AIx), BP, pulmonary function (PF), and blood sampling for oxidative stress were conducted before (PRE), immediately after (IPE), and 90 min postexercise (90-P) (Figure 1). The battery was completed within 30 to 45 min. All IPE measurements were collected within 45 min of the cessation of exercise, and all 90-P measurements were collected between 90 and 120 min after completion of exercise.

Body composition was measured via hydrostatic weighing (Exertech, Dresbach, MN). Values were corrected for residual lung volume (residual lung volume=(0.01115·Age)+(0.019·Height)−2.24) and body volume estimates were converted to percent body fat using the Siri Equation.[11] The $\dot{V}O_{2\ max}$ test was performed on a cycle ergometer (Velotron, Spearfish, SD) after a 5-min warmup at 50 W and transitioned into a ramp protocol (resistance +1 W/2 s until <60 rpm occurred). Expired gas was sampled using indirect calorimetry (Cosmed Quark CPET, Concord, CA).

Exposure trials were conducted at the inhalation and pulmonary physiology core at the Center for Environmental Health Sciences at the University of Montana. Fires were generated using an in-line inhalation system woodstove stoked with western larch (*Larix occidentalis* Nutt). Fires were prepared 30 min before exercise using 1 kg of wood and kindling and were stoked with 300 g of wood every 15 min throughout the trial.

Participants kept a nutritional log for 24 h before each session and repeated dietary practices for each trial. Participants were exposed to filtered air (0 $\mu g \cdot m^{-3}$) and woodsmoke (250 $\mu g \cdot m^{-3}$) in a crossover design while exercising. Exposures were alternated and conducted in a blinded fashion. A third-party core-laboratory researcher was aware of the exposure condition until the conclusion of data collection. $PM_{2.5}$ levels were monitored continuously, and woodsmoke concentrations were maintained with $PM_{2.5}$ monitors (DustTrak, TSI, Model 8530, Shoreview, MN). Participants breathed through a modified mask to deliver woodsmoke. Exercise consisted of cycle ergometry at 70% $\dot{V}O_{2\ max}$ (Monark 828E, Langley, WA) for 45 min. HRV, PWV, AIx, BP, PF, and blood samples were collected at PRE, IPE, and 90-P. PRE and 90-P measurements were collected in the following order: HRV, PWV, AIx, BP, PF, and blood samples. IPE measurements were collected in the same order, except the blood samples were taken before HRV measurements and immediately after the cessation of exercise. When collecting IPE and 90-P measurements, researchers were blind to the pre-exercise values until the time of analysis.

HRV indices included the root mean square of successive differences (RMSSD), high frequency (HF), low frequency (LF), and the ratio LF:HF, as based on established methods.[5,12] Participants tested in a quiet, dimly lit room in supine position on an examination table. ECG electrodes were placed in a modified limb lead configuration using an iWorx system (iWorx, Dover, NH). Three

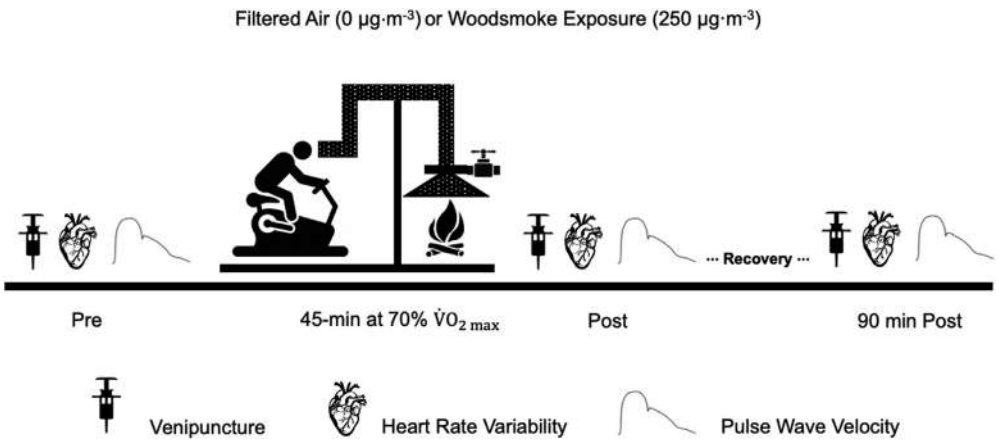

Figure 1. Experimental design. A crossover design was used for the investigation to examine differences in cardiovascular and oxidative responses with exposure to either filtered air (0 $\mu g \cdot m^{-3}$) or woodsmoke (250 $\mu g \cdot m^{-3}$).

10-min recordings were obtained (PRE, IPE, and 90-P), and the last 5-min segment was used for analysis on Kubios software (Kubios, V 2.2, Joensuu, Finland). As an a priori decision, all 4 HRV metrics would need to be altered by exposure to be considered physiologically significant, owing to the interrelational nature of the assessment.

PWV, AIx, and BP were obtained using the SphygmoCor XCEL device (Atcor Medical, Sydney, Australia) after 10 min of supine rest. Right side measurements were recorded until 3 values were obtained within 0.5 $m \cdot s^{-1}$. The femoral cuff was placed around the upper thigh, and carotid pulse was identified using applanation tonometry. PWV between the carotid and femoral arteries was calculated based on the arterial stiffness formula: PWV=distance (m)/transit time (s).

Blood was collected via venipuncture through an antecubital vein. Samples were collected in 10-mL heparinized tubes and immediately centrifuged at 1500 rpm for 15 min at 4°C, aliquoted, and stored at −80°C until assayed.

A panel of blood oxidative stress markers was measured for lipid (lipid hydroperoxides, LOOH, 8-isoprostane, 8-ISO) and protein damage (protein carbonyls, PC and 3-nitrotyrosine, 3-NT). Trolox equivalent antioxidant capacity (TEAC) was used as a marker of antioxidant status. Samples were subjected to a single freeze-thaw cycle and were kept on ice in the dark to prevent redox alterations.

TEAC assays were performed to measure antioxidant scavenging of 2,2á-azinobis-(3-ethyl-benzo-thiazoline-6-sulfonic acid) radical anions using a colorimetric reaction. Calculated TEAC values from unknown samples were compared to standard reactions with the water-soluble vitamin E analogue Trolox.[13]

Plasma LOOH were measured by the ferrous oxidation-xylenol orange assay using absorbance spectroscopy at a wavelength of 595 nm and compared with cumene hydroperoxide standards.[14] 8-ISO was assessed using a commercially available enzyme-linked immunosorbent assay kit according to the manufacturer's guidelines (Cayman Chemical, Ann Arbor, MI).

Oxidative protein damage was quantified using absorbance spectroscopy according to the established methods of Bradford,[15] followed by a commercially available enzyme-linked immunosorbent assay kit (Enzo Life Sciences, Farmingdale, NY and Cell Biolabs INC, San Diego, CA).

PF, a secondary variable, was assessed by forced vital capacity (FVC) and maximal voluntary ventilation (MVV, performed for 12 s) tests using a spirometer (MIR Spirobank, Elicott City, MD), with participants tested in the seated position. Participants were provided with verbal encouragement during PF tests. Data were analyzed using the WINSPIRO Pro software (Version 7.8, Ellicott City, MD).

A series of exposure × time repeated-measures analysis of variance with a Bonferroni correction was performed to compare the differences in mean values. The general linear model function was used in statistical software (V.25.0, SPSS Inc., Chicago, IL) for all analyses. A criterion alpha level of $P \leq 0.05$ was used to determine statistical significance. Data are reported as mean±SD. When analyzing HRV, to satisfy the normality assumption, a natural logarithmic transformation was performed on RMSSD, HF, and LF, and these were reported as lnRMSSD, lnHF, and lnLF before statistical analysis. The aim of the study was to determine if woodsmoke exposure during moderate-intensity exercise results in deleterious effects on variables of cardiovascular function.

Results

Ten active individuals (male: n=7, female: n=3) completed the study. Participant characteristics are presented in Table 1.

No interaction effect was observed for HR (P=0.965) or recovery of HR (P=0.784). No statistically significant difference was observed in HR during the exposure trials (P=0.541) or during the recovery of HR (P=0.734). HR was elevated similarly during the experimental trials. HR responses across the trial (Table 2), when comparing rest to IPE and 90-P, indicated a time effect whereby IPE was elevated (P<0.001). Average HR values returned to baseline at 90-P (P=0.06).

HRV was quantified for lnRMSSD, lnHF, lnLF, and LF:HF (Table 2). Analysis of lnRMSSD, lnHF, lnLF, and the LF:HF ratio indicated no interaction effects (lnRMSSD: P=0.912; lnHF: P=0.148; lnLF: P=0.905; LF:HF: P=0.109) and no between-trial differences (lnRMSSD: P=0.685; lnHF: P=0.843; lnLF: P=0.275; LF:HF: P=0.057); however, a main effect of time was observed (lnRMSSD: P=0.001; lnHF: P=0.003), indicating that parasympathetic activity was reduced in response to exercise. A significant reduction occurred at IPE (lnRMSSD: P=0.016; lnHF: P=0.005), with a recovery of both indices by 90-P (P>0.99). lnLF demonstrated a time effect (P=0.039). At 90-P, lnLF was lower than IPE (P=0.026) and pre-exercise values. Finally, examination of the LF:HF ratio indicated no time effect (P=0.189).

Analysis of PWV and AIx (Table 2) indicated no interaction effect (PWV: P=0.909; AIx: P=0.626), trial effect (PWV: P=0.856; AIx: P=0.136), or time effect (PWV: P=0.975; AIx: P=0.192), indicating that these markers were not significantly different. No statistical difference was observed in brachial BP for trial (SBP: P=0.883; DBP: P=0.769) or time (SBP: P=0.293; DBP: P=0.705).

Blood oxidative stress (Table 3) analyses indicated no interaction (P=0.699) trial (P=0.505), or time effect (P=0.711) on PC, indicating that the protein damage marker was not statistically different with exposure or exercise. 3-NT and LOOH data revealed no interaction (3-NT: P=0.784; LOOH: P=0.126) or time effect (3-NT: P=0.426; LOOH: 0.159); however, a main effect for trial was observed (3-NT: P=0.046; LOOH: P=0.011). 8-ISO demonstrated no interaction effect (P=0.800), trial effect (P=0.296), or time effect (P=0.158). Antioxidant capacity demonstrated a time effect (P=0.009), but no effect of trial (P=0.068) or interaction effect (P=0.234). TEAC was elevated at IPE (P=0.002) and 90-P (P=0.003). Similarly, for blood antioxidant potential, there was a time effect (P=0.026), with an elevation at IPE (P=0.003) that returned to pre-exercise values by 90-P (P=0.06).

The secondary PF data are presented in Table 4. For FVC, there was no interaction (P=0.700), trial (P=0.632), or time effect (P=0.555). Similarly, FEV_1 and

Table 1. Participant characteristics

Characteristic	*n=10*	*Males (n=7)*	*Females (n=3)*
Age (y)	30±11	31±13	27±2
Height (cm)	176±9	182±5	165±1
Weight (kg)	76±12	83±7	62±1
Body fat (%)	19±9	17±9	24±7
$\dot{V}O_{2\ max}$ (mL·kg^{-1}·min^{-1})	44±4	46±4	41±1

Data presented as mean±SD.

Table 2. Variables of cardiovascular function

Marker	*Filtered air trial*	*Woodsmoke trial*	*Main effect P*	*Interaction effect P*
	Mean±SD			
Heart rate (beats·min^{-1})				
PRE	53±7	49±11	0.734	0.784
IPE	68±9	72±10		
90-P	57±8	55±9		
lnRMSSD (ms)				
PRE	4.3±0.6	4.3±0.5	0.685	0.912
IPE	3.6±0.6	3.6±0.5		
90-P	4.2±0.5	4.3±0.6		
lnHF (ms^2)				
PRE	7.5±1.4	7.3±1.1	0.843	0.148
IPE	6.0±1.3	5.7±1.3		
90-P	7.2±1.1	7.5±1.3		
lnLF (ms^2)				
PRE	7.3±1.2	7.4±1.2	0.275	0.905
IPE	6.4±1.2	6.6±0.9		
90-P	7.2±1.1	7.4±0.9		
LF:HF				
PRE	1.0±0.5	1.2±1.0	0.057	0.109
IPE	2.5±1.8	4.2±3.6		
90-P	1.9±1.6	1.7±1.3		
PWV (m·s^{-1})				
PRE	5.9±1.3	5.4±1.3	0.856	0.909
IPE	5.8±1.4	5.8±1.2		
90-P	6.1±1.2	6.0±1.3		
Augmentation index (%)				
PRE	7.3±5.5	3.6±8.9	0.136	0.626
IPE	9.2±12.5	7.7±8.0		
90-P	5.7±3.0	3.6±9.7		

90-P, 90 min postexercise; HF, high frequency; IPE, immediately after exercise; LF, low frequency; PRE, before exercise; PWV, pulse wave velocity; RMSSD, root mean square of successive differences.

Table 3. Variables of oxidative stress

Marker	*Filtered air trial*	*Woodsmoke trial*	*Main effect P*	*Interaction effect P*
	Mean±SD			
Protein carbonyl (nmol·mg^{-1})				
PRE	0.18±0.03	0.17±0.02	0.505	0.699
IPE	0.20±0.03	0.17±0.02		
90-P	0.18±0.03	0.17±0.02		
3-Nitrotyrosine (μg·mL^{-1})				
PRE	3.4±2.1	3.0±1.6	0.426	0.784
IPE	3.8±2.4	3.3±2.3		
90-P	3.1±2.3	2.8±1.6		
Lipid hydroperoxides (μmol·L^{-1})				
PRE	1.7±1.6	3.3±1.6	0.159[a]	0.126
IPE	2.5±2.1	4.0±2.0		
90-P	2.6±1.9	3.2±2.1		
8-Isoprostane (pg·mL^{-1})				
PRE	18.8±9.6	22.2±12.2	0.296	0.800
IPE	24.9±12.5	31.3±26.1		
90-P	24.3±13.5	28.8±23.6		
TEAC (μmol·L^{-1})				
PRE	112.7±12.3	109.9±10.4	0.068	0.234
IPE	135.5±14.8	118.4±19.8		
90-P	125.2±16.3	120.9±16.4		

90-P, 90 min postexercise; IPE, immediately after exercise; PRE, before exercise; TEAC, Trolox equivalent antioxidant capacity.
[a]Significant main effect.

FEV_1/FVC exhibited no interaction (FEV_1: P=0.373; FEV_1/FVC: P=0.464), trial (FEV_1: P=0.940; FEV_1/FVC: P=0.694), or time effect (FEV_1: P=0.406; FEV_1/FVC: P=0.723). MVV also demonstrated no interaction (P=0.800), trial (P=0.667), or time effect (P=0.068).

Table 4. Pulmonary function variables

Variable	*Woodsmoke trial*	*Filtered air trial*
	n=10	*n=10*
	Mean±SD	
FVC (L)		
PRE	5.07±0.28	5.04±0.25
IPE	5.03±0.26	5.10±0.25
90-P	5.02±0.26	5.07±0.25
FEV_1 (L)		
PRE	4.03±0.21	4.02±0.19
IPE	4.09±0.22	4.05±0.21
90-P	4.05±0.20	4.09±0.20
FEV_1/FVC (%)		
PRE	80±2	80±2
IPE	81±2	80±2
90-P	81±2	81±1
MVV (L·min^{-1})		
PRE	146±12	146±10
IPE	159±14	159±10
90-P	141±12	149±11

90-P, 90 min postexercise; FVC, forced vital capacity; FEV_1, forced expiratory volume in 1 s; IPE, immediately after exercise; MVV, maximal voluntary ventilation; PRE, before exercise.

Discussion

Long-term woodsmoke inhalation is damaging to the cardiovascular system, but the acute effects of smoke exposure are not well understood. Recreational exercisers, wildland firefighters, and persons with chronic outdoor exposure during wildfire season are at risk for long-term cardiovascular health consequences.[8,9] Accordingly, using a laboratory-based pilot investigation, we examined the acute effects of exercise and woodsmoke-inhalation ($PM_{2.5}$ 250 μg·m^{-3}) on the primary variables of HRV, PWV, and oxidative stress. Indeed, inhalation of concentrated smoke (250 μg·m^{-3}) elicits inflammation and oxidative stress response known to be deleterious to cardiovascular health.[9,16] Although chronic smoke inhalation is detrimental to long-term cardiovascular function, our acute physiologic (HRV, PWV) and biochemical blood markers of oxidative stress were not statistically different between the smoke inhalation and filtered air challenges. This unexpected finding highlights the scientific importance of identifying acute physiologic and biochemical metrics that may better

inform the long-term consequences of woodsmoke inhalation. We interpret these negative findings to have been influenced by the experimental context in which the laboratory-based investigation was conducted, attributing potential causation to the following methodologic factors: 1) examination of a population—young, apparently-healthy subjects—presumably more resilient to this smoke inhalation challenge; 2) methodologic constraints (eg, sampling time points and selection of biochemical variables); 3) small sample size; and 4) insufficient dose, which did not capture perturbations due to smoke.

A key dependent variable of this investigation was HRV. Inability to maintain a high resting HRV, or exhibiting delayed rebound after exposure to a stressor, predicts an increased risk for mortality and sudden cardiac death.[17] Previous longitudinal findings demonstrated decreased HRV in those regularly exposed to woodsmoke PM in occupational settings.[7] Notably, depression of HRV after acute smoke inhalation signals problematic responses to reoccurring exposure. Accordingly, we examined HRV after a single woodsmoke challenge in apparently healthy research participants to examine the potential of declines in HRV that could inform recurrent exposures. Results indicated no statistically significant alterations existed for HRV (lnRMSSD, lnHF, lnLF, LF:HF) for up to 90 min after exposure, a finding that does not discount the deleterious effects of acute smoke inhalation. In contrast to our findings, HRV (SDNN, RMSSD, HFnu, and pNN50) was systematically reduced in 14 subjects after a 3-h intermittent exercise session with exposure at ~300 $\mu g \cdot m^{-3}$,[6] This divergent outcome may indicate that the duration of smoke exposure within the current study was insufficient to elicit changes, highlighting the need to identify a woodsmoke exposure (duration and/or $PM_{2.5}$ concentration) threshold that perturbs cardiovascular function. Moreover, age, fitness level, and health status—and the duration of HRV assessment (eg, longer sampling periods)—should be examined. Previous work demonstrated that HRV is acutely depressed in woodsmoke-exposed elderly populations.[18] Additionally, postexercise parasympathetic rebound is enhanced in fit and healthy populations,[5] suggesting our sample may have exhibited preserved HRV as compared to a sedentary population. Given the disparate outcomes of this study compared to others, future investigations should consider longer-duration baseline and post-intervention HRV analyses to confirm or refute the current findings.

PWV is predictive of elevated cardiovascular event risk,[19] although prior studies have examined the effects of woodsmoke exposure on PWV with equivocal results.[6,20] Our findings demonstrated that PWV was not statistically different after a 45-min exercise session in filtered air and woodsmoke exposure at 250 $\mu g \cdot m^{-3}$, a finding that is similar to a prior investigation reporting negative PWV outcomes 1 h postexercise with an exposure at 1000 $\mu g \cdot m^{-3}$.[20] However, PWV has been demonstrated to increase for up to 25 min after a 3-h bout of exercise and an exposure of 300 $\mu g \cdot m^{-3}$.[6] Collectively, PWV may be more responsive to exposures between 1 and 3 h in duration.[6,20,21] In support, a recent study examined the effects of woodsmoke exposure at 3 different concentrations (100 $\mu g \cdot m^{-3}$, 250 $\mu g \cdot m^{-3}$, and 500 $\mu g \cdot m^{-3}$) under resting conditions, finding that PWV was elevated 24 h after all exposures.[21] The magnitude of the elevation was similar across all 3 concentrations, signifying that PWV may not respond to smoke in a dose-dependent manner.

AIx is an additional variable of arterial stiffness that is associated with elevated cardiovascular disease risk.[22,23] Our data demonstrated that AIx was not statistically different between the smoke exposure and filtered air trials for this healthy population. Previous research indicated AIx was elevated at a slightly higher dose of $PM_{2.5}$ (~314 $\mu g \cdot m^{-3}$) after 3 h of exposure with intermittent exercise[6]; however, this response was not replicated when intermittent exercise was performed for 1 h at a $PM_{2.5}$ dose of ~1000 $\mu g \cdot m^{-3}$.[20] Although these mixed findings cannot be explained currently, there is a proposed association between elevated oxidative stress and increased arterial stiffness, indicating they may occur in tandem.[24] Therefore, the exercise or exposure may require a yet-to-be determined duration or intensity threshold to elicit a measurable change in arterial stiffness.

All postexercise oxidative stress variables were not statistically different between woodsmoke and filtered air exposures as examined at these selected time points in healthy research participants. Previous research has demonstrated that a 1.5-h treadmill bout with woodsmoke exposure at 500 $\mu g \cdot m^{-3}$ resulted in an elevation of 3-NT immediately after exercise, but did not rise with an exposure at 250 $\mu g \cdot m^{-3}$.[8] In agreement with prior work, the current study indicated no statistically significant elevation after 45 min of exercise and smoke (250 $\mu g \cdot m^{-3}$), suggesting that 3-NT may respond in a dose-dependent manner. Moreover, our negative findings for PC are consistent with the previously mentioned study, where PC was also unaltered by woodsmoke exposure at both 250 $\mu g \cdot m^{-3}$ and 500 $\mu g \cdot m^{-3}$.[8] Importantly, these negative findings do not negate the detrimental impact of smoke inhalation on protein damage markers. By extension, we may have simply missed the spike in PC, as this marker has been shown to peak hours or days after the stressor[25,26]; thus, the selected sampling time points used currently may not have captured the peak response.

Lipid oxidative damage was quantified via plasma LOOH and 8-ISO. Previous research demonstrated no elevation of LOOH after a 1.5-h woodsmoke exposure during low-intensity exercise[8]; however, 8-ISO was elevated immediately after exercise at 2 inhalation doses (250 $\mu g \cdot m^{-3}$ and 500 $\mu g \cdot m^{-3}$). Moderate-intensity exercise (70% $\dot{V}O_{2\ max}$ cycling) was selected currently to elevate ventilatory rates; however, LOOH and 8-ISO were not statistically different after exercise with $PM_{2.5}$=250 $\mu g \cdot m^{-3}$. Nonetheless, LOOH is most responsive to high-intensity exercise,[27] suggesting that our exercise modality and $PM_{2.5}$ dose were insufficient to elicit elevations.

TEAC was also examined after exercise and woodsmoke exposure; however, it was not statistically different between woodsmoke and filtered air exposures. Previous work examined the TEAC response to higher woodsmoke exposure (average 375 $\mu g \cdot m^{-3}$) during a 1.5-h bout of treadmill walking and found TEAC was elevated in response to woodsmoke exposure.[8] However, when the prior 250 $\mu g \cdot m^{-3}$ exposure trial was examined alone, TEAC was not elevated in response to the exposure, in agreement with our TEAC values after a 45-min exposure at 250 $\mu g \cdot m^{-3}$. These collective findings indicate that TEAC may be responsive to woodsmoke exposure in a dose-dependent manner. Overall, we did not observe a statistically significant increase in postexercise oxidative stress markers between smoke inhalation and filtered air, which suggests that these markers respond differently based on exposure dose and duration, in addition to exercise intensity. We interpret this finding to indicate that our methodologic approach (eg, exercise dose, population, and sampling time points) did not elicit oxidative changes, a finding that would have otherwise been present in multiple markers across our oxidative stress panel.

PF was examined as a secondary group of variables in our healthy sample population. Findings indicated that FVC, FEV_1, and MVV were not statistically different for either exercise or exposure. Importantly, these findings are consistent with previous work that demonstrated no changes in FVC, FEV_1, or MVV after a 1.5-h exposure during treadmill walking[9] and highlights the fact that although smoke inhalation is clearly detrimental to long-term health,[28] acute physiologic responses to exposure may not always result.

LIMITATIONS

Study limitations include the nature and size of the sample. Owing to the age range, fitness level, and overall health of the examined sample, inferences that can be drawn from these results are limited proportionately. Thus, it is plausible that these methods, if applied to more susceptible populations (eg, age, chronic conditions), may have produced alternative outcomes for our grouped outcome measures of HRV, PWV metrics, and oxidative stress markers. Furthermore, female participants were underrepresented within this study (n=3) and the potential confounding influence of hormonal alterations (eg, menstrual cycle or contraceptive use) was not considered. For this reason, conclusions about female-specific responses cannot be drawn from the results of the current study. Furthermore, the dose of woodsmoke exposure may been insufficient to elicit adverse effects on the selected variables. The study design was predicated on the notion that a 7-d washout period is sufficient for the assessment of acute physiologic responses to smoke exposure, an assumption that should be confirmed with future investigations. Additionally, the study examined only 1 modality, duration, and intensity of exercise, limiting the application of these findings to similar exercise.

Conclusions

Chronic woodsmoke exposure is linked to negative health effects on cardiovascular control and oxidative stress, although the influence of acute smoke inhalation effects are not well defined. The current study quantified the acute effects of 45 min of exercise during woodsmoke exposure (250 $\mu g \cdot m^{-3}$) on markers of HRV, arterial stiffness, and oxidative stress; however, there were no statistically significant differences observed as compared to filtered air. Given the undeniable link between chronic smoke inhalation and cardiovascular health, we interpret the negative findings of the current study to have been limited by the experimental context in which the laboratory-based study was conducted. Future research should examine the threshold for duration, dose, and/or frequency of smoke exposure needed to produce acute perturbation in these parameters. Moreover, the health and fitness status of the investigated population may alter the exposure thresholds, meaning that participant age, training status, medical conditions, and sex-dependent differences should also be considered in future study designs.

Author Contributions: Study concept and design (JCQ, CLD); acquisition of data (CWR, KT, DR, KC, SG, KW, TQ); analysis of data (CWR, KC, SG, TQ); drafting of the manuscript (CWR, JCQ); critical revision of the manuscript (CWR, JCQ, KT, KC, SG, CLD); approval of the final manuscript (all authors).

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ORIGINAL RESEARCH

Injury Rates, Patterns, Mechanisms, and Risk Factors Among Competitive Youth Climbers in the United States

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Introduction—Competitive rock climbing is a fast-growing sport. Despite comprehensive reviews on adult climbing-related injuries, few pediatric-specific reviews exist, and studies exclusively on competitive youth climbers are needed. Objectives of this study include 1) estimating the injury rate (IR); 2) describing injury patterns and mechanisms; and 3) identifying injury risk factors in competitive youth climbers.

Methods—The study design was cross-sectional. Competitive youth climbers were included. Participants completed an anonymous questionnaire to document climbing injuries over the preceding 12 mo. Demographic data and data regarding injuries were collected. The IR was calculated. Analyses were performed to assess association between injury and multiple variables. Multivariate logistic regression was completed for significant variables to control for exposure time.

Results—The IR was 2.7 injuries per 1000 climbing hours. Hand/Finger injuries were most frequent; chronic overuse was the most common etiology. Injury severity was low overall. Risk factors significantly associated with climbing injury were climbing discipline (bouldering > sport/lead climbing), return to climbing while still in pain, finger taping, higher number of hours climbed per session and per year, climbing at higher bouldering difficulties, and unsupervised climbing.

Conclusions—The IR in competitive youth climbers was found to be lower than previously reported but higher than suggested by adult studies or those that exclude chronic injuries. Findings are consistent with types, severity, and mechanisms reported in other studies. Modifiable risk factors, especially return to climbing while still injured, warrant further prospective investigation.

Keywords: pediatrics, rock climbing, sports injuries, competitive rock climbing, injury risk factors

Introduction

Indoor competitive climbing is a fast-growing youth sport. As of July 2019, over 4300 youth (athletes <19 y of age) competed worldwide,[1] and average athlete age is decreasing.[1,2] Its inclusion in the 2020 Summer Olympics further increased its professionality and influence training regimens, injury rates, and injury profiles.[2-5]

Despite the high level of competition, youth climbers are more prone to making errors, underestimating objective risks, and sustaining injury owing to numerous well-described factors.[1,2,3,5] A study specifically examining youth climber injury showed greater than twice the risk of injury in competitive climbers vs recreational ones.[6]

Several comprehensive studies and reviews exist for adult climbing-related injuries, but few specifically describe patterns, rates, and risk factors of injury in youth climbers or include only climbers aged ≤18 y. A 2018 meta-analysis on pediatric climbing injuries highlighted this knowledge gap in light of the need to obtain an accurate picture of injury risk and severity; the analysis called for future studies to determine injury rates relative to activity level to enact preventative safety measures for youth competitive climbers.[3]

This study attempts to obtain, in a very limited way, early pediatric-specific data regarding competitive climbers by estimating rates, patterns, mechanisms, and risk factors of injury.

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Methods

This was a retrospective, cross-sectional study to estimate the current frequency and impact of injuries sustained by competitive youth climbers (age 5−18 y). The primary objectives were to 1) estimate the rate of injuries per hour climbed; 2) describe the patterns and mechanisms of injury; and 3) identify risk factors for injury among children who participate in competitive rock climbing.

The "injuries in competitive youth climbers questionnaire" obtained data and was modified from a validated, international questionnaire on climbing injuries in adolescents.[6] Any added questions or segments were reviewed by a group of competitive climbers, coaches, pediatricians, a youth athletic trainer, and a physical therapist for face validity (n=15). The institutional review board at UT Southwestern Medical Center approved the study. Participation in the anonymous survey was explicitly voluntary. Completion was accepted as consent to participate.

The electronic survey was sent by email to coaches of youth climbing teams across the United States for distribution to their athletes via secure link. Information about participation was also posted online in climbing forums. Because the exact number of athletes approached could not be determined owing to the use of a secure link posted in climbing forums, the response rate was calculated as the percentage of completed surveys vs unique clicks of the link.

We used a timeframe of the preceding 12 mo when describing injuries reported, based exclusively on participant/parent recollection over this time period. The survey was distributed from February through April 2020 via REDCap (an electronic survey software), and results were returned to the study investigators for analysis.

The study focus was on indoor climbing (lead and bouldering) in climbers ≤18 y old. All respondents of this age who are current members of a US climbing team (or were members within the past 12 mo), who had competed in the past 12 mo or planned to compete in the next competitive season, and who completed the mandatory survey portions were included. There were no additional specific exclusion criteria.

Exposure was defined as competitive climbing participation. The primary outcome measure was self-reported injury sustained during climbing in the past 12 mo. Injury was defined as any pain or physical complaint and was characterized as acute ("single injury, pain, or physical complaint" that could be quantified in terms of time of onset or related to a specific event) or chronic ("ongoing pain and/or physical discomfort").

The survey collected the following information:

- Demographic information and medical history
- Participation in climbing disciplines
- Climbing time
- Injury prevention measures used
- Percentage of climbing time spent under direct adult supervision
- Types, anatomic locations, and mechanisms of injury
- Time to return to climbing, injury status at return, and athletes' reasons for returning
- Treatments required
- Difficulty of climbing being undertaken (using the Hueco/V scale for bouldering, which begins at V0 and increases to V14+, and the Yosemite decimal scale, which begins at 5.4 and increases to 5.15+, for sport/lead climbing)
- Level of competition
- Other sport participation and injuries sustained during those sports

A previous study evaluating general injury rates and patterns in youth climbers enrolled 50 competitive climbers.[6] This was used as our minimum sample size because total population and effect size were unknown.

Descriptive data are reported as mean±SD for normally distributed continuous data, median (IQR) for ordinal data, or percentage, as appropriate. All data obtained were self-reported.

Potential risk factors evaluated for athlete-reported injury included demographic information, climbing exposure/hours, other sport participation, discipline, difficulty, competitive level, climbing while in pain, preventative measures undertaken, and supervision. Factors were evaluated independently against acute, chronic, and multiple injuries reported, as well as against all injuries (inclusive of acute and chronic).

Inferential statistics were determined for these potential risk factors and their relationships to reported injury. A significance level of $P<0.05$ was chosen. Given the small sample size, the Fisher exact test was performed to calculate the crude odds ratio (OR), significance level, and 95% confidence intervals (CIs). Multivariate logistic regressions controlling for exposure hours were performed in secondary analysis for significant findings to yield adjusted ORs. We chose this method becaue it had been successfully employed to assess risk factors for injury in pediatric climbers[3,6] and would allow our results to be compared against data from the most recent related meta-analysis. Independent t tests were used where appropriate for continuous vs categorical data, and Pearson's correlation scatterplots were created for purely continuous data.

Statistical testing was performed using data obtained from our institutional REDCap survey tool and the statistical software JASP 2020 (Version 0.12.2).

Results

Fifty-two respondents aged 7 to 18 y completed all mandatory portions of the survey. Six participants completed only demographic sections, and 2 answered limited questions about climbing time but provided no injury data and thus were excluded. Table 1 summarizes participant characteristics and participation. Digital survey distribution strategies resulted in a response rate of 67%, although because this was calculated as "unique clicks" vs "completed surveys," it likely overestimates the true response rate. Thirty-four participants (65%) reported competing at least at the regional level of competition; all competed in local competitions or higher. Respondents reported climbing 2.5±0.6 h per session and approximately 389±191 $h{\cdot}y^{-1}$.

Twenty-two climbers (42%) reported an acute injury. Of these, 6 (12%) were re-injuries after previous recovery, and 7 (14%) reported multiple acute injuries. When chronic injuries were added, 34 climbers (65%) reported a new injury. Fifty-two participants recorded a total of 56 injuries, resulting in an injury proportion of 108 per 100 athletes. The overall injury rate was 2.9 per 1000 participation hours. The injury rate was 1.7 per 1000 participation hours when considering only acute injuries (Table 2).

Hand/Finger injuries represented the most common type of injuries reported (36%). Other body areas injured included elbow and wrist (13% each); head (8%); hip/pelvis, shoulder, and lower leg/ankle (5% each); neck and foot/toe (4% each); and thigh and knee (2% each). No facial, dental, back, or chest injuries were reported. Equal numbers of sprain/strain, tendinitis/tendinosis, and pulley injuries were reported (9 each). Four concussions and 3 fractures were reported. Overuse/Repetitive stress injuries were the most commonly reported mechanism (70%). Other mechanisms included falls (14%) and performing a large/strenuous move off of a small hold (14%).

In regard to treatment for injuries, self-performed or coach-assisted aid without involvement of medical personnel was most common (first-aid, taping=44% of treatments), followed by outpatient physician visit (27%) and physical therapy (17%). Three required emergency department visits, and 1 required surgical intervention.

Overall, 31 injuries (55%) resulted in at least 1 d of climbing time lost. For acute injuries, the most common amount of time taken off was 1 to 4 wk (15 climbers), whereas chronic injuries/ongoing pain resulted in most climbers taking no time off.

Table 1. Baseline characteristics and demographics

Characteristic	*n (%)*	*Mean±SD or Median (IQR)*
Total participants	52	
Sex		
Male	23 (44)	
Female	29 (55)	
Age (y)		14±3
5–7	0 (0)	
8–9	3 (6)	
10–11	7 (14)	
12–13	11 (21)	
14–15	12 (23)	
16–17	8 (15)	
18	11 (21)	
Body mass index ($kg{\cdot}m^{-2}$)		19.0±2.6
Member of USA Climbing	34 (65)	
Competes at more than local level	34 (65)	
Climbing hours per session		2.5±0.6
Climbing hours per year		389±191
Bouldering difficulty (Hueco scale)		V5–V6 (V4-V7)
Sport climbing difficulty (Yosemite decimal scale)		5.11c–5.11d (5.11a–5.12a)
Teams represented	20	

Ninety-one percent of reported acute injuries occurred during bouldering ($P<0.001$). Bouldering difficulty was also associated with self-reported injury; athletes who reported injuries also reported climbing more difficult problems/routes (V5 and below vs V6 and above, $P=0.04$). Total reported injury number increased similarly (R=0.33, 95% CI 0.06–0.55, $P=0.02$). These results remained significant when controlled for exposure hours ($P=0.03$). Sport/Lead climbing difficulty was not associated with increased risk in our limited data set. As climbing time per year increased, so did the report of any climbing-related injury ($P=0.001$). Climbers were also more likely to report injuries if their sessions were longer, especially >2 h in a single session ($P<0.001$).

Another potentially significant risk factor was return to climbing while still injured or in pain. Fifty-two percent reported climbing while in pain or returning after suspected injury while still hurting. Early return increased the odds of any reported injury (OR 24.4, 95% CI 4.6–130.1, $P<0.001$), acute injury (OR 3.5, 95% CI 1.1–11.3, $P<0.04$), and chronic injury (OR 15.5, 95% CI 3.8–66.7, $P<0.001$) after controlling for climbing exposure hours. Our small group of athletes reported varying reasons for premature return, including a belief that the injury was minor (29%), fear of losing climbing skills (25%), concern about upcoming competition (25%), fear

Table 2. Injury rates and patterns

Injury pattern	*Injury rate, injuries per 1000 climbing hours*
Total	2.9
Acute injuries only	1.7
Injured climbers	n (% of climbers)
Total	34 (65)
Any acute injury	22 (42)
Multiple acute injuries	7 (14)
Chronic injuries/significant climbing-related pain	24 (46)
Reinjury after previous complete recovery	6 (12)
Injury number	n (% of injuries)
Total	56; mean = 1/climber, SD 1
Acute injuries	34 (61)
Chronic injuries/significant climbing-related pain	22 (39)
Anatomic location of injuries	n (% of injuries)
Head	4 (8)
Acute	4
Chronic	0
Face/Mouth	0 (0)
Neck	2 (4)
Acute	1
Chronic	1
Chest	0 (0)
Shoulder	3 (5)
Acute	1
Chronic	2
Upper arm	0 (0)
Elbow	7 (13)
Acute	5
Chronic	2
Forearm/Wrist	7 (13)
Acute	6
Chronic	1
Hand/Finger	20 (36)
Acute	9
Chronic	11
Hips/Pelvis	3 (5)
Acute	2
Chronic	1
Thigh/Upper leg	1 (2)
Acute	1
Chronic	0
Knee	1 (2)
Acute	1
Chronic	0
Lower leg/Ankle	3 (5)
Acute	2
Chronic	1
Foot/Toe	2 (4)
Acute	1
Chronic	1
Acute injury type	n (% of injuries)
Sprain/Strain	9 (27)
Tendinitis/Tendinosis	9 (27)
Pulley injury/rupture	9 (27)
Concussion	4 (12)
Fracture	3 (9)
Mechanism of all injuries (acute and chronic)	n (% of injuries)
Overuse/Repetitive Stress	39 (70)
Fall	8 (14)
Large/Strenuous move off of small hold	8 (14)
Not specified	1 (2)
Treatments required for injuries	n (% of treatments tried)[a]
None	2 (3)
Self-administered first aid	16 (21)
Taping	18 (23)
Visit to pediatrician orthopedist, or sports medicine	21 (27)
Visit to emergency department	3 (4)
Cast or splint	3 (4)
Physical therapy	13 (17)
Surgery	1 (1)

[a]More than 1 response accepted.

of losing a spot on a climbing team (10%), peer pressure (10%), and pressure from a coach or parent (3%).

Use of finger taping as a preventative measure increased the odds of reported injury by a factor of 1.4 (95% CI 1.4−4.5, P=0.04) when adjusted for climbing hours.

A statistically significant association between percentage of time an athlete spends climbing under direct adult supervision and injury was suggested for both bouldering and lead climbing (Figures 1 and 2).

Current age, age at which the athlete began competing, sex, other sport participation, other sport injury, level of competition, safety skills taught by the team or learned outside of the team setting, use of a hang board training device, presence of past medical conditions, and warming up/cooling down were not statistically associated with increased or decreased odds of injury generally nor any subcategory of injury in our limited data set (Table 3).

Discussion

Our study attempts to add data to the growing body of literature examining rate, proportion, types, and mechanisms of injury and risk factors for injury in competitive

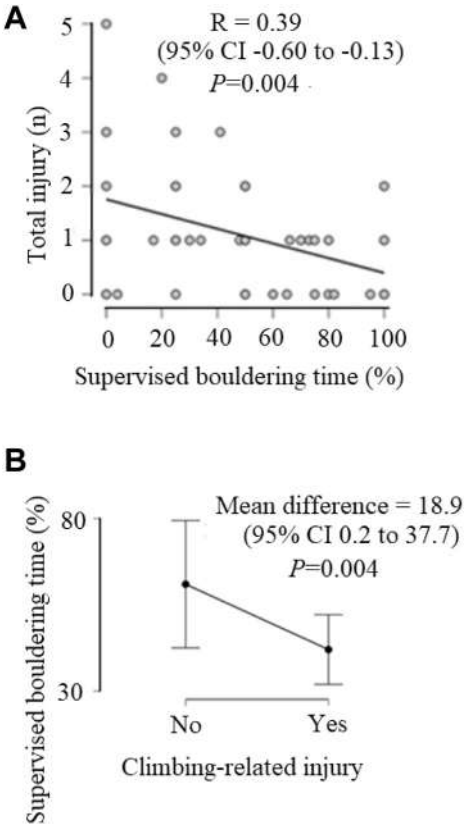

Figure 1. (A) Supervised bouldering time vs total injury number. (B) Mean difference in development of a climbing-related injury vs percentage of time spent bouldering while supervised.

youth climbers, but it is vital to reiterate that all data are both self-reported and from an extremely small sample of climbers. Although lead climbing and bouldering tend to be male dominated at the elite level, the demographics of the sport are changing rapidly. Our study had nearly equal numbers of male and female participants, which may represent this change or may represent a bias in surveying. Despite the small number of participants and low response rate, which limit broad interpretation of our results, we were able to obtain data from a wide geographic area representing 20 climbing teams in all regions of the United States and climbers competing at different levels.

Injury types reported were consistent with those described in other available climbing literature, with hand/finger injuries dominating.[1,3,6-16] Repetitive/Overuse was the most common self-reported mechanism of injury, consistent with many other studies on the subject, despite the vagueness of the phrase "repetitive overuse." Falls and making a large move were also commonly reported mechanisms, again consistent with previous data.[4,12,14,17-19]

Most injuries reported in this small study required little time away from climbing. It is unclear whether this was due to mild severity, earlier return than appropriate, a combination of both, or the small sample of respondents and recall bias. The lack of time away and overwhelming treatment with first aid and outpatient visits suggests that severe acute injury is rare in elite youth climbers.

Our injury rate (2.9 per 1000 climbing hours) was lower than previously reported for elite youth climbers[6] but still within the range reported in a recent meta-analysis on youth climbing injuries.[2] Our lower injury rate might be due to our small sample, underreporting of minor or chronic injuries by the athletes, or failure to recall these injuries. Our self-reported injury rate could also be an overreporting based on the specific nature of the questions we asked. Overall, our injury rate is close to that among competitive youth swimming/diving and track and field and much lower than the reported injury rate for football, hockey, soccer, and basketball.[19]

Previous studies have shown mean ages of competitive youth climbers in their late teens. This was consistent with our study as well; our youngest participants were 8 y old (n=3), and multiple children aged ≤11 y participated, but 20% of respondents were aged 18 y. We do not yet

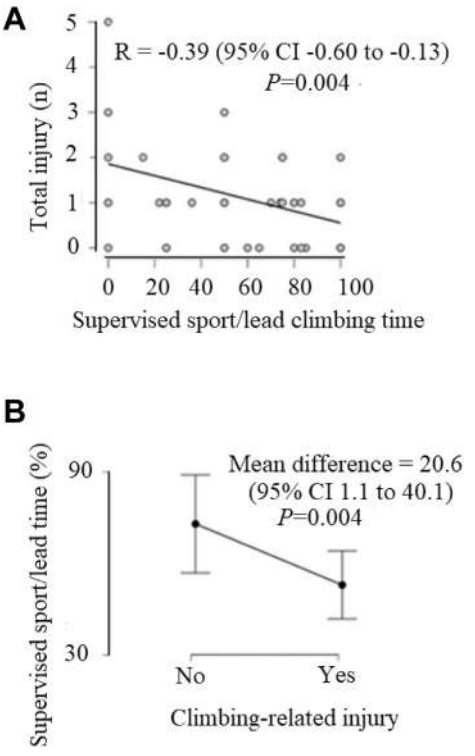

Figure 2. (A) Supervised sport/lead climbing time vs total injury number. (B) Mean difference in development of a climbing-related injury vs percentage of time spent sport climbing while supervised.

Table 3. Variables and their association with injury

Variable	*Odds ratio (OR) (95% confidence interval)*	P *value*	*Adjusted OR (95% confidence interval)*	*Adjusted* P *value*
Member of USA Climbing				
Any injury or significant climbing-related pain	2.8 (0.8–9.2)	0.13	–	–
Acute injury (at least 1)	1.2 (0.3–2.6)	0.78	–	–
Acute injuries (multiple)	3.0 (0.6–15.0)	0.22	–	–
Chronic injuries/pain	2.8 (4.5–88.9)	0.13	–	–
Returned to climbing while still injured or in pain				
Any injury or significant climbing-related pain	24.3 (4.6–130.1)	<0.001[a]	52.5 (4.9–567.8)	<0.001[a]
Acute injury (at least 1)	3.5 (1.1–11.3)	0.04[b]	1.6 (0.3–6.2)	0.06
Acute injuries (multiple)	2.5 (4.9–15.7)	0.42	3.4 (0.6–21.0)	0.14
Chronic injuries/pain	15.5 (3.8–66.7)	<0.001[a]	18.3 (3.9–87.5)	<0.001[a]
Plays other sports				
Any injury or significant climbing-related pain	0.6 (0.2–2.0)	0.56	–	–
Acute injury (at least 1)	1.0 (0.3–3.0)	0.96	–	–
Acute injuries (multiple)	1.1 (0.2–5.6)	0.89	–	–
Chronic injuries/pain	0.7 (0.2–2.3)	0.78	–	–
Safety skills taught by team				
Any injury or significant climbing-related pain	1.4 (0.2–9.1)	1.00	–	–
Acute injury (at least 1)	0.5 (0.1–3.1)	0.64	–	–
Acute injuries (multiple)	0.2 (0.2–1.4)	0.13	–	–
Chronic injuries/pain	3.4 (0.3–32.4)	0.38	–	–
Safety skills learned outside of team setting				
Any injury or significant climbing-related pain	4.1 (0.9–17.3)	0.07	–	–
Acute injury (at least 1)	2.0 (0.5–8.9)	0.48	–	–
Acute injuries (multiple)	3.9 (0.7–21.7)	0.126	–	–
Chronic injuries/pain	3.8 (0.7–20.2)	0.16	–	–
Use of hang board at home				
Any injury or significant climbing-related pain	3.4 (1.0–11.6)	0.07	–	–
Acute injury (at least 1)	1.2 (0.4–3.9)	0.78	–	–
Acute injuries (multiple)	4.6 (0.5–41.1)	0.22	–	–
Chronic injuries/pain	2.9 (0.9–9.4)	0.16	–	–
Warming up before practice				
Any injury or significant climbing-related pain	1.9 (0.1–32.9)	0.65	–	–
Acute injury (at least 1)	0.8 (0.9–1.1)	0.89	–	–
Acute injuries (multiple)	5.9 (0.3–99.4)	0.40	–	–
Chronic injuries/pain	0.8 (0.1–12.9)	0.89	–	–
Cooling down after practice				
Any injury or significant climbing-related pain	0.5 (0.2–1.6)	0.25	–	–
Acute injury (at least 1)	1.1 (0.3–2.9)	0.84	–	–
Acute injuries (multiple)	0.4 (0.1–2.3)	0.42	–	–
Chronic injuries/pain	0.5 (0.2–1.4)	0.26	–	–
Use of finger taping for injury prevention				
Any injury or climbing-related pain	2.5 (0.7–9.0)	0.23	1.4 (1.1–4.5)	0.02[b]
Acute injury (at least 1)	1.2 (0.4–4.0)	0.78	0.6 (0.2–2.1)	0.24
Acute injuries (multiple)	0.3 (0.03–2.5)	0.40	0.3 (0.03–3.3)	0.39
Chronic injuries/pain	5.1 (1.5–17.0)	0.01[b]	4.3 (1.2–15.2)	0.03[b]
Competing at higher than the recreational or local level				
Any injury or climbing-related pain	6.0 (1.7–21.3)	0.01[b]	2.0 (0.5–7.8)	0.33
Acute injury (at least 1)	3.2 (0.9–11.0)	0.07	0.5 (0.2–1.9)	0.33
Acute injuries (multiple)	0.4 (0.1–2.0)	0.25	1.6 (0.3–10.0)	0.62
Chronic injuries/pain	2.8 (0.7–6.8)	0.08	1.8 (0.5–6.7)	0.34

[a]designates P<0.01.
[b]designates P<0.05.

know the full impact of climbing-related epiphyseal injuries, nor do we understand the relationship between age-related developmental choices (eg, risk-taking behavior and peer influence) and their effects on injuries, so this could be an area to explore in further research.

This study attempts to elucidate the details of how returning to climbing while still injured affects climbing-related injuries. The reliance on self-reported data, small sample, and high likelihood of confounders limits broad interpretation, but given the persistent association between early return to climbing and increased injury risk and the larger effect size than expected seen in our study when controlling for exposure, this may still be clinically meaningful. Our study also begins to evaluate the athletes' own reasons for returning while in pain, and better understanding of the developmental psychology of these climbers may help influence how to best mediate these risks while discussing their specific fears and desires in an age-appropriate manner.

Because 91% of reported acute injuries occurred during bouldering, and bouldering difficulty also increased the risk of reported injury, these limited data suggest that bouldering itself may be an independent risk factor, perhaps owing to the dynamic forces involved in bouldering and the fall potential, and could be further evaluated in subsequent studies. Direct supervision of athletes appeared to decrease the risk of injury. These relationships represent an area of injury prevention to explore with more robust studies than ours and may help shape future training and competition planning.

Overall, the factors most associated with increased risk of reported injury when adjusted for confounders were returning to climbing while still injured or in pain, total number of hours climbed per year, unsupervised climbing, and use of finger taping as a preventative measure. Finger taping has previously been associated with increased injury risk,[6] and we speculate that climbers use the tape as a way to return to activity prematurely, resulting in an increased risk of chronic injury.

The factors associated with increased total number of reported injuries were bouldering difficulty, climbing unsupervised, and a higher number of climbing hours per session. Even when difficulty and supervision were adjusted for climbing exposure hours and athlete age, they remained significant.

LIMITATIONS

The sample size of our study is the most limiting factor, severely limiting the confidence and applicability of the study. Because of the distribution methodology, we are unable to determine how many climbers did not open the survey; the nonresponse rate only represents those who clicked the link but did not complete the survey. The survey link was posted online and sent to athletes by coaches, creating additional selection bias. The impact of these factors on our statistics and their generalizability is unknown but creates significant bias and difficulty in interpreting the data and associated statistics presented.

The timing of this study led to significant limitations as well. Surveys were sent out in February 2020, when the scope of the COVID-19 pandemic was not yet known. Data collection finished in April 2020. Lockdowns limited access to potential participants, and because only electronic responses could be obtained, internet access could have limited accessibility for survey completion.

This study relied on cross-sectional, retrospective, self-reported data, and thus establishing causal relationships is not possible. The design can only be used to give the odds of injury in relation to risk factors. Our small sample size produced large ORs with very wide CIs; acceptance of results at even the lowest limits of the CI should be done extremely cautiously.

Climbers and their parents were asked to remember minor injuries over 12 mo, introducing recall bias. The young age of the children included also may have led to reporting bias, even if the athletes' parents assisted. The older age of many of our participants is limiting as well: 20% of respondents were 18 y old, which limits application of the limited data gathered to the broader youth climbing community. Because coaches were involved in the distribution of the survey, athletes may have felt "pressured" to complete it, introducing another source of potential reporting bias. Exclusion of climbers who were not competitive in the past 12 mo may also have underestimated the number of severe injuries owing to sport dropout if these injuries were career-ending.

The injury data are also subject to self-diagnosis. The majority of respondents in our limited sample treated their injuries without the aid of a medical professional, and although attempts were made to objectively categorize injuries, self-report in a very small total number of respondents remains inherently limited, especially when using these data to calculate injury rates.

Conclusions

Our study adds data, albeit limited, to the growing body of literature on injuries in pediatric competitive climbers. The overall climbing injury rate was lower than previously reported, and repetitive overuse represented the most common mechanism. Bouldering and total climbing hours were risk factors for reported injury. Cumulative

climbing time may increase risk of injury. Hand/Finger injuries predominated reported injury types.

Returning to climbing while still in pain and the risk of injury appeared to be related as well. Understanding the young athlete's unique developmental psychology and identifying opportunities for injury prevention in this realm may represent an area of interest for future researchers to develop injury prevention techniques.

Further examination of the modifiable risk factors this small study suggest, such as early return to climbing, finger taping, discipline, difficulty level, climbing time, and supervision, may be useful to aid in the development of robust, unified injury prevention strategies. Larger, prospective studies can be performed.

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ORIGINAL RESEARCH

Kids With Altitude: Acute Mountain Sickness and Changes in Body Mass and Total Body Water in Children Travelling to 3800 m

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Introduction—We explored the incidence of acute mountain sickness (AMS) and extravascular lung water (ELW) in children in relation to changes in body composition and peripheral blood oxygenation (S_pO_2) during 1 week of acclimatization to 3800 m.

Methods—In a prospective cohort study, 10 children (7 female, ages 7–14 y) and 10 sex-matched adults (ages 23–44 y) traveled via automobile from sea level to 3000 m for 2 nights, followed by 4 nights at 3800 m. Each morning, body mass and body water (bioelectrical impedance), S_pO_2 (pulse oximetry), AMS (Lake Louise Questionnaire), and ELW (transthoracic echocardiography) were measured.

Results—No differences were found between children and adults in S_pO_2 or ELW. At 3800 m 7 of 10 children were AMS+ vs 4 of 10 adults. Among those AMS+ at 3800 m, the severity was greater in children compared to adults (5±1 vs 3 ± 0; P=0.005). Loss of body mass occurred more quickly in children (day 5 vs day 7) and to a greater extent (−7±3% vs −2±2%; P<0.001); these changes were mediated via a larger relative loss in total body water in children than in adults (−6±5% vs −2±2%; P=0.027).

Conclusions—Children demonstrated a higher incidence of AMS than adults, with greater severity among those AMS+. The loss of body water and body mass at high altitude was also greater in children, albeit unrelated to AMS severity. In addition to awareness of AMS, strategies to maintain body weight and hydration in children traveling to high altitudes should be considered.

Keywords: high altitude, maturation, physiology, pulmonary artery pressure, peripheral oxygenation, pediatric

Introduction

High-altitude travel that was once exclusive to mountaineers, explorers, and scientists is becoming increasingly popular with travelers. Families are taking annual holidays to resorts in Colorado (~2000–3500 m), trekking through the Himalayas (~2500–5000 m), and visiting high mountain villages in both the European Alps and the Andes (~2500–3500 m) on an ever-increasing scale. In each of these scenarios, children are more frequently accompanying their parents to altitudes up to and above 3000 m, yet decades of high-altitude research have focused on the physiological responses to low-oxygen environments almost exclusively in adults. Upon traveling to >3000 m, ~75% of adults have at least mild symptoms of acute mountain sickness (AMS), and a limited number of studies report either increased[1-3] or similar[4,5] incidence of AMS symptoms in children compared to adults; however, little is known about the integrative physiological acclimatization processes in children and the expression of AMS symptoms. The International Climbing and Mountaineering Federation medical commission consensus statement proposes that high-altitude travel is safe for children >14 wk old,[6] yet descriptions of symptoms and guidelines for safe daily ascent rates (ie, 300–500 m·day^{-1}) have been exclusively based on observations of adult trekkers. As such, there are limited

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scientific data on the related risks of traveling to altitude in children, so recommendations are broadly generalized and largely anecdotal.

In addition to differences in perception, stresses, and discomfort related to travel,[1] children demonstrate large developmental differences in physiological factors relevant to high-altitude acclimation; specifically, these differences in children are demonstrated in ventilatory,[7,8] autonomic,[9] and cerebrovascular regulation.[10-12] Collectively, these physiological differences may result in a phenotype in children that alters their acclimatization profile, both in magnitude and timing. Young children (6–9 y) display a larger increase in pulmonary artery systolic pressure than adolescents (14–16 y)[13] and adults after 18 to 40 h at 3450 m,[14] which may explain reports of an increased occurrence of high altitude pulmonary edema in young populations.[15-17] Nevertheless, no published data exist indicating whether these differences are maintained during periods of acclimatization or if they are preceded by increases in extravascular lung water. Renal excretion of bicarbonate enhances the ventilatory acclimatization to hypoxia in adults[18-20] but can lead to loss of body water and dehydration. Young children are generally at a greater risk for dehydration than adults,[21] which may be exacerbated during periods of travel and further increase the risk for AMS-like symptoms. Although peripheral blood oxygenation (S_pO_2) is a poor independent predictor of AMS, maturational differences throughout the oxygen transport cascade,[22,23] combined with differences in ventilatory, autonomic, and cerebrovascular regulation in children, may thereby influence the development of AMS and the overall tolerance of high-altitude travel.

The aim of this work was to explore high altitude travel tolerance in a group of children and adults during a passive, staged ascent to a research facility at 3800 m. We hypothesized that 1) children would be less tolerant of high-altitude than adults, based on symptoms associated with AMS and increased ELW; and 2) increases in symptoms of AMS in children would be associated with reductions in S_pO_2 and total body water.

Methods

This study was approved by the clinical research ethics board of the University of British Columbia, as well as the institutional review board at University of California Irvine. All experimental protocols and procedures conformed to the standards set by the Canadian government tri-council policy statement for integrity in research, as well as the Declaration of Helsinki, except for registration in a database. A detailed verbal and written explanation of the measurements was provided to participants and parents/guardians before participation. All participants and parents provided written, informed consent, and child participants provided written assent.

Ten children and adolescents (ages 7–14 y, 7 female) and 10 sex-matched adults (ages 23–44 y) participated in this study. None had any pre-existing diagnosed medical conditions. All participants lived <600 m above sea level and had not spent significant time (>1 h) above 2000 m in the 5 mo prior, confirmed by parents/participants. All children had at least 1 biological parent included in the adult group; 3 of the adults were not biologically related to any of the children but provided a sex match between the 2 groups.

This study was part of a larger expedition investigating cardiac, ventilatory, and cerebrovascular acclimatization to high altitude in children and adults. All participants traveled to Irvine, California, (25 m above sea level) for baseline testing. From Irvine, participants were driven ~8 h to the Crooked Creek Station (White Mountain Research Center, 3000 m above sea level), where they spent 2 nights acclimatizing. This stop at 3000 m was included to minimize the adverse physiological effects of rapid ascent to high altitude, help the children overcome any travel discomfort, and habituate both the children and adults to the testing at high altitude. The group was then driven to the Barcroft Field Station (White Mountain Research Center, 3800 m), where testing took place over the course of the next 5 d (Figure 1). All meals were provided by staff at each station and participants ate ad libitum. Caffeine and alcohol were restricted 12 h prior to testing, and participants were instructed to have light snacks only 4 h before testing. No acetazolamide or other altitude-related medications were taken by any of the participants before or during the trip. The team included researchers from 4 different institutions, 2 wilderness medicine physicians, parents of the children, and 3 individuals who served as activity coordinators for the children. Importantly, apart from self-determined vigorous exercise, there were no restrictions on activities for children or adults. Children were actively engaged in a wide range of games and activities throughout the trip, and participants were free to go on short hikes in the area surrounding the facilities after testing was completed each day. This model aimed to create an environment similar to other recreational family trips to higher altitudes.

At baseline and at the same time each morning before communal breakfast, measurements were taken of peripheral blood oxygenation, resting heart rate, bodyweight, and bioelectrical impedance for total body water. A questionnaire for AMS was also administered at this

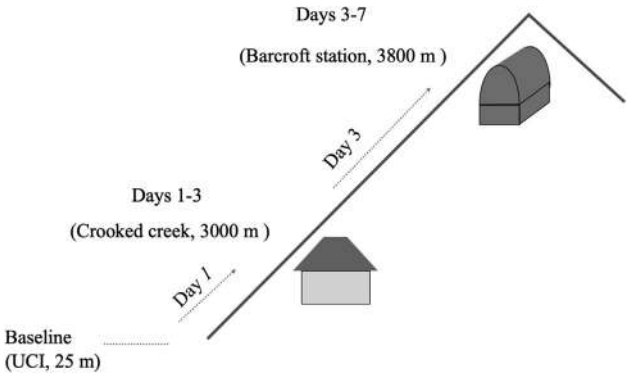

Figure 1. Chronological illustration of the trip timeline to the White Mountain Research Center. Baseline testing was performed at the University of California Irvine on the day before ascent.

time. In addition, on day 1 at Barcroft Station, the S_pO_2 and AMS measurements were repeated ~8 to 10 h after arrival.

Pulse oximetry (Rad-5, Masimo) was used to measure S_pO_2 and heart rate. Participants were seated upright in a chair for at least 2 min before measurement, and hands were warmed with an electric heating pad if necessary. S_pO_2 and heart rate are reported as the average of 3 recordings during a 15-s period once signals had stabilized.

Self-report of symptoms of AMS was recorded through a written 2018 Lake Louise questionnaire, with researchers present to ensure that the terminology used was clearly understood. The presence of AMS was identified by a headache score of at least 1 and by the 4 symptoms totaling a score of at least 3.[24]

Height and sitting height were measured with a portable stadiometer (Seca, Hamburg, Germany) during baseline testing at low altitude. Daily measurements of body mass and total body water were made using an electronic scale with foot-to-foot bioelectrical impedance (TBF-410, Tanita, Japan). For the children, the predicted offset to age at peak height velocity (ie, the predicted years prior to or past age at peak height velocity) was calculated as a marker of maturation.[25]

The presence of extravascular lung water was identified by examination of ultrasound lung comet artifacts at baseline, after 1 night at 3000 m, and at 2 additional points over 4 d at 3800 m (after 2–4 h, 3 nights). Bilaterally, 4 windows were imaged using a commercially available ultrasound machine (Vivid Q, GE, Fairfield, CT, USA) with an M5-S 1.5 to 4.6 MHz transducer. The number of B-line artifacts was manually counted from each image and recorded as a total comet tail score. The condensed 4-window view has been previously validated against the 8-window view for clinical purposes.[26]

The ActivPAL micro accelerometer (PAL Technologies Limited; Glasgow, Scotland, UK) was used to assess physical activity (steps per day). Participants wore the device on the anterior aspect of the right thigh, placed within a flexible nitrile sleeve and attached using Tegaderm Transparent Film (3M Health Care; St. Paul, MN, USA). Participants were asked to wear the device continuously for 7 d at baseline and throughout days 3 to 6 at high altitude. Day 7 was not included because this was spent being driven back to Irvine.

Descriptive statistics are presented as mean±SD. A Student's t-test was used to identify differences in descriptive characteristics at baseline. Analysis of the primary outcome variables (AMS symptoms, body weight, body water, S_pO_2) was performed using a 2-way repeated measures analysis of variation (factors: time and age group). Similar analysis was performed on a change score from baseline where applicable. When necessary, interactions and main effects were deconstructed using t-tests with Bonferroni correction. Linear regression was further used to test the relationships among weight change, AMS, and S_pO_2 in each group. Statistical significance was set a priori at $P<0.05$, and all analyses were performed using SPSS (Version 25, SPSS; Chicago, IL).

Results

Participant characteristics are presented in Table 1. As expected, adults were taller and heavier and had a higher percent body fat and a lower percent total body water ($P<0.05$ for all).

Upon arrival at 3000 m, total AMS scores were elevated compared to baseline in both children and adults

Table 1. Participant characteristics

	n	*F/M*	*Age range (y)*	*Age (y)*	*Height (cm)*	*Weight (kg)*	*Body fat (%)*	*Total body water (%)*
Children	10	7/3	7–14	9.8±2.5	141±15	34.5±9.8	15±7	60±4
Adults	10	7/3	23–44	34.7±7.1[a]	171±6[a]	65.9±11.8[a]	23±9[a]	56±6[a]

Values represent mean±SD.

[a]Significant ($P<0.05$) difference between children and adults.

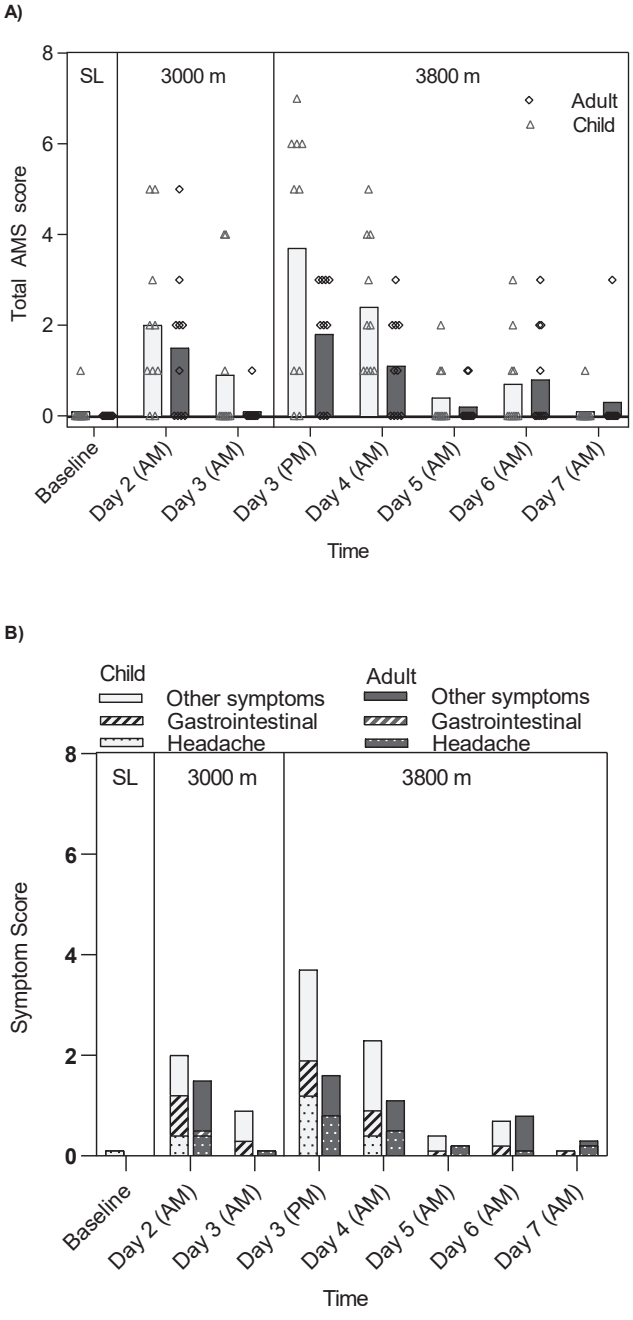

Figure 2. Acute mountain sickness score based on the 2018 Lake Louise scoring system in children and adults at sea level and at high altitude. (A) Total acute mountain sickness score. Significant main effects for age (P=0.006) and time (P<0.001) exist, but no interaction was found (P=0.131). (B) Symptom-specific scores. "Other" represents fatigue, dizziness, and clinical functional score.

(P<0.01) with no age by time interaction. A second increase in AMS score occurred in both groups on the evening of arrival at 3800 m (P<0.01), with a higher mean AMS score for the children, but this was not significant (4±1 vs 2±1, respectively; P=0.07). Importantly, although the sickest 3 adults each reported a total AMS score of 3 (mild AMS) on the first night at 3800 m, 6 of 10 children reported a total AMS score of 5 or greater (moderate AMS, Figure 2A).

Symptom-specific AMS scores are reported in Figure 2B. Headache, fatigue, dizziness, and functional scores were similar at all times between children and adults, but nausea-related gastrointestinal symptoms were more severe in children on day 2 AM (first morning at

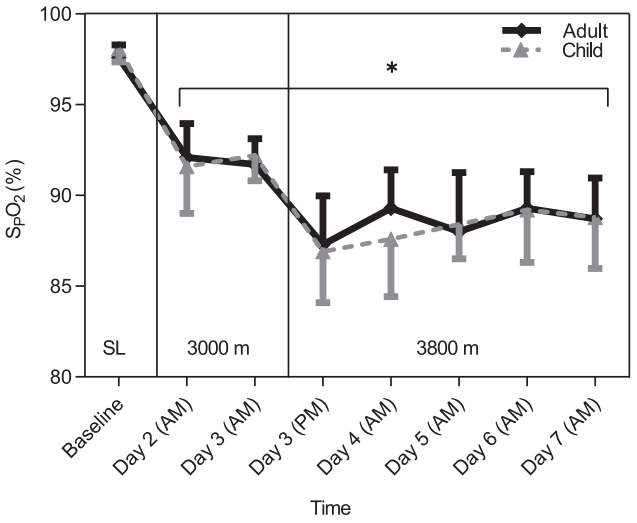

Figure 3. Peripheral oxygenation (S_pO_2 %) measured via finger pulse oximetry in children and adults during a 1 wk visit to high altitude. A significant main effect of time was observed ($P<0.01$), but not for group ($P=0.70$) or interaction ($P=0.86$). No difference between groups was present at any time. S_pO_2 was reduced at each altitude and tended to increase over the 5 d at 3800 m. *Significant difference from baseline ($P<0.05$).

3000 m, $P=0.005$), day 3 PM (+8 h after arrival at 3800 m, $P<0.001$), and day 4 AM (after 1 night at 3800 m, $P=0.004$). These differences subsided by the second morning at 3800 m.

Peripheral oxygenation was reduced with each increase in altitude (Figure 3), with a slow recovery (+2% in children, +1% in adults; $P=0.005$) over the 5 d spent at 3800 m. No age by time interaction was found, and there were no correlations between S_pO_2 and AMS on any of the testing days.

At all points, children had a higher resting heart rate than adults; however, no age by time interaction was found. Resting heart rate increased with each increase in altitude (Table 2), but there was no difference between groups in either the absolute change or percent change at any time.

Only a small number of lung comets (observed comet tail score range = 0–3) was identified in either group, with no differences between children and adults or across altitudes. The number of individuals with nonzero scores during each measurement is presented in Table 3. No individual displayed B-lines in more than 1 intercostal window.

For total body weight loss, an age by time interaction was found ($P<0.001$). In the children, a reduction (−2±2%, $P=0.02$) in total body weight relative to baseline was observed by day 5 of travel (3800 m), and this reduction continued through to the final day of high altitude (day 7, 3800 m), where total body weight was reduced by 8±3% ($P<0.001$) compared to baseline (Figure 4, Panel A). In contrast, body weight was maintained within a much narrower range in the adults, where a significant change from baseline was only observable on the final day at high altitude (−2±2%; $P=0.03$). These changes appear to be driven by a larger reduction in total body water in children (Figure 4, Panel B), who had lost a significantly greater fraction of total body water by the final day of high altitude compared to adults (−7±5% vs −2±2%, respectively; $P=0.03$). In both children and in adults, no relationship was found between AMS and change in body weight or between AMS and change in body water on a day-to-day basis or over the course of the entire week.

Steps per day are reported in Table 4. To provide context to these data, day 3 included the drive from the Crooked Creek station to Barcroft Station; participants completed testing on days 3, 4, and 6, and there was no testing on day 5. Participants instead went for a hike in the local area. The children achieved more daily steps

Table 2. Resting heart rate (beats·min^{-1}) over the course of 1 wk at high altitude

	Baseline	*Day 2*	*Day 3 (AM)*	*Day 3 (PM)*	*Day 4*	*Day 5*	*Day 6*	*Day 7*
Children	92±11[a]	101±11[a]	97±13[a]	103±14[a]	99±16[a]	96±13[a]	89±16[a]	90±15[a]
Adults	73±24	78±23	79±21	82±24	76±20	74±76	76±24	77±23

Values represent mean±SD.

[a]Significant difference ($P<0.05$) between children and adults.

Table 3. Individuals demonstrating a positive identification of ultrasound lung comets (score >0)

	Baseline	*Day 2*	*Day 3*	*Day 6*
	Sea level	*3000 m*	*3800m*	*3800 m*
Children	1/10	0/10	1/10	0/10
Adults	1/10	1/10	1/10	1/10

than the adults at baseline and day 4 (Group; P=0.02, Table 4); however, analysis of the change from sea level baseline indicates no differences in the pattern or magnitude of change in steps per day at high altitude (P=0.49).

Discussion

In this study, we observed subjective symptoms and objective physiological acclimatization in children and adults over a week-long stay at high altitude, including 5 d at 3800 m. We report no differences in peripheral oxygenation or extravascular lung water at any time between the children and adults; however, the incidence of AMS was greater in children upon arrival at 3800 m; children also experienced a significantly greater loss in total body water and total body weight by day 6 of high altitude. No relationship between either S_pO_2 or extravascular lung water and AMS was found on any of the days.

Initial ascent to 3000 m elicited a comparable mean AMS score for children and adults. Despite no significant difference in the mean scores of AMS at this altitude, pooled data from the 2 mornings indicate that only 2 of 10 adults were AMS+ (total score ≥3, including headache) at the first station, whereas 5 of 10 children were AMS+ on at least 1 of the first 2 mornings. Subsequently, upon ascent to the upper station, total mean AMS score tended to be greater in the children (P=0.07); however, the current investigation was likely underpowered to detect significant differences. When the first night and first morning at 3800 m were both considered, 7 of 10 children were AMS+ vs only 4 of 10 adults. Furthermore, among those who were AMS+, the severity was worse in the children (5±1) compared to the adults (3±0; P=0.005). The increased incidence and severity of AMS observed in children was apparent after each increase in altitude and potentially was even underreported based on unmeasured observations of acute "waves" of sickness in the children in between structured times of testing. For example, 1 girl spent several hours (under close observation) with peripheral saturations between 68 and 75% during night 1 at 3000 m but woke up and reported a score of 0 on the Lake Louise questionnaire the next morning. It should be noted that her parents reported her as susceptible to snoring at night, and her peripheral oxygen saturation improved immediately once she transitioned from a fetal to a supine position and her airway was opened using a head tilt and chin lift maneuver. In multiple other cases, children became acutely incapacitated for several hours at a time with nausea and headaches, but they were subjectively fine by the time the next self-report questionnaire was submitted.

Although we provide evidence of increased AMS incidence within our cohort, previous reports comparing AMS in children and adults remain largely inconclusive. For example, in young children ages 6 mo to 5 y, the incidence of AMS was elevated compared to adults upon arrival at 3500 m; however, no difference was observed between teenagers (ages 13−17 y) and adults.[3] In a larger study composed of 20 father-child pairs (children aged 9−12 y), no difference in the cumulative incidence of AMS was reported; however, differences in the severity of symptoms were not considered.[5] Children have primarily shown similar[27-29] or increased incidence of AMS[2,4,30-32] compared to adult control groups. However, to our knowledge only 1 study has shown a reduced susceptibility in AMS in children, after rapid ascent via train to 3500 m.[33]

The current definition of AMS requires at least a mild headache, and we demonstrated comparable prevalence and severity of headache between children and adults; however, the presence of increased gastrointestinal symptoms in children without an increased headache score suggests that the etiology of high-altitude illness in children may present differently than in adults. Evidently, even the process of being away from home may trigger symptoms associated with AMS in children. In a study of 9- to 14-y-olds (n=405) attending summer camp at sea level in California, 21% reported symptom scores equating to a positive diagnosis of AMS, compared to 28% of similar-aged children who were AMS+ at an actual high altitude (2835 m) summer camp in Colorado.[1] In the current investigation, all children had at least 1 parent present, which may reduce some of the distress associated with being away from home; however, it is possible that maturational differences exist in the perception and reporting of AMS symptoms. Despite this, similar scores were reported between groups at baseline, after families had already spent 1 night in a hotel at sea level.

Ascent to high altitude can be associated with a loss in body weight owing to a combination of high altitude-induced anorexia[34] (and thus decreased energy intake), dehydration, increased physical activity energy

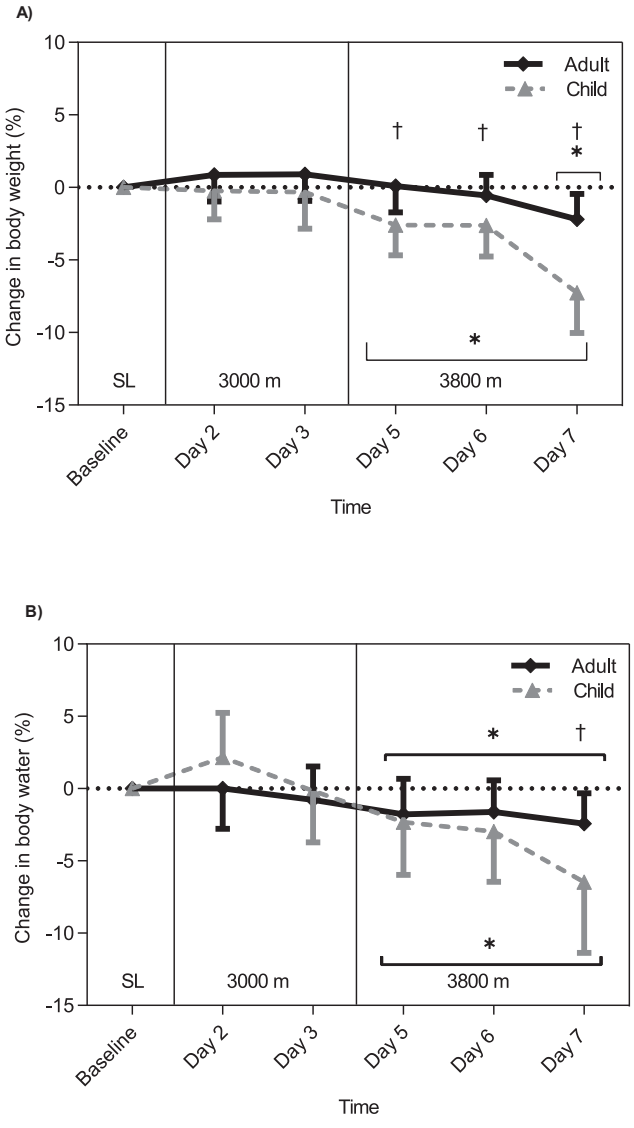

Figure 4. (A) Change in body weight and (B) change in body water during a 1-wk visit to high altitude in children and adults. Values represent mean±SD. *Significant difference from baseline ($P<0.05$). †Difference between groups ($P<0.05$).

expenditure, and increased metabolic rate.[35-37] We found that despite an abundance of available food and drink, total body weight started to decline earlier in children compared to adults and to a larger degree. Specifically, by the final day at the upper station, children had lost 8% of their total body weight, vs 2% in the adults. Importantly, this greater reduction in body weight in children was driven largely by a reduction in total body water, with a relative water loss over 2 times greater than that of adults, which may also be in part related to children having a higher mean total body water percent compared to adults.[38] Although water intake was not

Table 4. Daily step count at baseline and over the first 4 days at 3800 m

	Baseline	*Day 3*	*Day 4*	*Day 5*	*Day 6*
Children	16,787±3038	13,174±3863	16,755±4133	23,037±5789	15,777±7365
Adults	9470±2400[a]	10,258±2454	11,095±1612[a]	21,180±7219	12,179±3454

Values represent mean±SD.
[a]Between-group difference ($P<0.05$).

measured, reports of inadequate water consumption are common in children,[39,40] with the potential for even greater disruptions to water balance when traveling. On this trip, all participants were constantly prompted to have water with them at all times and were actively reminded to drink to ensure adequate fluid intake and avoid body water loss[41]; however, voluntary drinking does not always guarantee adequate hydration.[42] Although renal function reaches maturity around 2 y of age based on glomerular filtration rates and urine concentrating and diluting capacity,[43] it is not known whether maturational differences in bicarbonate excretion exist during acclimatization, which would have a profound impact on overall fluid status. Furthermore, at baseline, the children would be expected to have a 5 to 10% lower hemoglobin concentration compared to the adults.[44] The greater loss in body water observed in children may in part be related to a larger reduction in plasma volume to increase arterial oxygen content through hematoconcentration.

Children are also at a greater risk of travel-related diarrhea than adults.[45] We do report higher scores on gastrointestinal symptoms in children compared to adults; however, the current questionnaire only specifically asks about nausea and vomiting, and statistical differences between groups did not persist for the entire length of stay at 3800 m. We cannot determine if a higher propensity for travelers' diarrhea in children could have been a cause of the larger change in body composition and total body water compared to adults, or if it is simply an independent additional risk that may exacerbate the complications associated with dehydration.

For the remainder of the total weight loss, we are unable to determine if this was due to decreased appetite and caloric intake in children, which may be related to the increased gastrointestinal symptoms in children, or due to a larger increase in energy expenditure. Our indicators of energy expenditure and metabolism, step count and heart rate, showed no age-related difference in the pattern of change from baseline, suggesting that the greater loss of body weight in children may be related to reductions in energy intake rather than expenditure. Additionally, we used a single-frequency (50 kHz) bio impedance device for estimating body water. This high frequency is well suited to estimating intracellular, but not extracellular, water content.[46] Body water analysis using low and high frequencies can estimate both intracellular and extracellular water compartments and therefore provide a more precise assessment of total body water. Future studies in children should consider compartmental body water measures to properly quantify and identify changes in fluid balance, as well as measurement of food intake, metabolic rate, and related satiety hormones.

With reductions in size and improvements in portability of ultrasound technology, the identification of lung comet tails is a simple and robust way to detect extravascular lung water.[47,48] Based on reports of the increased prevalence of high altitude pulmonary edema in children,[17,49] as well as observations of an increased pulmonary artery systolic pressure in children at high altitude relative to adolescents and adults,[13,14] we hypothesized that children would also have a larger lung comet tail score than adults at high altitude. Our hypothesis was not supported. Although high altitude pulmonary edema is a very rare condition with a prevalence proportional to the rate of ascent and the altitude achieved, affecting less than 1[50] to 16%[51] of adults traveling between 2500 m and 5500 m, our findings suggest that the development of extravascular lung water can be attenuated using a cautious ascent profile while traveling to moderate-high altitudes, albeit in a small sample of individuals. Furthermore, the absence of prolonged, strenuous exercise (ie, passive ascent) likely limited the increase in pulmonary artery pressure that would contribute to the development of extravascular lung water.

Despite differences in the development of AMS between children and adults, there were no differences in S_pO_2 at any point throughout the trip. During acclimatization to high altitude, a progressive increase in alveolar ventilation increases the arterial pressure of O_2 and S_pO_2 over time, and this slow recovery of S_pO_2 at the upper station was observed (and similar) in both groups. Importantly, this recovery of S_pO_2 indicates that ventilatory acclimatization is indeed likely happening at a similar rate in children and adults. Furthermore, in both groups, there was no relation between S_pO_2, change in S_pO_2, and any symptom of AMS, ELW, or change in body weight on or between any days. We acknowledge that the cause of AMS is much more complex than can be explained by S_pO_2 alone.[52]

LIMITATIONS

This work has limitations. It describes a very small sample size, across a broad maturational range and mixed sex. There is no questionnaire for AMS specifically for children (with the exception of pre-verbal children[53]) or adolescents, so differences in perception and self-reporting of symptoms between children and adults could have influenced the AMS scores. Our single-frequency measure of body water was limited, and we did not measure energy intake or water intake or have any direct measure of daily hydration status.

Conclusions

During a stepped and passive ascent to high altitude, children, like adults, became symptomatic with acute increases in altitude, desaturated to a similar degree, and showed improvement with increasing time spent at altitude. Children became sick more frequently than adults, and children who developed AMS had more severe symptoms than adults with AMS and significantly more gastrointestinal-related symptoms. Importantly, weight loss at high altitude occurs sooner in children and occurs to a greater degree, driven largely by a loss in total body water. Awareness of AMS and strategies to maintain body weight and hydration are important for children traveling to high altitudes.

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ORIGINAL RESEARCH

Search and Rescue in the Pacific West States

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Introduction—The training practices and the level of medical oversight of search and rescue (SAR) organizations in the US National Park Service (NPS) Pacific west region is not known.

Methods—A database of SAR teams in the NPS Pacific west region was assembled using public sources. SAR team leaders received an electronic survey between May and December 2019. A descriptive analysis characterizing team size, technical and medical training protocols, and medical oversight was completed. Results are reported as median (interquartile range, range).

Results—Of the 250 SAR teams contacted, 39% (n=97) completed our survey. Annual mission volume was 25 (10–50, 1–200). Team size was 30 members (22–58, 1–405). SAR teams most frequently trained in helicopter operations (77%), low-angle rope rescue (75%), and avalanche rescue (43%). Nearly all teams (99%) had members with some medical training: first aid or cardiopulmonary resuscitation (89%), emergency medical technicians (75%), registered nurses or midlevel providers (52%), and physicians (40%). SAR members administered field medical care (84%), often in coordination with EMS (77%). Medical direction was present on a minority of teams (45%), most frequently by a provider specialized in emergency medicine (68%). Expanded medical procedures were permitted on 21% of SAR teams.

Conclusions—SAR teams across the NPS Pacific west region had composition and training standards similar to those surveyed previously in the US intermountain states. Healthcare professionals were present on most teams, typically as team members, not as medical directors. Few SAR teams use medical protocols in remote care environments.

Keywords: SAR, wilderness medicine, first responder, National Park Service, EMS

Introduction

Search and rescue (SAR) operations in the United States occur under the auspices of multiple agencies, and to the authors' knowledge there are no unified protocols for how SAR teams are organized or operate. They are most frequently housed within county sheriff's offices, although this varies by state. Teams may have affiliations with federal, state, or local government, and unaffiliated, nonprofit groups also exist. Although numerous reports have characterized the demands of National Park Service (NPS) SAR teams, peer-reviewed literature capturing the diversity of US SAR teams outside of the NPS system is limited.[1-3] The only publication to date primarily evaluating non-NPS SAR organization, training, and mission volume is a regional analysis of the intermountain west SAR services completed in 2013.[4] Our objective was to further characterize the prehospital SAR response system, with an analysis of SAR teams in the contiguous NPS Pacific west region states.

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Methods

We assembled a database of SAR teams within the NPS Pacific west region using public Internet sources. This database included all identifiable SAR teams in the states of California, Idaho, Nevada, Oregon, and Washington, organized by both state and county. We searched local government webpages, including those of sheriff's offices, county clerk's offices, county and regional SAR

Table 1. Team leader survey

	Survey question	*Multiple-choice/Free-text response*	
1.	Which type of search and rescue does the SAR team that you are team leader of primarily specialize in? (select the most applicable)	Mountaineering/Ground search and rescue unit (including technical rope rescue) Maritime search and rescue unit (including dive rescue) Mounted posse search and rescue unit K9 search and rescue unit	Air Patrol search and rescue unit off Highway vehicle search and rescue unit Urban/Disaster search and rescue unit Cave search and rescue unit Combat search and rescue unit
2	What is your team's average annual SAR call volume?		
3	What is the number of volunteer team members on your SAR team?		
4	What is the number of paid team members on your SAR team?		
5	Does your SAR team require medical certification or licensing prior to joining?	Yes	No
6	What medical certification or licensing does your SAR team require prior to joining? (select all that apply)	First aid/CPR Emergency medical responder (EMR) Emergency medical technician (EMT) Advanced emergency medical technician (AEMT)	Paramedic Other—Please specify the certification or licensing requirement.
7	Does your SAR team provide medical training for team members?	Yes	No
8	What medical training does your SAR team provide for team members? (select all that apply)	First aid/CPR EMR EMT	AEMT paramedic Other—Please specify the medical training provided.
9	Please select the medical certifications or licenses that any of your team members have achieved. (select all that apply)	First aid/CPR—how many? EMR—how many? EMT—how many? AEMT Paramedic—how many?	Nurse (RN)—how many? Nurse practitioner (NP)—how many? Physician assistant (PA)—how many? Physician (MD or DO)—how many?
10	We are conducting a separate survey of physicians on SAR teams. Are you willing to provide email contact for your team physician(s) for this purpose?	Yes	No
11	If yes, please input the email address for your team physician(s).		
12	How many of these medical providers also have formal wilderness medical certifications?		
13	Please select all skill sets that your team members receive formal training in.	Swift water rescue Dive rescue Avalanche rescue High-angle rope rescue Low-angle rope rescue	Cave/Confined space rescue Helicopter operations Mechanized vehicle operations Incident command system Other—Please specify the skill training provided

(continued on next page)

Table 1 *(continued)*

	Survey question	*Multiple-choice/Free-text response*	
14	During SAR operations, who provides patient medical care?	SAR team members Local EMS	Other—Please specify your patient care providers during SAR missions.
15	Are your local EMS providers cross training in SAR operations?	Yes	No
16	Does your SAR team have a medical director?	Yes I don't know what a medical director is	No
17	Is your medical director a physician?	Yes I don't know	No
18	Which of the following specialties does your medical director belong to?	Emergency medicine Family medicine Internal medicine Surgery	Other—Please specify your medical director's specialty. I don't know
19	Please indicate all of the following activities in which your medical director participates.	Create/Update medical protocols SAR team training activities SAR missions Medical treatment consultation	Prescribing medications for use in field-based patient care Other—Please specify the activities your medical director participates in.
20	Various state and national agencies outline the skills that can be performed by different levels of medical providers. Do your local SAR medical protocols allow for expanded medical scopes of practice given the remote nature of SAR missions? (For example, medication administration, dislocation reduction, intravenous access, supraglottic airway use, etc.)	Yes I don't know	No
21	Please describe the specifics for your expanded protocols.		

SAR, search and rescue.

webpages, and Facebook pages, to identify publicly listed email addresses for SAR team leaders. Search terms included "county name" followed by "search and rescue," "SAR," "mountain rescue," and "sheriff's posse." When a publicly listed team leader or point-of-contact was not available via web search, we made telephone calls to the parent organization, typically a sheriff's office or office of emergency services, to locate an appropriate email or phone contact. When a SAR organization with multiple specialty teams was encountered (eg, mountaineering team, K-9 team, dive team), each specialty team was considered unique and was represented as such in our database. Identifying information for each SAR team in the database included state, county, parent organization, subspecialty, email contact, and phone contact. After the database of SAR team leaders was compiled, each was sent an initial survey by email (Table 1). After this initial distribution of the team leader survey, we made telephone calls to all nonresponders through directly listed phone numbers that had been previously identified or by contacting the parent organization. Before closing data collection, a final email was sent to each nonresponder. Through this method, up to 3 contact attempts were made for each of the 250 teams identified in our region of interest.

The team leader survey consisted of general questions regarding the specialty of the SAR team, annual call volume, volunteer and paid member count, and specific questions regarding the medical training of members and the role of medical specialists on the team (Table 1). Data collection took place between May and December 2019, with entries cataloged in the research electronic data capture (REDCap) application. Results were both quantitatively and descriptively analyzed using Microsoft

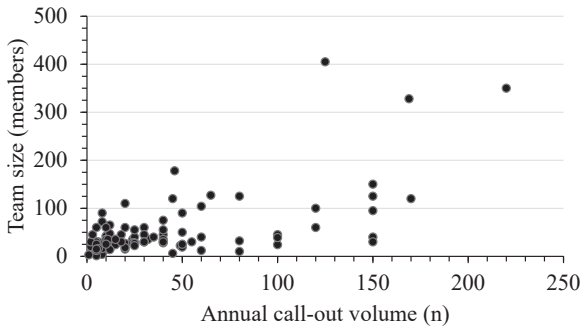

Figure 1. Annual call volume relative to team size. Data are presented using each reporting search and rescue team as an independent point.

Excel. Quantitative analysis included mean±SD or median (interquartile range, range) for team size, call volume, number of volunteers with specific medical training, and the proportion of respondents answering in the affirmative for our categorical questions (eg, proportion of teams requiring members to be trained in first aid). Descriptive analysis was required for free-text responses, including questions regarding training provided by SAR teams and types of expanded medical protocols permitted under medical direction. The University of California San Francisco, Fresno, institutional review board approved the study design and protocol.

Results

Of the 250 SAR teams contacted, 39% (n=97) completed our survey. Response rates by state were as follows: California (44%, n=33/75), Oregon (44%, n=24/54), Nevada (38%, n=3/8), Washington (36%, n=29/81), and Idaho (25%, n=8/32).

Team types included mountaineer (66%), off-highway vehicle (7%), K9 (7%), maritime (4%), mounted posse (4%), air (3%), cave rescue (2%), urban (1%), and unspecified (5%). Median annual mission volume was 25 (10–50, 1–220), and median size was 30 volunteer members (22–58, 1–405) and 0 paid members (0–1, 0–25). Large teams were outliers in the data set; only 3 teams were composed of over 300 volunteer members, and 8 teams reported over 100 volunteer members (Figure 1). The remaining teams (85%) reported having fewer than 100 volunteer members. Technical teams were trained in helicopter operations (78%), high-angle rope rescue (62%), avalanche rescue (44%), swift water rescue (41%), and dive rescue (14%) (Figure 2).

Among the SAR teams in the NPS Pacific west region that responded to the survey, one-third (34%) required members to have minimum medical training, with first aid or cardiopulmonary resuscitation (CPR) being the most common requirement (29%). Medical training for members was provided by 85% of teams; first aid or CPR was the most common (78%) training provided. Medical training included emergency medical responder (20%), emergency medical technician (EMT) (14%), advanced EMT (AEMT), or EMT paramedic (EMT-P) (4%), and "other" (19%), which included EMT recertification for members who had lapsed EMT certifications, tactical casualty combat care (TCCC), prehospital trauma life support, automated external defibrillator use, wilderness first responder, wilderness first aid, and advanced wilderness life support.

The overwhelming majority of teams (99%) had medically trained members: first aid or CPR (89%); EMT, AEMT, or EMT-P (75%); registered nurses, nurse practitioners, or physician assistants (52%); or physicians (40%) (Table 2). The number of members per team with each type of medical training is shown in Figure 3. Members with wilderness medicine training were present in most teams (57%, n=56). The median number of members per team with completed wilderness medicine training was 2 (0–7, 0–120).

SAR team members provided direct patient care on the majority of responding teams (84%, n=81/97). Local EMS were also involved in direct patient care on 77% of SAR teams. Other organizations involved in direct patient care included air ambulance and air medics. Only 23% of teams reported joint training with local EMS for rescue scenarios (n=22).

Of the 97 responding teams, 45% reported having SAR medical directors (n=44). These individuals were mostly physicians (77%, n=34) and trained in emergency medicine (68%, n=25) or family medicine (15%, n=5).

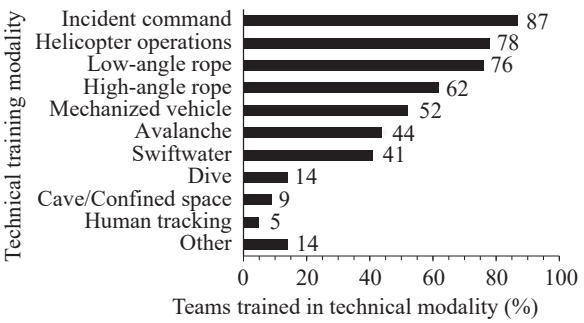

Figure 2. Percentage of teams that trained members in specific modality. The majority of teams reported training members in more than 1 discipline. Free-text responses for other included K9 search, human tracking, equine handing and packing, open water/surf rescue, GPS/compass use, orienteering, and urban rescue.

Table 2. The presence of medically trained members on SAR teams

Training level	*All*	*CA (n=33)*	*WA (n=29)*	*OR (n=24)*	*ID (n=8)*	*NV (n=3)*
First aid/CPR	86 (89)	28 (85)	25 (86)	22 (92)	8 (100)	3 (100)
EMT-B	73 (75)	28 (85)	20 (69)	16 (67)	6 (75)	3 (100)
WFA/WFR	56 (57)	21 (63)	15 (51)	15 (62)	4 (50)	1 (33)
RN	50 (52)	20 (61)	13 (45)	12 (50)	3 (38)	2 (66)
MD/DO	39 (40)	20 (61)	10 (34)	8 (33)	1 (13)	–
EMT-P	39 (40)	15 (45)	13 (45)	10 (42)	1 (13)	–
EMR	38 (39)	14 (42)	8 (28)	10 (42)	4 (50)	2 (66)
PA	16 (16)	7 (21)	4 (14)	4 (17)	1 (13)	–
AEMT	16 (16)	4 (12)	6 (21)	4 (17)	1 (13)	1 (33)
NP	9 (9)	4 (12)	1 (3)	2 (8)	2 (25)	–

Data are presented as the number and percentage of teams per state that reported having at least 1 team member with given certification/education. AEMT, advanced EMT; CPR, cardiopulmonary resuscitation; EMR, emergency medical responder; EMT-B, EMT-basic; EMT-P, EMT-paramedic; MD/DO, physician; PA, physician's assistant; RN, registered nurse; WFA, wilderness first aid; WFR, wilderness first responder.

Forty-four teams reported having a medical director who was typically involved in updating medical protocols (84%, n=37), training with the SAR team (69%, n=29), and participating in SAR missions (50%, n=22). When available, the SAR medical director participated in medical consultation on 56% of reporting teams (n=25) and prescription of medications for use in field-based patient care on 23% of teams (n=10).

SAR medical protocols allowed for expanded scope-of-practice on 21% of reporting teams (n=20), permitting members to perform medical care that exceeded individual team member credentialing. Dislocation reduction (n=14) and epinephrine administration (n=8) were most frequently reported, but SAR teams also had expanded protocols for airway stabilization (n=2), establishing intravenous access (n=2), and analgesia administration (n=1).

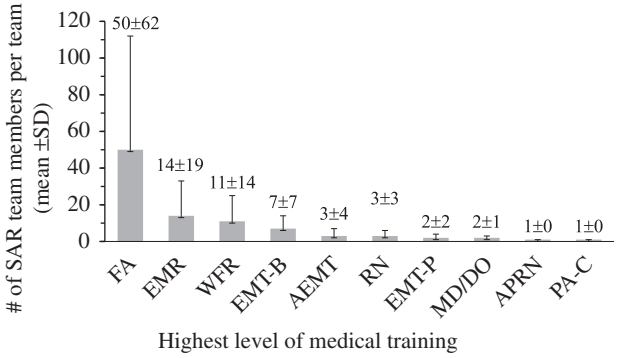

Figure 3. Number of individuals on reporting teams (mean±SD), categorized by level of medical training. FA, first aid; WFR, wilderness first responder; EMR, emergency medical responder; EMT-B, EMT-basic; AEMT, advanced EMT; EMT-P, EMT-paramedic; RN, registered nurse; MD/DO, physician; APRN, advanced practice registered nurse; PA-C, physician's assistant.

Discussion

The majority of teams responding to our survey of NPS Pacific west region SAR organizations were relatively small (<50 members). They typically trained for a diverse range of rescue scenarios and basic first aid care, similar to teams operating in the US National Parks and Canadian National Parks.[1,5,6] Although a large proportion of organizations had professional medical providers among volunteer members, far fewer reported having physicians (MD or DO) as team members or serving as established medical directors. Respondents reported frequent coordination with local EMS, but joint training experiences were more limited. These findings suggest that opportunities to improve care in remote and austere environments where long transit times are anticipated could be achieved through expanded medical oversight of responding SAR teams and improved coordination of care with EMS.

The results of our survey among NPS Pacific west region SAR teams were largely consistent with the earlier findings of the intermountain region survey.[4] Although these surveys were not contemporaneous, SAR teams that responded to our survey had slightly higher rates of minimum medical training among volunteers (basic first aid or CPR) compared to responding teams in the intermountain region (89 vs 79%). Our responding teams were more likely to report having members with wilderness medical training, including wilderness first aid, wilderness first responder, or advanced wilderness life support (57 vs 23%). This difference may be due in part to growth in the availability and popularity of wilderness medicine training in the past decade. Based on anecdotal experience, we think that opportunities for training in wilderness medicine have increased, but we are not aware of any studies that support this perception. The NPS

intermountain and Pacific west surveys reported that teams operated under established medical direction at similar rates (45 vs 41%). In both regions, medical directors most often specialized in emergency medicine. Expanded medical protocols were more common among intermountain teams (45 vs 21%), which may reflect a need that accompanies longer transport times in more remote areas of the intermountain region.[5,7,8] Local EMS was involved in care during most missions in both regions; however, there were low rates of EMS/SAR joint training in both regions (35% NPS intermountain and 23% NPS Pacific west regions).

The need for medical care during SAR missions varies greatly by mission.[5,7] Reviews of EMS in NPS units found that the acuity and severity of injuries in the field are generally low, suggesting that using volunteers, as seen in the NPS Pacific west region, is likely appropriate.[2,3,9,10] Volunteers with more advanced medical training, including physicians, often chose to limit their practice to first-aid level interventions; in other cases, scope of practice was restricted by the SAR parent agencies.

Although acuity of illness and injury in the wilderness setting is typically low, examples of expanded medical protocols being implemented in the field by civilian SAR teams show that these interventions are feasible and can play an important role in stabilizing critically injured patients when transit times are prolonged.[11] Standardized expanded medical protocol models exist in both military and NPS wilderness settings and have been shown to save lives in the prehospital environment.[12] The TCCC model uses tourniquets, hemostatic agents, nasopharyngeal airways, analgesia, and antibiotics in the battlefield.[12] The NPS Parkmedic program is similar, training first responders as AEMTs and expanding scope of practice through additional procedural and pharmacologic training.[3,13] The additional training with offline medical protocols and online support via radio communications with local emergency departments allows Parkmedics to provide an expanded level of care in the backcountry during SAR missions, without requiring the presence of advanced providers on site. Parkmedic and TCCC protocols are used by well-trained professionals (EMTs and soldiers, respectively), but there are more manuals and guidelines for expanding medical protocols under medical direction in civilian EMS and SAR systems. The Washington State Department of Health prolonged prehospital emergency care course and the International Commission for Mountain Emergency Medicine multiple trauma management guidelines promote evidence-based practices for civilian rescuers in remote environements.[14,15] Similarly, the Wilderness Medical Society (WMS) has clinical practice guidelines for current, evidence-based care in remote environments.[16]

The adoption of evidence-based, expanded medical protocols similar to those described by WMS and other sources may be limited by the scope of practice under which US first responders have historically operated. The results of our survey suggest that within the NPS Pacific west region, basic first aid is commonly employed by SAR volunteers until EMS can be reached and the patient transported to definitive care. Advanced life support may be delayed when teams operate without expanded medical protocols. The results of a 2018 study conducted in the central Pyrenees suggested that providing advanced life support in the field when transport times are prolonged may improve patient outcomes. After the addition of physicians and nurses to provide advanced prehospital care to mountain rescue teams, there was a significant decrease in mortality rate during SAR missions (9 to 3%). The author predicted cost savings over the next 15 y to be in excess of $203 million per year.[17] A similar professionalization is not currently feasible in the United States for numerous reasons, including relatively low population density and inadequate funding for similar interventions. However, many NPS Pacific west region SAR teams have advanced providers. The intervention in the Pyrenees supports the notion that providing advanced care in prehospital settings might decrease mortality.

Our survey found that there was limited medical director oversight among NPS Pacific west region SAR teams. Few teams reported having expanded medical protocols for their volunteer members. The experiences of TCCC, NPS Parkmedics, and mountain rescue organizations in the Pyrenees suggest that that addition of medical protocols suited to the unique environment might promote the provision of advanced prehospital care. This view aligns with the National Association of EMS Physicians position statement.[18] Teams can use existing clinical practice guidelines, such as those provided by the WMS or by local and state jurisdictions to expand prehospital emergency care.

LIMITATIONS

Our survey had a response rate of only 39%. The intermountain region survey had a higher response rate at 56%.[4] This is a major limitation that provides significant room for error in our characterizations. The research team was unable to identify specific patterns for nonresponse to our survey, which may represent a flaw in our methods for collecting SAR team leader contact information. Our survey assumes that SAR team leaders have the resources and time to maintain accurate and current records about their active membership, which may not be the case in all circumstances. The validity of the data collected in our survey depends on respondents being specific in their

responses. Our survey relied on free-response fields for respondents to describe expanded medical protocols. This presents opportunities for mischaracterization or misinterpretation of responses by the research team. Survey methodology is subject to bias. Survey respondents may misinterpret questions, enter data incorrectly, or be subject to nonresponse bias, which occurs when people are unwilling to respond to a survey owing to a factor that makes them differ greatly from people who respond.

This survey only queried organization and training of SAR teams and did not study mission-specific demands or outcomes. We did not collect data on the medical certifications of rescuers on specific missions. This limits discussion of mission-specific outcomes before and after implementation of expanded medical protocols.

Conclusions

SAR organizations in the NPS Pacific west region were typically trained for a variety of rescue scenarios according to local geography, including high-angle rope rescue, whitewater rescue, and avalanche rescue. The composition and training standards of teams was similar to the composition and training standards of teams in the intermountain region. Fewer SAR teams in the NPS Pacific west region had medical directors compared to teams in the intermountain region, despite the presence of medical professionals as members. Few SAR teams in the NPS Pacific west region used expanded medical protocols. Clinical practice guidelines and models are available to teams that wish to expand medical protocols under appropriate medical direction.

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ORIGINAL RESEARCH

Foot-Launched Flying Sport Fatalities in the Canton of Berne, Switzerland

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Introduction—Foot-launched flying sports such as paragliding, hang gliding, and speedflying are popular recreational activities that all pose a significant risk of accidents resulting in injuries or fatalities. We describe the epidemiology, incident circumstances, and findings of forensic examinations of fatalities in these sports.

Methods—In this retrospective analysis, we analyzed different parameters of paragliding, hang gliding, and speedflying fatalities in the canton of Berne, Switzerland, between 2000 and 2018. The data sources were police reports, forensic medical reports, the Swiss Hang Gliding Association, and the Swiss Council for Accident Prevention.

Results—In the given period, 40 incidents resulting in 42 fatalities were recorded (2 incidents involved 2-seaters). Three of the 40 incidents did not fulfill the inclusion criteria and were excluded. The deadliest phase was midflight. Collapse of the glider (n=9; 36%) was the leading cause of accidents among paraglider pilots. Multiple trauma (n=21; 54%) was the most frequent cause of death. In the forensic examination, all deaths (n=39) were assessed as accidents.

Conclusions—The different categories of foot-launched flying sports varied in the causes of accidents and deaths. Fatalities in speedflying involved young pilots exposing themselves to high-risk situations. Fatalities may be prevented by enhancing education and training and promoting a cautious attitude among pilots.

Keywords: adventure sports, paragliding, hang gliding, speedflying, accident, cause of death

Introduction

Foot-launched flying sports are popular activities in both the alpine and flat regions of Switzerland. The Swiss Hang Gliding Association (Schweizerischer Hängegleiter-Verband, SHV) was founded in 1974, soon after the first hang glider flight in Switzerland. The SHV is an association that coordinates flight training, conducts pilot examinations on behalf of the federal office of civil aviation, and promotes the interests of pilots. In 2018, the SHV had 16,798 members and included all foot-launched flying sports.[1] The SHV divides these sports into the following categories: paraglider, miniwing, hang glider, powered paraglider/hang glider, fixed-wing aircraft, and speedflyer. Each category requires a specific license. The paraglider license includes the miniwing and speedflyer, with the latter requiring an additional license. Miniwings (14–20 m^2) and speedflyers (<14 m^2) are smaller than paragliders (>20 m^2). Speedflyers reach velocities up to 120 $km \cdot h^{-1}$ (https://www.shv-fsvl.ch). The special geometry of speedflyers allows the pilot to perform new maneuvers and proximity flights by adapting the glide ratio. The powered versions are only allowed to be motorized with an electric motor. In Switzerland, powered foot-launched flying sports are legally equivalent to the flying of aircraft and therefore rarely practiced owing to bureaucratic obstacles.[2]

Based on a survey of 30,000 Swiss citizens, the Swiss Council for Accident Prevention (SCAP) estimated that 810 Swiss paragliders and 180 Swiss participants in other flying sports (hang gliding, powered paraglider/hang gliding, speedflying, flying a fixed wing aircraft, base-jumping, and skydiving) have an accident each year.[3] In

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the period of 2000 to 2017, 158 people died in Switzerland while flying a foot-launched glider (https://www.bfu.ch). Between 2009 and 2018, these sports averaged 8 fatalities per year in Switzerland. In comparison, mountaineering (n=24) and ski touring (n=19) accounted for more annual fatalities, whereas base jumping (n=7) and climbing (n=4) accounted for fewer.[3] Fatalities in mountaineering, skiing, skydiving, climbing, and gliding are mostly attributed to traumatic death, with natural deaths being less frequent.[4]

Because the literature is sparse and not up to date, it is important to report on the epidemiological data to enable further research and develop safety measures.[5] The previously cited 2017 article was the first and only one to separate speedflying from paragliding.[5] The distinction of the different categories is important because each sport requires unique flight techniques and specific equipment and involves category-associated risk factors. Our aim was to investigate the epidemiology, circumstances of death, and forensic examination results of foot-launched flying sport fatalities.

Methods

We performed a retrospective analysis of fatalities that occurred between 2000 and 2018 during foot-launched flying sports in the large mountainous canton of Berne, which is 1 of 26 cantons in Switzerland. Paragliding, hang gliding, speedflying, fixed-wing aircraft, and miniwing categories were investigated. Fixed-wing aircraft fatalities were added to hang gliding owing to the shared flight techniques and common risk factors of the 2 aircraft types. The Institute of Legal Medicine of the University of Bern registered the date and location of the fatalities successively after they occurred. The SHV and SCAP also registered and reported the fatalities independently. The cases from these 3 sources were matched and verified using the files of the responsible public prosecutors' offices (Jura-Lakeland, Emmental-Oberaargau, Midland, and Highland). The crucial source of data for our study was the files of the public prosecutors' offices, including forensic (external inspection, autopsy, and toxicology testing), police (testimonies, photographic and cartographic documentation with the exact coordinates of the incident sites, photographic documentation of the gliders, and avionic data), and medical reports.

The following parameters of interest were extracted from the files for analysis: general epidemiological data, cause of death, results of the toxicology testing, causes of the incident, equipment (including paraglider difficulty classes), the pilot's experience, and environmental factors. The paraglider difficulty classification ranged from the easy European Norm A to the difficult European Norm D classes.

We classified the weather conditions as follows: unproblematic (calm weather, normal or good conditions), problematic (gusty winds or strong winds [>25 $km \cdot h^{-1}$], increased turbulence, or fog in the flying area), and insufficient for classification (nonpilot witnesses or unknown). Nonpilot witnesses were not taken into account for evaluation of the weather conditions because they are inexperienced in assessing flight-specific weather.

Because of the importance of the files in our study, cases were excluded if their files were not found in the responsible public prosecutors' offices.

Data were entered into Microsoft Excel before analysis. They are presented as mean±SD for continuous variables and percentages and are rounded to the nearest whole number for the categorical variables, if the denominator was greater than 9.

We investigated reports that included the results of an official case investigation conducted by the responsible department of public prosecution that owns the data. The files are publicly available for research purposes if the use is authorized by the department of public prosecution. The written request to use the files for this study was approved by the responsible public prosecutor's office.

Results

The number of cases registered in our institute corresponded to the number of cases registered by the SCAP and SHV. Forty-two fatalities occurred in all foot-launched flying sporting activities during the study period. Three files for these confirmed cases were not found in the public prosecutors' offices and were excluded from further analysis. Consequently, the given results include 39 fatalities in 37 cases (4 people died in 2 tandem paragliding accidents). Paragliding accounted for 27 (69%), speedflying for 7 (18%), and hang gliding for 5 (13%) fatalities. No cases involving miniwings or powered paragliders/hang gliders were recorded. Therefore, 3 categories are shown: paragliding (P), hang gliding (H), and speedflying (S). The annual number of fatalities in the canton of Berne, as well as nationwide data, from 2000 through 2018 are shown in Figure 1. Speedflying information was included in the database from the beginning of 2008.

The epidemiological results show that only 1 fatality with a female pilot (3%) was reported (hang gliding). The 2 other females were passengers in tandem paragliders. Table 1 and Figure 2 show the epidemiological results. The glider-specific age distribution shows the predominantly young age of the speedflyers.

Table 2 shows the accident circumstances. Most of the 37 accidents occurred midflight, followed by take-off and landing. After successful take-off, 17 accidents (65%) occurred in the first 10 min of the flight. The accident happened after more than 1 h in only 1 case. The 2 incomplete preflight checks indicated in Table 2 were due to insufficiently closed harnesses with open leg belts, causing a fall out of the harness. Equipment failure was not blamed for any fatal accidents; however, in 1 case improper maintenance of the equipment resulted in death. In this case, a test pilot shortened the paraglider's lines using knots and placed them incorrectly. As a consequence, the lines were destabilized, and they all ripped during a spiral dive. For 28 victims (72%; 17P, 4H, 7S), the use of a helmet was verified in the documents. For the other cases, no information on helmet-wearing was available. As shown in Table 3, the rescue parachute was deployed in only 5 of 23 possible situations (22%; 5P), and in only 1 case (4%; 1P) was the parachute able to fully deploy. This pilot had a nonsteerable rescue parachute. He was unable to avoid collision with a rock face and fell to his death. In 12 cases, the height above the ground was ≤100 m for the event causing the accident.

Figure 3 shows the distribution of fatalities for different glider difficulties. The proportion of glider-associated fatalities was higher among more demanding paragliders. Additionally, 2 aerobatic (5%), 2 tandem (5%), and 1 unknown paraglider (3%) were reported.

Data regarding the years of experience and number of flights were rare and mostly incomplete. Table 3 shows the available data on the pilots' experience. Five of 7 speedflyer pilots had held their license for ≤2 y. Because the literature on speedflying is scant, a short description of all speedflying fatalities is listed in Table 4.

Data on the environment are presented in Table 3. In 2 cases, the altitude of the accidents was >4000 m above mean sea level (4100 m and 4080 m, 5%; 1P, 1S, respectively). In both cases, take-off errors occurred after climbing the summit of a mountain. Three of the 6 cases from 2500 m to 4000 m (16%; 4P, 2S) were also caused by take-off errors. These alpine take-offs were characterized by ice, firn, and rocks. All of the speedflying fatalities occurred in the 15-km-long Lauterbrunnen valley.

When an event occurred during the flight (n=22), the altitude above ground was 0 to 50 m in 4 cases (18%; 2P, 1H, 1S), 50 to 100 m in 8 cases (36%; 6P, 1H, 1S);=, and over 150 m in 7 cases (32%; 7P). In 3 of 22 cases, this information was not provided. Take-off and landing errors, direct collisions with the ground, and unclosed harnesses were excluded, resulting in 22 cases.

The medical data and forensic examinations revealed that autopsies were performed for only 10 victims (26%) and revealed pre-existing medical conditions in 3 cases (30%): 1) atheromatosis, 2) coronary heart disease and pulmonary emphysema, and 3) atheromatosis and atherosclerosis. For the individual with the coronary heart disease, the report discussed whether this finding could have been relevant as the cause of the incident. The most likely cause of death for each victim, obtained from an external inspection and rarely from autopsy, is listed in Table 5. In 1 case, the manner of death was initially considered suicide because the pilot did not close the harness correctly. However, witnesses reported later that the pilot fought for his life, holding onto the harness. Therefore, the case was finally assessed as an accident. Consequently, all incidents were thus assessed as accidents. One person (3%; 1P) was conscious, and resuscitation was not necessary when found. The death, due to cerebrocranial trauma, occurred the same day after admission to the hospital.

Discussion

The number of foot-launched flying sport fatalities registered at our institute, SCAP, and with SHV were congruent. Therefore, it can be assumed that most cases in the area being studied were known. Three cases were excluded because the corresponding files were not available. The SCAP had 16,798 members nationwide at the end of 2018; however, once a license has been obtained, it is not mandatory to be an SHV member.[1] Because the exact number of active pilots and the number of accidents in the canton of Berne itself are unknown, it was not possible to calculate mortality rates. Among the British Hang Gliding and Paragliding Association, member fatality rates (fatalities per 100,000 participants per year) of 40 from hang gliding and 47 from paragliding were reported; the rates for speedflying were not available.[5]

The distribution of fatal accidents over the years in the canton of Berne and throughout Switzerland showed a similar dynamic (Figure 1). Although the absolute numbers did not increase, the number of active pilots and nonfatal accidents increased[1,3] (https://www.bfu.ch). The new sport of speedflying, which emerged in 2008, may have maintained the level of the absolute number of foot-launched sport fatalities. The high rate of fatalities among pilots between 20 and 30 y of age in Berne compared with the rates in Switzerland in general (https://www.bfu.ch) can be explained by the fact that the data from this decade include 6 of 7 speedflying fatalities. The speedflying danger zone, Lauterbrunnen Valley (canton of Berne), contributed to this mismatch in distribution by attracting mainly these pilots. Speedflyer pilots were the youngest pilots and the youngest British Hang Gliding

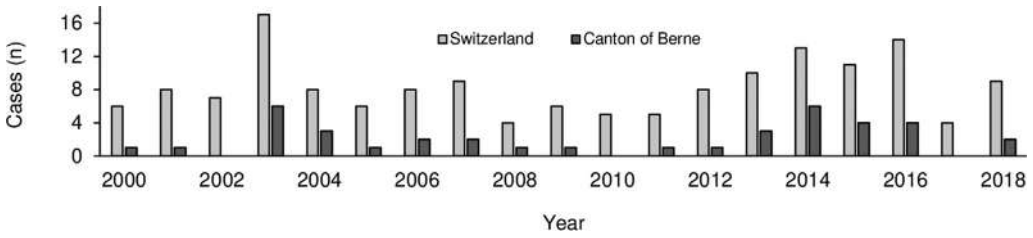

Figure 1. Annual number of fatalities in Switzerland (https://www.bfu.ch) and in the canton of Berne from 2000 to 2018.

and Paragliding Association members among the foot-launched flying sports' accidents, excluding parasailing. This sport is not performed in Switzerland and is not part of the SHV. The miniwing is more of a niche product and offers few advantages over a paraglider or a speedflyer and is therefore rarely flown.

The sex distribution in this study (92% male, 8% female) showed a slightly higher proportion of males than in other studies. In Austria, 86% were male and 14% female, and in Great Britain, 88% were male, 9% female, and 3% unknown.[5,6] Both studies included fatal and nonfatal accidents. The relative risk of crashing was found to be higher in females.[6] The rates for deadly accidents for each sex could not be calculated because the SHV does not record members' sex.

Detailed analyses of nonfatal accidents have shown that the midflight[6] or landing[7,8] phases are the 2 most dangerous phases of the flight in terms of the absolute numbers of accidents. In Switzerland, between 2011 and 2017, the landing phase resulted in most of the nonfatal accidents, mostly followed by the take-off phase.[9-15] Our study found that most fatal accidents in the canton of Berne occurred midflight, followed by the take-off phase. The transformation of the higher potential energy during the take-off and mid-flight phases to kinetic energy during a fall could be the reason for the more severe impact. In contrast, rough landings, especially in difficult terrain,[7] tend to result in lower extremity and spinal injuries.[8]

Table 2 shows certain category-specific differences for the causes of the accidents. Glider collapse was the most frequent cause of fatal accidents in this study (24% of all cases, 36% of all paragliders). In 409 consecutive paragliding accidents in Germany, the collapse of the canopy was the most reported cause of accidents (33%). Although the rigid wing of hang gliders protects them from collapse, they are more at risk during the take-off phase. Owing to their size and weight, or rather the principle of inertia, a take-off abort is usually not possible, with take-off failure being potentially fatal.

It has been said that a long flight can be compared with a long car drive[16] and can cause mental exhaustion.[17] The duration of the flights (only 1 case >1 h) did not suggest that long flights led to fatalities. Thus, mental exhaustion is unlikely to have played a role.

Equipment failure was not blamed for any fatalities in our study. Other studies also reported that equipment failure was a rare cause of accidents (1%, n=405; 3%, n=2037).[6,18] However, quality, approved, and presumably safe equipment was reported to be important for paragliding pilots.[19] The rates of helmet use and the installation of rescue parachutes (both 100% considering only the cases with known status) among the fatalities in this study show their importance for pilots in Switzerland. In comparison, the SCAP reported that only 16% of skiers and snowboarders wore helmets in the 2002 to 2003 winter season in Switzerland. This, however, increased consistently to 92% in the winter season

Table 1. Epidemiological data, absolute and relative numbers

	All cases n=39 (%)	*Paragliding n=27 (%)*	*Hang gliding n=5*	*Speedflying n=7*
Age (y), mean±SD	42±14	44±13	50±8	27±4
Sex, n (%)				
Male	36 (92)	25 (93)	4	7
Female	3 (8)	2 (7)	1	0
Nationality, n (%)				
Switzerland	31 (79)	21 (78)	5	5
Foreign country	8 (21)	6 (22)	0	2

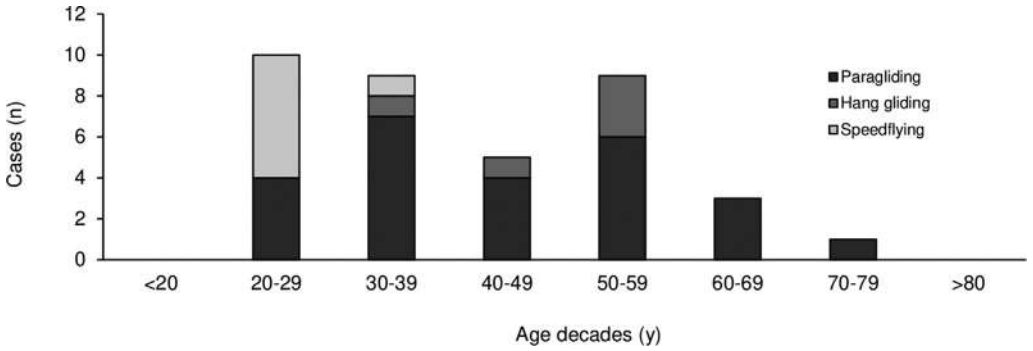

Figure 2. Number of fatalities for the different age decades.

of 2015 to 2016 (https://www.bfu.ch). This indicates that participants may become more safety conscious over time.

Although the rescue parachute installation rate was 100%, the pilots rarely used the parachute. Of 24 cases, only 5 pilots pulled their parachutes, and only 1 succeeded in fully deploying the parachute. The low percentage of use could be due to a lack of emergency training, in which the practice of pulling the rescue parachute becomes a reflex action. The low altitude aggravated the situation. In 12 cases, the event that led to the accident happened at <100 m above the ground, which is usually not sufficient to use a rescue parachute. A free fall of 100 m with g=9.81 $m \cdot s^{-1}$ takes 4.5 s. After deciding to use the rescue parachute, it takes 4 s to find the handle, grab it, pull it, throw it, and let the parachute fully deploy.[20] The isolated opening time of a new rescue parachute is approximately 1 s (from throw to full deployment).[20] The low altitude and the rare use of the rescue parachute led us to make 2 recommendations: repeating safety courses and ensuring a sufficient distance from the ground and the slope.

The increasing proportion of glider-associated accidents, with an increase in glider difficulty (Figure 3), supports the warnings of the SHV, flight schools, and manufacturers that high-end gliders can become out of control more easily.[9-15] Experienced pilots have fewer accidents during the take-off phase compared with beginners; however, a large number of accidents resulting from collapse occurred among experienced pilots.[18] A potential explanation for the discrepancy between the take-off and the mid-flight phase events might be equipment choice, if experienced pilots tend to choose more demanding gliders. It has also been suggested that experienced pilots take more risks.[21]

Data regarding pilot experience were rarely reported. Focusing on experience among speedflyer pilots, 5 of 7

Table 2. Accident circumstances; collision with objects includes objects on the ground and in the air

	All cases n=37; n (%)	*Paragliding n=25; n (%)*	*Hang gliding n=5*	*Speedflying n=7*
Flight phase				
Take-off	12 (27)	3 (12)	3	4
Mid-flight	24 (70)	21 (84)	2	3
Landing	1 (3)	1 (4)	0	0
Cause of accident				
Collapse of the glider	9 (24)	9 (36)	0	0
Take-off error	8 (22)	2 (8)	3	3
Collision with object	6 (16)	4 (16)	0	2
Oversteering	4 (11)	2 (8)	1	1
Incomplete preflight check	2 (5)	2 (8)	0	0
Failed maneuver	2 (5)	2 (8)	0	0
Collision with flight object	1 (3)	1 (4)	0	0
Landing error	1 (3)	1 (4)	0	0
Improper maintenance	1 (3)	1 (4)	0	0
Unknown	3 (8)	1 (4)	1	1

Table 3. Rescue parachute use, pilot experience, and environmental parameters

	All cases n=37; n (%)
Rescue parachute	
On board	24 (65)
Used	5 (22)
Not used	19 (78)
Not on board	0 (0)
Unknown[a]	13 (35)
Experience	
Student pilot	1 (3)
Pilot license	20 (54)
Multiple pilot licenses	9 (24)
Professional	3 (8)
No license	2 (5)
Unknown	2(5)
Height of the event (m)[b]	
0–1000	4 (11)
>1000–2000	15 (41)
>2000–3000	12 (32)
>3000–4000	2 (5)
>4000	2 (5)
Unknown	2 (5)
Weather conditions	
Problematic	6 (16)
Unproblematic	22 (59)
Insufficient for classification	8 (22)
Unknown	1 (3)

[a]Note that in the cases in which the rescue parachute status was unknown, use of it was impossible.

[b]"Event" refers to the altitude where the problem started to develop. In the cases of take-off errors, the altitude of the launch site is indicated.

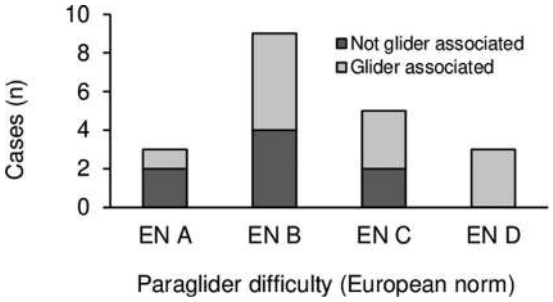

Figure 3. Number of fatalities and association with different glider difficulties in paragliding. Not glider-associated: incomplete preflight check, collisions; glider-associated: take-off errors, collapses, oversteering, spiral dive.

died within the first 2 y after receiving their license. The first 2 y after gaining a license are the most accident prone, irrespective of the number of completed flights.[18] This is despite speedflyers already requiring a paragliding license before speedflying training. Experience appeared to decrease the risk of some types of mishaps[5,7] while increasing the risks of others.[7,21] To reduce accidents, all pilots should strictly maintain use of the recommended checklists to avoid preventable mistakes and should take care in glider selection.[20]

Bad weather conditions or weather changes are often described as risk factors[6,7,18,22] and play a contributing role in up to 33% of all accidents,[23] becoming risk factors for deadly accidents. For base jumping, the environmental factors linked to fatal accidents were mostly a result of poor decision making.[24] Although 5 of 6 fatalities in bad weather conditions were thought to have been provoked by these conditions, most of the fatalities (59%) occurred in unproblematic weather conditions.

The famous Lauterbrunnen Valley attracts speedflyer pilots, and a similar aggregation is seen in base jumping. The base jumping fatality list compiled by BLiNC magazine, a community base jumping website, reported that 51 (56%) of 91 fatalities in Switzerland between 1994 and 2019 occurred in the Lauterbrunnen Valley. The popular region allows up to 10 base jumps per day, leading to a multiplication of the absolute risk.[24] The circumstances of speedflying fatalities in this challenging region show that these pilots tend to expose themselves to high-risk situations, either through the use of demanding and dangerous launch sites or through proximity flying.

The elevation exposure during the accident in 8 cases was between 2500 and 4100 m above mean sea level, referred to as moderate altitude.[25] These altitudes are potentially sufficient for some degree of hypoxia or even high altitude illness.[25-27] A systematic review suggested that hypobaric hypoxia at moderate altitudes may lead to cognitive and psychomotor deficits in learning, reaction times, decision-making, and certain types of memory.[25] However, the results at moderate altitude were inconsistent,[25] with reported thresholds for cognitive deficits ranging from 1525 m when exercising to 4572 m at rest.[28,29] There has been shown to be an increase in reaction time during exercise (80% peak oxygen consumption, $\dot{V}O_{2\ peak}$) in individuals at a normobaric hypoxia equivalent to 2200 m.[30] At moderate altitudes, $\dot{V}O_{2\ peak}$, respiratory rate, and heart rate are increased only during the take-off phase.[16] Combined with the physical exertion required to reach take-off sites by hiking or mountaineering, hypobaric hypoxia may have played a role in these take-off fatalities.

The medical and forensic data showed a high proportion of cases (54%) without any medical interventions. This suggests that the victims sustained from severe injuries from the accident. The causes of death

Table 4. Event description and assumed causes of fatal speedflying accidents

Case	*Event description*	*Assumed cause*
1	Take-off failure with crampons on Jungfrau (4100 m), slipped, glided over glacier, fall of over 50 m	Crosswinds disarranged the chute immediately before take-off
2	Take-off over cliff, stall, collapse of the wing, fall of over 80 m	Oversteering of the speedflyer
3	Cliff take-off failure, fall of over 120 m	Slipped with sneakers on wet grass
4	During take-off, fall in a crevasse	Choice of wrong take-off slope
5	Low flight, collision with a tree	Flying too low
6	Spiral dive not terminated until collision with the ground	Loss of consciousness or inability to terminate the spiral dive
7	Crash without injuries in hostile terrain, refused rescue by a helicopter, slipped, fall of off a cliff	Slipping in steep terrain

listed in Table 5 underline this suggestion on the basis of a high proportion of multiple trauma (54%). The literature is consistent about the fact that foot-launched flying sports accidents are often severe.[7,22] Hang gliding pilots seem to be more prone to head and neck injuries and tend to die from cerebrocranial trauma. Other studies have shown that hang gliding pilots are more likely to experience head and neck injuries than pilots in the other categories.[5,6] The high proportion of external-only inspections (74%) without autopsy did not reveal the exact cause of death, as would be expected from autopsies. Therefore, we are not able to discuss the importance of traumatic aortic rupture as a hidden and missed injury, as described in the literature.[13,31,32]

In comparison with a case of suicide, in which a skydiver manipulated his rescue system,[33] suicidal intentions were suggested in only 1 case with an unclosed harness. The suicide suggestion was ruled out later by witness testimony. In 1 case in which an autopsy was done, the accident mechanism showed the involvement of pre-existing medical conditions (coronary heart disease and pulmonary emphysema). In this case, the 64-y-old pilot flew without any steering impulses into a cliff. A case series also suggested that 1 of 6 skydivers who were

Table 5. Forensic examination and toxicological analysis, absolute and relative numbers

	All cases n=39; n (%)	*Paragliding n=27; n (%)*	*Hang gliding n=5; n*	*Speedflying n=7; n*
Body examination				
External inspection	29 (74)	19 (70)	3	7
Autopsy (with/without external inspection)	10 (26)	8 (30)	2	0
Toxicological analysis				
Tested	10 (26)	8 (30)	2	0
Positive	0 (0)	0 (0)	0	0
Negative	10 (100)	8 (100)	2	0
Not tested	29 (74)	19 (70)	3	7
Cause of death				
CCT	7 (18)	4 (15)	2	1
Multiple trauma with CCT	18 (46)	14 (52)	2	2
Multiple trauma	3 (8)	2 (7)	0	1
Fat embolism	3 (8)	3 (11)	0	0
Internal/External bleeding	3 (8)	1 (4)	1	1
Unclear	5 (13)	3 (11)	0	2
Resuscitation at accident site				
Yes	17 (44)	15 (56)	1	1
No	22 (56)	12 (44)	4	6
Absolute signs of death	21 (95)	12 (44)	4	6
Patient responsive	1 (5)	0 (0)	0	0

CCT, craniocerebral trauma.

autopsied could have lost consciousness owing the presence of coronary heart disease, outflow obstruction, and myocardial hypertrophy and may therefore have not opened the parachute.[34] However, there was no evidence of the influence of pre-existing medical conditions on the accidents. Therefore, because there is seldom danger to others, we do not recommend compulsory medical certificates, as is done in general aviation. An increased number of autopsies could help to give better-founded recommendations, in this regard.

To fully understand the causes and dynamics of foot-launched flying fatalities, a detailed examination of these cases is required.

LIMITATIONS

Data were gathered for nonresearch purposes by rescue teams, the police, and forensic doctors, and mostly without specific knowledge of foot-launched flying sports. Most of the causes of death were determined only by external inspection. Therefore, the medical results must be evaluated with caution and are only listed as the most probable cause of death. Records of pre-existing medical conditions and prescribed drugs were rare. Consequently, the data were incomplete, contained obvious assumptions, and included witness testimony. Nonpilot observations need to be assessed critically owing to possible misinterpretations.

The low number of accidents involving fatalities between 2000 and 2018, missing files, and the retrospective method used limited the value of the general statements about foot-launched flying sport fatalities.

The accidents occurred within a period of 20 y. The temporal dynamics, technical developments, adaptations of the education system, and other new security measures were not taken into consideration and may therefore have created a bias. Because the literature was sparse and not up to date, it was difficult to make comparisons with the results of this study.

Conclusions

The categories of foot-launched flying sports differed in the causes of accidents and causes of death. The causes of death appeared to be traumatic in nature, and all incidents were categorized as accidents. The deceased speedflying pilots were on average 18 to 23 y younger than the paraglider/hang glider pilots and often died in high-risk situations. High altitude take-off sites, incomplete or missing preflight checks, flying close to the slope, and use of demanding gliders were identified as potential risk factors. We see prevention possibilities in education and training, the implementation of preflight checklists, and familiarizing pilots with the technique of throwing the rescue parachute. We recommend that all pilots fly and choose their glider cautiously, considering their experience and their flying skills.

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Author Contributions: Design of the study (FS, CS); acquisition of the data (FS, CS); analysis of the data (FS); interpretation of the data (FS, CS); drafting of the manuscript (FS); critical revision of the article; approval of the final manuscript (FS, CS, CJ).

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ORIGINAL RESEARCH

Foot-Launched Flying Sport Fatalities in the Canton of Berne, Switzerland

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Introduction—Foot-launched flying sports such as paragliding, hang gliding, and speedflying are popular recreational activities that all pose a significant risk of accidents resulting in injuries or fatalities. We describe the epidemiology, incident circumstances, and findings of forensic examinations of fatalities in these sports.

Methods—In this retrospective analysis, we analyzed different parameters of paragliding, hang gliding, and speedflying fatalities in the canton of Berne, Switzerland, between 2000 and 2018. The data sources were police reports, forensic medical reports, the Swiss Hang Gliding Association, and the Swiss Council for Accident Prevention.

Results—In the given period, 40 incidents resulting in 42 fatalities were recorded (2 incidents involved 2-seaters). Three of the 40 incidents did not fulfill the inclusion criteria and were excluded. The deadliest phase was midflight. Collapse of the glider (n=9; 36%) was the leading cause of accidents among paraglider pilots. Multiple trauma (n=21; 54%) was the most frequent cause of death. In the forensic examination, all deaths (n=39) were assessed as accidents.

Conclusions—The different categories of foot-launched flying sports varied in the causes of accidents and deaths. Fatalities in speedflying involved young pilots exposing themselves to high-risk situations. Fatalities may be prevented by enhancing education and training and promoting a cautious attitude among pilots.

Keywords: adventure sports, paragliding, hang gliding, speedflying, accident, cause of death

Introduction

Foot-launched flying sports are popular activities in both the alpine and flat regions of Switzerland. The Swiss Hang Gliding Association (Schweizerischer Hängegleiter-Verband, SHV) was founded in 1974, soon after the first hang glider flight in Switzerland. The SHV is an association that coordinates flight training, conducts pilot examinations on behalf of the federal office of civil aviation, and promotes the interests of pilots. In 2018, the SHV had 16,798 members and included all foot-launched flying sports.[1] The SHV divides these sports into the following categories: paraglider, miniwing, hang glider, powered paraglider/hang glider, fixed-wing aircraft, and speedflyer. Each category requires a specific license. The paraglider license includes the miniwing and speedflyer, with the latter requiring an additional license. Miniwings (14–20 m^2) and speedflyers (<14 m^2) are smaller than paragliders (>20 m^2). Speedflyers reach velocities up to 120 $km \cdot h^{-1}$ (https://www.shv-fsvl.ch). The special geometry of speedflyers allows the pilot to perform new maneuvers and proximity flights by adapting the glide ratio. The powered versions are only allowed to be motorized with an electric motor. In Switzerland, powered foot-launched flying sports are legally equivalent to the flying of aircraft and therefore rarely practiced owing to bureaucratic obstacles.[2]

Based on a survey of 30,000 Swiss citizens, the Swiss Council for Accident Prevention (SCAP) estimated that 810 Swiss paragliders and 180 Swiss participants in other flying sports (hang gliding, powered paraglider/hang gliding, speedflying, flying a fixed wing aircraft, base-jumping, and skydiving) have an accident each year.[3] In

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the period of 2000 to 2017, 158 people died in Switzerland while flying a foot-launched glider (https://www.bfu.ch). Between 2009 and 2018, these sports averaged 8 fatalities per year in Switzerland. In comparison, mountaineering (n=24) and ski touring (n=19) accounted for more annual fatalities, whereas base jumping (n=7) and climbing (n=4) accounted for fewer.[3] Fatalities in mountaineering, skiing, skydiving, climbing, and gliding are mostly attributed to traumatic death, with natural deaths being less frequent.[4]

Because the literature is sparse and not up to date, it is important to report on the epidemiological data to enable further research and develop safety measures.[5] The previously cited 2017 article was the first and only one to separate speedflying from paragliding.[5] The distinction of the different categories is important because each sport requires unique flight techniques and specific equipment and involves category-associated risk factors. Our aim was to investigate the epidemiology, circumstances of death, and forensic examination results of foot-launched flying sport fatalities.

Methods

We performed a retrospective analysis of fatalities that occurred between 2000 and 2018 during foot-launched flying sports in the large mountainous canton of Berne, which is 1 of 26 cantons in Switzerland. Paragliding, hang gliding, speedflying, fixed-wing aircraft, and miniwing categories were investigated. Fixed-wing aircraft fatalities were added to hang gliding owing to the shared flight techniques and common risk factors of the 2 aircraft types. The Institute of Legal Medicine of the University of Bern registered the date and location of the fatalities successively after they occurred. The SHV and SCAP also registered and reported the fatalities independently. The cases from these 3 sources were matched and verified using the files of the responsible public prosecutors' offices (Jura-Lakeland, Emmental-Oberaargau, Midland, and Highland). The crucial source of data for our study was the files of the public prosecutors' offices, including forensic (external inspection, autopsy, and toxicology testing), police (testimonies, photographic and cartographic documentation with the exact coordinates of the incident sites, photographic documentation of the gliders, and avionic data), and medical reports.

The following parameters of interest were extracted from the files for analysis: general epidemiological data, cause of death, results of the toxicology testing, causes of the incident, equipment (including paraglider difficulty classes), the pilot's experience, and environmental factors. The paraglider difficulty classification ranged from the easy European Norm A to the difficult European Norm D classes.

We classified the weather conditions as follows: unproblematic (calm weather, normal or good conditions), problematic (gusty winds or strong winds [>25 $km \cdot h^{-1}$], increased turbulence, or fog in the flying area), and insufficient for classification (nonpilot witnesses or unknown). Nonpilot witnesses were not taken into account for evaluation of the weather conditions because they are inexperienced in assessing flight-specific weather.

Because of the importance of the files in our study, cases were excluded if their files were not found in the responsible public prosecutors' offices.

Data were entered into Microsoft Excel before analysis. They are presented as mean±SD for continuous variables and percentages and are rounded to the nearest whole number for the categorical variables, if the denominator was greater than 9.

We investigated reports that included the results of an official case investigation conducted by the responsible department of public prosecution that owns the data. The files are publicly available for research purposes if the use is authorized by the department of public prosecution. The written request to use the files for this study was approved by the responsible public prosecutor's office.

Results

The number of cases registered in our institute corresponded to the number of cases registered by the SCAP and SHV. Forty-two fatalities occurred in all foot-launched flying sporting activities during the study period. Three files for these confirmed cases were not found in the public prosecutors' offices and were excluded from further analysis. Consequently, the given results include 39 fatalities in 37 cases (4 people died in 2 tandem paragliding accidents). Paragliding accounted for 27 (69%), speedflying for 7 (18%), and hang gliding for 5 (13%) fatalities. No cases involving miniwings or powered paragliders/hang gliders were recorded. Therefore, 3 categories are shown: paragliding (P), hang gliding (H), and speedflying (S). The annual number of fatalities in the canton of Berne, as well as nationwide data, from 2000 through 2018 are shown in Figure 1. Speedflying information was included in the database from the beginning of 2008.

The epidemiological results show that only 1 fatality with a female pilot (3%) was reported (hang gliding). The 2 other females were passengers in tandem paragliders. Table 1 and Figure 2 show the epidemiological results. The glider-specific age distribution shows the predominantly young age of the speedflyers.

Table 2 shows the accident circumstances. Most of the 37 accidents occurred midflight, followed by take-off and landing. After successful take-off, 17 accidents (65%) occurred in the first 10 min of the flight. The accident happened after more than 1 h in only 1 case. The 2 incomplete preflight checks indicated in Table 2 were due to insufficiently closed harnesses with open leg belts, causing a fall out of the harness. Equipment failure was not blamed for any fatal accidents; however, in 1 case improper maintenance of the equipment resulted in death. In this case, a test pilot shortened the paraglider's lines using knots and placed them incorrectly. As a consequence, the lines were destabilized, and they all ripped during a spiral dive. For 28 victims (72%; 17P, 4H, 7S), the use of a helmet was verified in the documents. For the other cases, no information on helmet-wearing was available. As shown in Table 3, the rescue parachute was deployed in only 5 of 23 possible situations (22%; 5P), and in only 1 case (4%; 1P) was the parachute able to fully deploy. This pilot had a nonsteerable rescue parachute. He was unable to avoid collision with a rock face and fell to his death. In 12 cases, the height above the ground was ≤100 m for the event causing the accident.

Figure 3 shows the distribution of fatalities for different glider difficulties. The proportion of glider-associated fatalities was higher among more demanding paragliders. Additionally, 2 aerobatic (5%), 2 tandem (5%), and 1 unknown paraglider (3%) were reported.

Data regarding the years of experience and number of flights were rare and mostly incomplete. Table 3 shows the available data on the pilots' experience. Five of 7 speedflyer pilots had held their license for ≤2 y. Because the literature on speedflying is scant, a short description of all speedflying fatalities is listed in Table 4.

Data on the environment are presented in Table 3. In 2 cases, the altitude of the accidents was >4000 m above mean sea level (4100 m and 4080 m, 5%; 1P, 1S, respectively). In both cases, take-off errors occurred after climbing the summit of a mountain. Three of the 6 cases from 2500 m to 4000 m (16%; 4P, 2S) were also caused by take-off errors. These alpine take-offs were characterized by ice, firn, and rocks. All of the speedflying fatalities occurred in the 15-km-long Lauterbrunnen valley.

When an event occurred during the flight (n=22), the altitude above ground was 0 to 50 m in 4 cases (18%; 2P, 1H, 1S), 50 to 100 m in 8 cases (36%; 6P, 1H, 1S);=, and over 150 m in 7 cases (32%; 7P). In 3 of 22 cases, this information was not provided. Take-off and landing errors, direct collisions with the ground, and unclosed harnesses were excluded, resulting in 22 cases.

The medical data and forensic examinations revealed that autopsies were performed for only 10 victims (26%) and revealed pre-existing medical conditions in 3 cases (30%): 1) atheromatosis, 2) coronary heart disease and pulmonary emphysema, and 3) atheromatosis and atherosclerosis. For the individual with the coronary heart disease, the report discussed whether this finding could have been relevant as the cause of the incident. The most likely cause of death for each victim, obtained from an external inspection and rarely from autopsy, is listed in Table 5. In 1 case, the manner of death was initially considered suicide because the pilot did not close the harness correctly. However, witnesses reported later that the pilot fought for his life, holding onto the harness. Therefore, the case was finally assessed as an accident. Consequently, all incidents were thus assessed as accidents. One person (3%; 1P) was conscious, and resuscitation was not necessary when found. The death, due to cerebrocranial trauma, occurred the same day after admission to the hospital.

Discussion

The number of foot-launched flying sport fatalities registered at our institute, SCAP, and with SHV were congruent. Therefore, it can be assumed that most cases in the area being studied were known. Three cases were excluded because the corresponding files were not available. The SCAP had 16,798 members nationwide at the end of 2018; however, once a license has been obtained, it is not mandatory to be an SHV member.[1] Because the exact number of active pilots and the number of accidents in the canton of Berne itself are unknown, it was not possible to calculate mortality rates. Among the British Hang Gliding and Paragliding Association, member fatality rates (fatalities per 100,000 participants per year) of 40 from hang gliding and 47 from paragliding were reported; the rates for speedflying were not available.[5]

The distribution of fatal accidents over the years in the canton of Berne and throughout Switzerland showed a similar dynamic (Figure 1). Although the absolute numbers did not increase, the number of active pilots and nonfatal accidents increased[1,3] (https://www.bfu.ch). The new sport of speedflying, which emerged in 2008, may have maintained the level of the absolute number of foot-launched sport fatalities. The high rate of fatalities among pilots between 20 and 30 y of age in Berne compared with the rates in Switzerland in general (https://www.bfu.ch) can be explained by the fact that the data from this decade include 6 of 7 speedflying fatalities. The speedflying danger zone, Lauterbrunnen Valley (canton of Berne), contributed to this mismatch in distribution by attracting mainly these pilots. Speedflyer pilots were the youngest pilots and the youngest British Hang Gliding

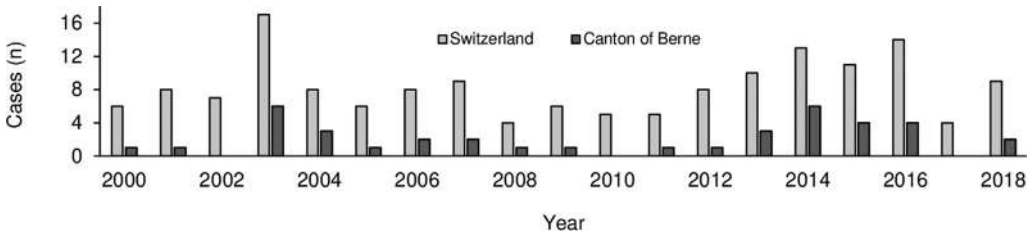

Figure 1. Annual number of fatalities in Switzerland (https://www.bfu.ch) and in the canton of Berne from 2000 to 2018.

and Paragliding Association members among the foot-launched flying sports' accidents, excluding parasailing. This sport is not performed in Switzerland and is not part of the SHV. The miniwing is more of a niche product and offers few advantages over a paraglider or a speedflyer and is therefore rarely flown.

The sex distribution in this study (92% male, 8% female) showed a slightly higher proportion of males than in other studies. In Austria, 86% were male and 14% female, and in Great Britain, 88% were male, 9% female, and 3% unknown.[5,6] Both studies included fatal and nonfatal accidents. The relative risk of crashing was found to be higher in females.[6] The rates for deadly accidents for each sex could not be calculated because the SHV does not record members' sex.

Detailed analyses of nonfatal accidents have shown that the midflight[6] or landing[7,8] phases are the 2 most dangerous phases of the flight in terms of the absolute numbers of accidents. In Switzerland, between 2011 and 2017, the landing phase resulted in most of the nonfatal accidents, mostly followed by the take-off phase.[9-15] Our study found that most fatal accidents in the canton of Berne occurred midflight, followed by the take-off phase. The transformation of the higher potential energy during the take-off and mid-flight phases to kinetic energy during a fall could be the reason for the more severe impact. In contrast, rough landings, especially in difficult terrain,[7] tend to result in lower extremity and spinal injuries.[8]

Table 2 shows certain category-specific differences for the causes of the accidents. Glider collapse was the most frequent cause of fatal accidents in this study (24% of all cases, 36% of all paragliders). In 409 consecutive paragliding accidents in Germany, the collapse of the canopy was the most reported cause of accidents (33%). Although the rigid wing of hang gliders protects them from collapse, they are more at risk during the take-off phase. Owing to their size and weight, or rather the principle of inertia, a take-off abort is usually not possible, with take-off failure being potentially fatal.

It has been said that a long flight can be compared with a long car drive[16] and can cause mental exhaustion.[17] The duration of the flights (only 1 case >1 h) did not suggest that long flights led to fatalities. Thus, mental exhaustion is unlikely to have played a role.

Equipment failure was not blamed for any fatalities in our study. Other studies also reported that equipment failure was a rare cause of accidents (1%, n=405; 3%, n=2037).[6,18] However, quality, approved, and presumably safe equipment was reported to be important for paragliding pilots.[19] The rates of helmet use and the installation of rescue parachutes (both 100% considering only the cases with known status) among the fatalities in this study show their importance for pilots in Switzerland. In comparison, the SCAP reported that only 16% of skiers and snowboarders wore helmets in the 2002 to 2003 winter season in Switzerland. This, however, increased consistently to 92% in the winter season

Table 1. Epidemiological data, absolute and relative numbers

	All cases n=39 (%)	*Paragliding n=27 (%)*	*Hang gliding n=5*	*Speedflying n=7*
Age (y), mean±SD	42±14	44±13	50±8	27±4
Sex, n (%)				
Male	36 (92)	25 (93)	4	7
Female	3 (8)	2 (7)	1	0
Nationality, n (%)				
Switzerland	31 (79)	21 (78)	5	5
Foreign country	8 (21)	6 (22)	0	2

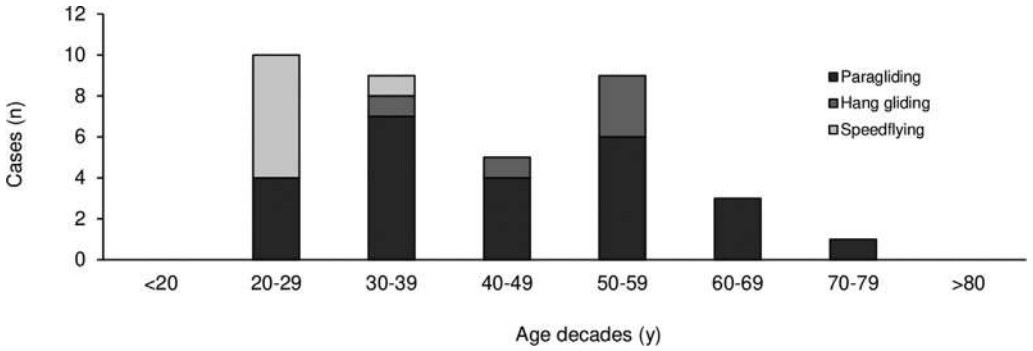

Figure 2. Number of fatalities for the different age decades.

of 2015 to 2016 (https://www.bfu.ch). This indicates that participants may become more safety conscious over time.

Although the rescue parachute installation rate was 100%, the pilots rarely used the parachute. Of 24 cases, only 5 pilots pulled their parachutes, and only 1 succeeded in fully deploying the parachute. The low percentage of use could be due to a lack of emergency training, in which the practice of pulling the rescue parachute becomes a reflex action. The low altitude aggravated the situation. In 12 cases, the event that led to the accident happened at <100 m above the ground, which is usually not sufficient to use a rescue parachute. A free fall of 100 m with g=9.81 $m \cdot s^{-1}$ takes 4.5 s. After deciding to use the rescue parachute, it takes 4 s to find the handle, grab it, pull it, throw it, and let the parachute fully deploy.[20] The isolated opening time of a new rescue parachute is approximately 1 s (from throw to full deployment).[20] The low altitude and the rare use of the rescue parachute led us to make 2 recommendations: repeating safety courses and ensuring a sufficient distance from the ground and the slope.

The increasing proportion of glider-associated accidents, with an increase in glider difficulty (Figure 3), supports the warnings of the SHV, flight schools, and manufacturers that high-end gliders can become out of control more easily.[9-15] Experienced pilots have fewer accidents during the take-off phase compared with beginners; however, a large number of accidents resulting from collapse occurred among experienced pilots.[18] A potential explanation for the discrepancy between the take-off and the mid-flight phase events might be equipment choice, if experienced pilots tend to choose more demanding gliders. It has also been suggested that experienced pilots take more risks.[21]

Data regarding pilot experience were rarely reported. Focusing on experience among speedflyer pilots, 5 of 7

Table 2. Accident circumstances; collision with objects includes objects on the ground and in the air

	All cases n=37; n (%)	*Paragliding n=25; n (%)*	*Hang gliding n=5*	*Speedflying n=7*
Flight phase				
Take-off	12 (27)	3 (12)	3	4
Mid-flight	24 (70)	21 (84)	2	3
Landing	1 (3)	1 (4)	0	0
Cause of accident				
Collapse of the glider	9 (24)	9 (36)	0	0
Take-off error	8 (22)	2 (8)	3	3
Collision with object	6 (16)	4 (16)	0	2
Oversteering	4 (11)	2 (8)	1	1
Incomplete preflight check	2 (5)	2 (8)	0	0
Failed maneuver	2 (5)	2 (8)	0	0
Collision with flight object	1 (3)	1 (4)	0	0
Landing error	1 (3)	1 (4)	0	0
Improper maintenance	1 (3)	1 (4)	0	0
Unknown	3 (8)	1 (4)	1	1

Table 3. Rescue parachute use, pilot experience, and environmental parameters

	All cases n=37; n (%)
Rescue parachute	
On board	24 (65)
Used	5 (22)
Not used	19 (78)
Not on board	0 (0)
Unknown[a]	13 (35)
Experience	
Student pilot	1 (3)
Pilot license	20 (54)
Multiple pilot licenses	9 (24)
Professional	3 (8)
No license	2 (5)
Unknown	2(5)
Height of the event (m)[b]	
0–1000	4 (11)
>1000–2000	15 (41)
>2000–3000	12 (32)
>3000–4000	2 (5)
>4000	2 (5)
Unknown	2 (5)
Weather conditions	
Problematic	6 (16)
Unproblematic	22 (59)
Insufficient for classification	8 (22)
Unknown	1 (3)

[a]Note that in the cases in which the rescue parachute status was unknown, use of it was impossible.

[b]"Event" refers to the altitude where the problem started to develop. In the cases of take-off errors, the altitude of the launch site is indicated.

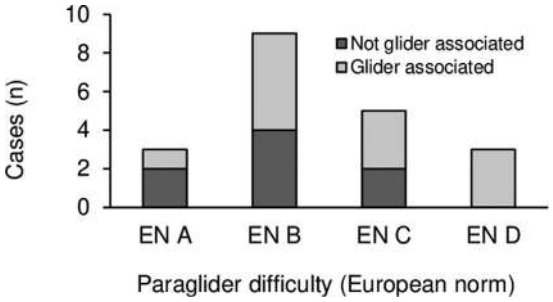

Figure 3. Number of fatalities and association with different glider difficulties in paragliding. Not glider-associated: incomplete preflight check, collisions; glider-associated: take-off errors, collapses, oversteering, spiral dive.

died within the first 2 y after receiving their license. The first 2 y after gaining a license are the most accident prone, irrespective of the number of completed flights.[18] This is despite speedflyers already requiring a paragliding license before speedflying training. Experience appeared to decrease the risk of some types of mishaps[5,7] while increasing the risks of others.[7,21] To reduce accidents, all pilots should strictly maintain use of the recommended checklists to avoid preventable mistakes and should take care in glider selection.[20]

Bad weather conditions or weather changes are often described as risk factors[6,7,18,22] and play a contributing role in up to 33% of all accidents,[23] becoming risk factors for deadly accidents. For base jumping, the environmental factors linked to fatal accidents were mostly a result of poor decision making.[24] Although 5 of 6 fatalities in bad weather conditions were thought to have been provoked by these conditions, most of the fatalities (59%) occurred in unproblematic weather conditions.

The famous Lauterbrunnen Valley attracts speedflyer pilots, and a similar aggregation is seen in base jumping. The base jumping fatality list compiled by BLiNC magazine, a community base jumping website, reported that 51 (56%) of 91 fatalities in Switzerland between 1994 and 2019 occurred in the Lauterbrunnen Valley. The popular region allows up to 10 base jumps per day, leading to a multiplication of the absolute risk.[24] The circumstances of speedflying fatalities in this challenging region show that these pilots tend to expose themselves to high-risk situations, either through the use of demanding and dangerous launch sites or through proximity flying.

The elevation exposure during the accident in 8 cases was between 2500 and 4100 m above mean sea level, referred to as moderate altitude.[25] These altitudes are potentially sufficient for some degree of hypoxia or even high altitude illness.[25-27] A systematic review suggested that hypobaric hypoxia at moderate altitudes may lead to cognitive and psychomotor deficits in learning, reaction times, decision-making, and certain types of memory.[25] However, the results at moderate altitude were inconsistent,[25] with reported thresholds for cognitive deficits ranging from 1525 m when exercising to 4572 m at rest.[28,29] There has been shown to be an increase in reaction time during exercise (80% peak oxygen consumption, $\dot{V}O_{2\ peak}$) in individuals at a normobaric hypoxia equivalent to 2200 m.[30] At moderate altitudes, $\dot{V}O_{2\ peak}$, respiratory rate, and heart rate are increased only during the take-off phase.[16] Combined with the physical exertion required to reach take-off sites by hiking or mountaineering, hypobaric hypoxia may have played a role in these take-off fatalities.

The medical and forensic data showed a high proportion of cases (54%) without any medical interventions. This suggests that the victims sustained from severe injuries from the accident. The causes of death

Table 4. Event description and assumed causes of fatal speedflying accidents

Case	*Event description*	*Assumed cause*
1	Take-off failure with crampons on Jungfrau (4100 m), slipped, glided over glacier, fall of over 50 m	Crosswinds disarranged the chute immediately before take-off
2	Take-off over cliff, stall, collapse of the wing, fall of over 80 m	Oversteering of the speedflyer
3	Cliff take-off failure, fall of over 120 m	Slipped with sneakers on wet grass
4	During take-off, fall in a crevasse	Choice of wrong take-off slope
5	Low flight, collision with a tree	Flying too low
6	Spiral dive not terminated until collision with the ground	Loss of consciousness or inability to terminate the spiral dive
7	Crash without injuries in hostile terrain, refused rescue by a helicopter, slipped, fall of off a cliff	Slipping in steep terrain

listed in Table 5 underline this suggestion on the basis of a high proportion of multiple trauma (54%). The literature is consistent about the fact that foot-launched flying sports accidents are often severe.[7,22] Hang gliding pilots seem to be more prone to head and neck injuries and tend to die from cerebrocranial trauma. Other studies have shown that hang gliding pilots are more likely to experience head and neck injuries than pilots in the other categories.[5,6] The high proportion of external-only inspections (74%) without autopsy did not reveal the exact cause of death, as would be expected from autopsies. Therefore, we are not able to discuss the importance of traumatic aortic rupture as a hidden and missed injury, as described in the literature.[13,31,32]

In comparison with a case of suicide, in which a skydiver manipulated his rescue system,[33] suicidal intentions were suggested in only 1 case with an unclosed harness. The suicide suggestion was ruled out later by witness testimony. In 1 case in which an autopsy was done, the accident mechanism showed the involvement of pre-existing medical conditions (coronary heart disease and pulmonary emphysema). In this case, the 64-y-old pilot flew without any steering impulses into a cliff. A case series also suggested that 1 of 6 skydivers who were

Table 5. Forensic examination and toxicological analysis, absolute and relative numbers

	All cases n=39; n (%)	*Paragliding n=27; n (%)*	*Hang gliding n=5; n*	*Speedflying n=7; n*
Body examination				
External inspection	29 (74)	19 (70)	3	7
Autopsy (with/without external inspection)	10 (26)	8 (30)	2	0
Toxicological analysis				
Tested	10 (26)	8 (30)	2	0
Positive	0 (0)	0 (0)	0	0
Negative	10 (100)	8 (100)	2	0
Not tested	29 (74)	19 (70)	3	7
Cause of death				
CCT	7 (18)	4 (15)	2	1
Multiple trauma with CCT	18 (46)	14 (52)	2	2
Multiple trauma	3 (8)	2 (7)	0	1
Fat embolism	3 (8)	3 (11)	0	0
Internal/External bleeding	3 (8)	1 (4)	1	1
Unclear	5 (13)	3 (11)	0	2
Resuscitation at accident site				
Yes	17 (44)	15 (56)	1	1
No	22 (56)	12 (44)	4	6
Absolute signs of death	21 (95)	12 (44)	4	6
Patient responsive	1 (5)	0 (0)	0	0

CCT, craniocerebral trauma.

autopsied could have lost consciousness owing the presence of coronary heart disease, outflow obstruction, and myocardial hypertrophy and may therefore have not opened the parachute.[34] However, there was no evidence of the influence of pre-existing medical conditions on the accidents. Therefore, because there is seldom danger to others, we do not recommend compulsory medical certificates, as is done in general aviation. An increased number of autopsies could help to give better-founded recommendations, in this regard.

To fully understand the causes and dynamics of foot-launched flying fatalities, a detailed examination of these cases is required.

LIMITATIONS

Data were gathered for nonresearch purposes by rescue teams, the police, and forensic doctors, and mostly without specific knowledge of foot-launched flying sports. Most of the causes of death were determined only by external inspection. Therefore, the medical results must be evaluated with caution and are only listed as the most probable cause of death. Records of pre-existing medical conditions and prescribed drugs were rare. Consequently, the data were incomplete, contained obvious assumptions, and included witness testimony. Nonpilot observations need to be assessed critically owing to possible misinterpretations.

The low number of accidents involving fatalities between 2000 and 2018, missing files, and the retrospective method used limited the value of the general statements about foot-launched flying sport fatalities.

The accidents occurred within a period of 20 y. The temporal dynamics, technical developments, adaptations of the education system, and other new security measures were not taken into consideration and may therefore have created a bias. Because the literature was sparse and not up to date, it was difficult to make comparisons with the results of this study.

Conclusions

The categories of foot-launched flying sports differed in the causes of accidents and causes of death. The causes of death appeared to be traumatic in nature, and all incidents were categorized as accidents. The deceased speedflying pilots were on average 18 to 23 y younger than the paraglider/hang glider pilots and often died in high-risk situations. High altitude take-off sites, incomplete or missing preflight checks, flying close to the slope, and use of demanding gliders were identified as potential risk factors. We see prevention possibilities in education and training, the implementation of preflight checklists, and familiarizing pilots with the technique of throwing the rescue parachute. We recommend that all pilots fly and choose their glider cautiously, considering their experience and their flying skills.

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Author Contributions: Design of the study (FS, CS); acquisition of the data (FS, CS); analysis of the data (FS); interpretation of the data (FS, CS); drafting of the manuscript (FS); critical revision of the article; approval of the final manuscript (FS, CS, CJ).

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ORIGINAL RESEARCH

Quantifying Risk in Air Sports: Flying Activity and Incident Rates in Paragliding

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Introduction—The volume, nature, and risks of paragliding are poorly quantified. More comprehensive understanding, including incident rates allowing comparison to similar disciplines, will help direct and appraise safety interventions.

Methods—Paraglider pilots were surveyed regarding experience, incidents, recordkeeping, and risk perception. The survey could not capture those who had left the sport or died, so a subset of responses from UK pilots was compared to records from an incident database.

Results—There were 1788 (25%) responses from 7262 surveyed. Respondents flew a total of 87,909 h in 96,042 flights during 2019. Local flying was most frequent (n=37,680 flights, 39%) but a higher proportion of hours were spent flying cross-country (n=33,933 h, 39%). The remainder were spent in competition, hike and fly, tandem, aerobatic, or instructional flight. Flying incidents led to 103 (6%) respondents seeking medical attention, attending hospital, or missing a day of work in 2019. Near misses were reported by 423 (26%) pilots. Asymmetry and rotational forces typically led to incidents, and limb and back injuries resulted. Pilots frequently failed to throw their reserve parachutes. Only 3 (0.6%) incidents involved equipment failure, with the remainder attributed to control or decision errors. Incident rates of paragliding were estimated as 1.4 (1.1–1.9) deaths and 20 (18–27) serious injuries per 100,000 flights, approximately twice as risky as general aviation and skydiving.

Conclusions—Incidents usually resulted from pilot error (control and decision), rather than equipment failure. Future safety interventions should focus on improving glider control skills and encouraging reserve parachute deployment.

Keywords: aviation, accidents, surveys and questionnaires, risk

Introduction

Paragliding is a growing discipline of unpowered flight and a popular wilderness activity. Equipment performance has rapidly improved since its inception in the late 1970s, and flights of several hours are now common, with the present distance record standing at 588 km in a single, 11-h, unpowered flight.[1] However, paragliding remains hazardous, with pilots at risk of collision with terrain or one another and at risk of environmental injury secondary to hypoxia, cold, or acceleration forces.[2] Increasing efforts are being made to match performance gains with safety initiatives.[3] There is a need for better epidemiologic information to assess the effectiveness of interventions and to make paragliding's risks comparable to those of related disciplines, such as general aviation and skydiving.

The Paraglider Manufacturers Association estimated that there were 127,000 active paraglider pilots worldwide in 2014.[4] At present, incident numbers are tallied from self-reports and published annually by national associations. Authorities (typically the police) are notified of fatalities, which are usually then investigated by representatives of national associations on their behalf. Incident numbers are less useful than incident rates, because rates indicate whether decreasing incident numbers represent a real improvement in safety or simply a change in flying frequency. One author arrived at a total of 47.1 fatalities per 100,000 participant-years in UK paragliding, based on membership data from the British

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Hang Gliding and Paragliding Association (BHPA).[5] However, paragliding, like other outdoor pursuits, is a diverse activity, and people fly in many different ways. Some pilots only fly locally in smooth laminar conditions, whereas others go long distances in turbulent air. Some focus on mountain flying, aerobatics, commercial tandem flying, or international competition. Some pilots fly most days, others once a year on vacation. Equipment choices may range from stable, certified gliders to high-performance prototypes. Because paraglider pilots do not file flight plans, the total flying time and time spent in these different types of flying are unknown. Estimations based on membership numbers do not consider the volume, type, or environment of the activity in question.

The most useful metrics in paragliding safety are the incident rates adopted by powered aviation, such as incidents per flight, hours, or kilometers flown. The goal of this study was to quantify the nature, volume, and risks of paragliding activities through a largescale survey and to conduct an analysis of a national incident database. The effects of activity, currency (recent experience), and risk perception on incidents was considered, and incident rates for fatalities and serious injuries by flight volume were derived.

Methods

The study made use of 2 distinct sources of data: a new survey and a pre-existing incident database. In the survey, a cohort of pilots from a controlled list was questioned to obtain a more complete picture of their flying activities. The survey tool, which could not capture those who had died, left the sport, or been too injured to respond, was combined with records from the European Hang Gliding and Paragliding Union (EHPU) incident database to provide additional detail on incident numbers and character.

A survey instrument (Table 1) was built in Survey Monkey (Momentive Inc, San Mateo, CA), in accordance with the recommendations in the international handbook of survey methodology for list-based surveys of high-coverage populations.[6] The survey was in 4 parts. The first captured demographics and primary discipline of flight. Only those who chose paragliding and provided answers relating to paragliding were included in the results. The second quantified total experience, the third focused specifically on flying activities in the year 2019, and the final part focused on incidents in 2019. Participants were asked how their responses were generated (from memory or written records), and the survey concluded with a single question about perception of risk. The instrument was piloted with 20 participants and received ethical approval from the University of Portsmouth science faculty research ethics committee (SFEC 2019-115).

The population surveyed was drawn from the readership database of *Cross Country* magazine (Cross Country International Ltd., UK), the largest international free-flight publication. Their database was fully compliant with the general data protection regulations, with explicit opt-in permission for web surveys, and activity of the registered email addresses was checked annually, allowing for accurate calculation of response rate. An individual email invitation and unique login were sent to all 7262 readers who had opted in for web surveys on February 24, 2020. A single reminder email was sent 5 d later, and data collection was closed 14 d later (after 72 h with no further responses). The data collection period was kept short to minimize variation in the length of recall required for pilots recounting their 2019 experiences. Comprehensible partial responses were included in the analysis where possible, and the number of included responses is expressed as n values in the results.

BHPA entries on the EHPU European incident database were downloaded on January 27, 2020, after further ethical review (UoP ethics screening tool ETHICS-10049). The download included 1296 reports, of which 1034 related to paragliding, dated between January 8, 2012 and January 21, 2020. The reports in which the pilots were uninjured were manually reviewed to exclude reports relating to nonflying incidents (eg, equipment issues identified during servicing) and reports of fatal injury were screened to exclude deaths unrelated to paragliding. Fatal injuries were defined (by the database) as death "within 30 d following the accident." Serious injuries were defined as "more than 48 h in hospital, fractures (except for finger, nose, toe), hemorrhage, ligament rupture." Other data fields included flight type, pilot age and sex, total weight in flight, nationality, pilot rating and courses, flying hours, glider and reserve parachute, incident specifics, and witness accounts. The BHPA also provided member numbers for the years 2012 to 2019 and an anonymized version of its current member database (to February 7, 2020), which included members' age, sex, and pilot rating.

Incident rates per flight and per hour were estimated based on the number of incidents (nonfatal and fatal) for BHPA pilots on the EHPU database as the numerator and the flying activities reported by the 368 BHPA pilots who responded to the survey as the denominator. Only survey responses from BHPA pilots were used in this comparison. Definitions from the human factors analysis and classification system (HFACS) were used to classify causal factors in incident reports.[7] The HFACS taxonomy was developed by for the United States Navy and Marine

Table 1. Survey questions and responses

How old are you? 48±12 y
What is your gender? [Single select]
Female: 101 (6%); male: 1543 (93%); prefer not to say: 11 (1%)
Are you a member of any of these flying associations? [Multiselect]
APPI: 136 (8%); BHPA: 368 (22%); DHV: 76 (5%); FFVL: 4 (6%); FSVL: 73 (4%); USHPA: 213 (13%); other: 710 (42%); not a member: 136 (8%)
How many years have you been flying [paragliders]? 10 (4–20) y
How many [paragliding] flying hours do you have in total? 350 (123–900) h
What proportion of your flights do you log? [Single select]
All: 805 (48%); most: 461 (28%); some: 173 (10%); few: 115 (7%); none: 99 (6%)
Which of these do you use to log your flights? [Multiselect]
Logbook (paper or electronic): 865 (52%); flight instrument: 868 (52%); online league: 577 (34%); other: 113 (7%); I don't log my flights: 105 (6%)
How many flight hours in total for 2019? 42 (20–80) h
How many flights in total for 2019? 44 (24–80) h
What kind of glider did you use for the majority of your flights? [Single select]
EN-A: 89 (5%); EN-B (low): 308 (19%); EN-B (high): 526 (32%); EN-C: 304 (18%); EN-D: 131 (8%); other: 83 (5%); unspecified: 214 (13%)
What percentage of your flying hours was spent doing each of the following types of flying? [%]
Under instruction: 6±16; local flying: 48±32; cross-country flying: 37±28; cross-country competition: 12±20; acrobatics: 2±8; hike and fly: 10±16; tandem flying: 3±9; commercial tandem flying: 4±15
What percentage of your flying hours was spent in each of the following conditions? [%]
Still air flights: 16±20; soaring flights: 30±25; thermal flights: 62±27
How were your flying days distributed through 2019? [Single select]
Short bursts (eg, holidays only): 208 (13%); flying season only (not the off-season): 307 (19%); throughout the year: 922 (56%); unspecified: 218 (13%)
What was your longest gap between flights in 2019? 7 (4–12) wk
Did you have to seek first aid or medical attention, attend hospital, or miss a day of work in 2019 because of a flying incident? [Single select]
Yes: 103 (6%); no: 1336 (81%); unspecified: 216 (13%)
If yes, did you have to stay in hospital for more than 24 h? [Single select]
Yes: 28 (2%); no/NA: 1233 (74%); unspecified: 404 (24%)
If yes, please provide brief details of the incident(s), including the circumstances and injury(s). [Free text]
Have you had a "near-miss" this year? [Single select]
Yes: 423 (26%); no: 1009 (61%); unspecified: 223 (13%)
If yes, please provide brief details of the near miss(es) [Free text]
How do you feel about the risks of your primary discipline of free flight? [Slider from "Certain you will never be injured" {0} to "Certain you will be injured at some point" {100}]
54 (34–70)
How did you generate your responses to this survey? [Slider from "From memory" {0} to "From records" {100}]
58 (7–88)

Respondents were asked at the start "What is your main discipline of flying? (The discipline of flying that you spent the most time doing in 2019.)" Only those who chose paragliding (1655 respondents) and provided answers relating to paragliding were included in the results. "Flying hours" were defined as "the time spent in the air between take-off and landing." A "flight" was defined as "a distinct launch and then landing (not a 'touch-and-go' or wagga)." A "near miss" was defined as "an unplanned event that had the potential to cause injury but did not result in an accident. For example, a massive collapse close to the ground, nearly colliding with another pilot, or being dragged along the ground." The responses are presented as number of responses, unless specifically indicated as mean±SD or median (interquartile range). Pilots might be members of multiple associations or have multiple logging methods, so percentages add up to more than 100.

APPI, Association of Paragliding Pilots and Instructors; BHPA, British Hang Gliding and Paragliding Association; DHV, Deutscher gleitschirm und drachenflugverband e.v. (German Paragliding and Hang Gliding Federation); FFVL, Fédération Française de vol libre (French Federation of Free Flight); FSVL, Fédération Suisse de vol libre (Swiss Federation of Free Flight); USHPA, United States Hang Gliding and Paragliding Association.

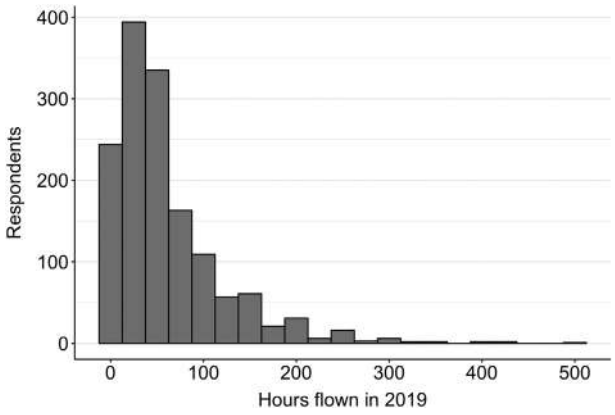

Figure 1. Histogram of hours flown by respondents in 2019 (n=1455).

Corps as an incident investigation tool. The taxonomy described 4 levels of human failure, 1 active and 3 latent: unsafe acts, preconditions for unsafe acts, unsafe supervision, and organizational influences. The active failures, unsafe acts, were divided into errors and violations. Errors represented a failure to achieve a goal, and violations were a deliberate deviation from prescribed practice. Although unsafe acts might have led directly to the incidents, they occurred in the context ("preconditions") of a particular set of environmental, physical, mental, and social conditions and supervisory and organizational cultures.

Data were analyzed using R Studio (V1.0.143, R Project for Statistical Computing, R Core Development Team, V 3.4.1), with advice from a statistician. Distribution of results was assessed using descriptive methods (skewness, outliers, and distribution plots) and inferential statistics (Shapiro-Wilk test). Data were presented as mean±SD if normally distributed (Shapiro-Wilk test nonsignificant) and as median (interquartile range) if not normally distributed (Shapiro-Wilk test significant). Logistic regression was performed to ascertain the effects of flying hours, currency, and risk perception on the likelihood of respondents reporting an incident or near miss in the survey. Significance (alpha) was set at $P<0.05$. Free-text fields were iteratively coded and then assessed quantitatively.

Results

Of the 7262 email invitations, 4914 (68%) were opened, 2028 (28%) were clicked through, and 1788 survey responses were received. The overall response rate was therefore 25%. There were 1524 complete responses (86%) and 254 partial responses (14%), and the time taken to complete the survey was 7 min (standard deviation not reported by survey tool).

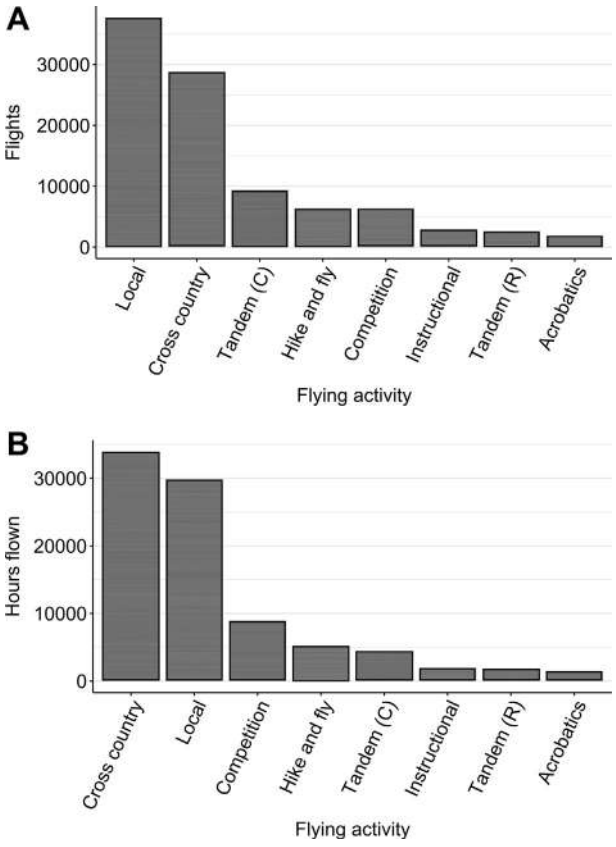

Figure 2. (A) Frequency of flights (n=1428) and (B) hours (n=1455) flown by respondents in 2019, broken down by activity. "Tandem (C)" is commercial, and "Tandem (R)" is recreational. "Flights" were defined as a distinct launch and then landing (not including "touch-and-go" flights) and "flying hours" as time spent in the air.

Of the 1788 respondents to the survey, 1655 flew paragliders as their primary discipline of flight and were included in the analysis. The age of these paraglider pilots was 48±12 y; 1543 were male (93%), 101 were female (6%), and 11 (0.6%) declined to specify. Results for total years and hours flying were right skewed: years flying was 10 (4–20) y, and total hours was 350 (123–900) h.

The 2019 results also had a right-skewed distribution. Total hours was 42 (20–80) h (Figure 1), and number of flights was 44 (24–80). The gap between flights in 2019 was 7 (4–12) wk, and the majority of pilots (n=922, 56%) flew throughout the year, rather than seasonally.

Respondents reported that they flew a total of 87,909 h in 96,042 flights during 2019. In addition, they were asked to indicate the percentages of their flying hours spent in engaging different types of paragliding flight. Most flights were local (37,680 flights, 39%, Figure 2A), but because of longer flight times, a higher proportion of hours was spent in cross-country (33,933 h, 38%) rather than local flying (23,845 h, 34%, Figure 2B).

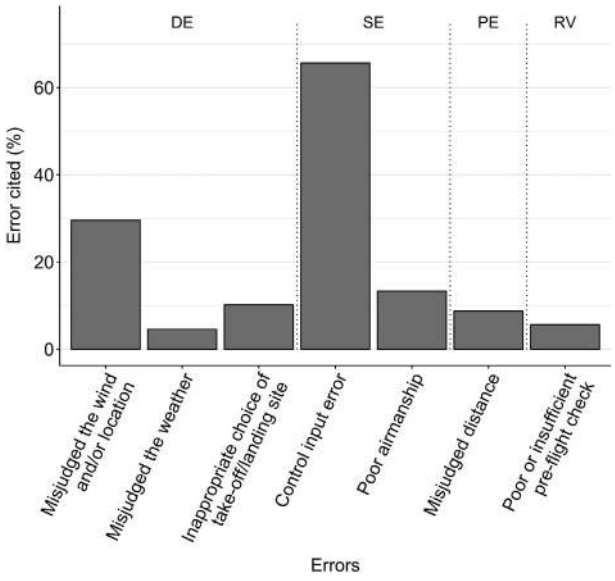

Figure 3. Reported errors from 67 incidents and 413 near misses (number of times each error cited, expressed as a percentage of the total number of incidents). Because the incidents often included more than 1 causal factor, the percentages in the figure do not add up to 100%. DE, decision error; SE, skill error; PE, perceptual error; RV, routine violation.

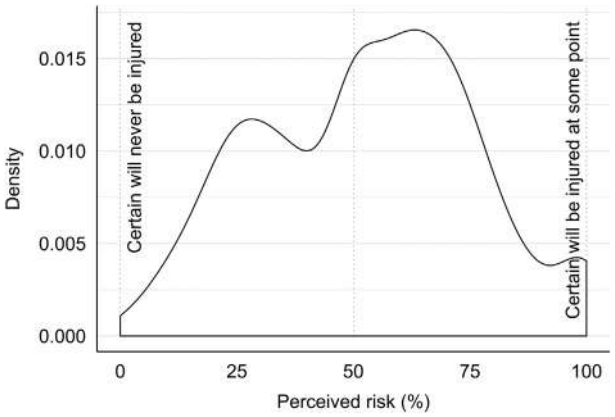

Figure 4. Density plot to demonstrate the distribution of perceived risk of paragliding (n=1226), from 0% ("certain will never be injured") to 100% ("certain will be injured at some point").

Of the total hours flown, the majority were in thermal conditions (n=59,167 h, 68%) followed by soaring (n=19,663 h, 22%) and still air ("top to bottom" flights [n=8,880 h, 10%]). Half of respondents (n=834, 50%) used a wing from the EN-B class. This is a midrange class of paraglider, designed to balance performance with safety and certified according to European Standard EN-926.[8] The remainder used a mix of beginner, more advanced, or specialist wings.

One hundred three respondents (6%) reported having to seek first aid or medical attention, attend hospital, or miss a day of work in 2019 because of a flying incident. Twenty-eight (2%) had to stay in hospital for more than 24 h. Four hundred twenty-three (26%) reported 1 or more near misses, defined as "unplanned events that had the potential to cause injury but did not result in accident." Considered in terms of hours flown, that equated to 31 serious injuries, 117 incidents, and 481 near misses per 100,000 h—or 29 serious injuries, 107 incidents, and 440 near misses per 100,000 flights, based purely on the survey data.

Sixty-seven (65%) reported details of their incidents. Three incidents (4%) led to severe polytrauma, and the remainder involved isolated (but in many cases serious) injuries to the limbs (n=45, 67%), spine (n=5, 8%), head (n=2, 3%), pelvis (n=2, 3%), and ribs (n=2, 3%). Four hundred thirteen (98%) reported details of their near misses. Only 3 of the 480 incidents (0.6%) and near misses involved equipment failure. The remainder included 1 or more categories of pilot error (Figure 3). When the causal factors were classified using definitions from the HFACS, the majority of incidents included skill-based (n=379, 79%) and decision-based (n=213, 44%) errors.[7] Control input errors were most frequently reported (n=315, 66%), followed by misjudgment of wind strength or aerology (n=142, 30%). One-hundred forty-two reports (30%) included a collapse, 28 (6%) a stall, and 27 (6%) a spin. Midair collisions or near misses, top-landing, and kiting the glider in strong winds were also recurring themes.

Respondents were asked to rate their perception of paragliding's risk using a slider, between 0 ("certain you will never be injured") and 100 ("certain you will be injured at some point"). The slider was initially positioned at 50, midway between the 2 extremes. The distribution of responses is displayed in Figure 4, and the perceived risk was 54 (34–70).

Logistic regression was performed to ascertain the effects of flying hours, currency, and risk perception on the likelihood of respondents reporting an incident or near miss in the survey. The fit of the logistic regression model was statistically significant (χ^2 (3)=45.0, P<0.001). There were statistically significant increases in the likelihood of reporting an incident or near miss with increasing hours and increased perception of risk, and a reduction with a lengthening gap between flights (Table 2). Expressed in terms of odds, for each additional hour flown, the odds of having an incident or near miss were fractionally increased by a factor of 1.003; with every additional week's gap between flights, the odds of having an incident or near miss were fractionally reduced by a factor of 0.976. Equally, with every percentage-point increase in

Table 2. Logistic regression of the effects of flying hours, gap between flights, and perceived risk on the likelihood of reporting an incident or near miss

Parameter	*Maximum likelihood estimates*					*Odds ratio estimates*	
	df	*Estimate*	*SE*	*Wald* χ^2	*Pr*>χ^2	*OR*	*95% CI*
Hours	1	0.002	0.001	5.8	0.016	1.002	1.000–1.005
Gap	1	−0.030	0.009	7.4	0.0067	0.976	0.959–0.993
Risk	1	0.013	0.003	22.8	<0.0001	1.013	1.008–1.019

perceived risk, the odds of having an incident or near miss were fractionally increased by a factor of 1.013.

Respondents were asked what proportion of their flights had been logged, on a 5-point scale (from "none" to "all"). The majority (n=1266, 77%) of respondents logged "most" or "all" of their flights. Respondents used 1 (or a combination of) flight instruments (n=868, 52%), paper or electronic logbooks (n=865, 52%), online tools (n=577, 35%), or other means (n=113, 7%). They were also asked how to indicate how their responses to the survey were generated (Figure 5), using another slider question (from 0 ["from memory"] to 100 ["from records"]). The responses clustered around each of the extremes. This was also reflected in, for example, the hours data, in which responses tended to either be very precise or were rounded to the nearest 10 or 100 h.

The survey data yielded a rate of 29 incidents leading to serious injury per 100,000 flights. However, as described in the introduction, the survey could not have captured incidents severe enough for a pilot to have left the sport midway through the year or fatal incidents. Consequently, the flying activities reported by the BHPA respondents to the survey were used as a denominator for the EHPU incident database reports, which included midyear reporting and fatalities. Three-hundred sixty-eight (22%) of the respondents to the survey were members of the BHPA. Their age was 52±11 y, and they had been flying 12 (5–21) y and 373 (121–900) h.

In 2019, they flew 35 (16–70) h and 41 (21–70) flights each, making a total of 18,452 h in 20,382 flights. The majority flew throughout the year (n=214, 58%) on EN-B wings (n=193, 52%), and the longest gap between flights was 8 (5–13) wk. Eighteen BHPA members (5%) reported an incident, 5 (1%) involving a hospital stay longer than 24 h. Eighty-seven (24%) reported near misses. In these parameters, the BHPA subset was similar to the rest of the cohort surveyed (BHPA members flew slightly fewer hours per year and were slightly older) and to BHPA membership data for the same time-period.

If the activities of the BHPA respondents could be considered typical of BHPA members in general, then it could be estimated that the total hours flown by all BHPA members in 2019 was 188,475 h in 218,903 flights. In the EHPU incident database, there were 3.0 (2.5–4.3) fatal injuries and 44 (40–60) serious injuries per year among BHPA members in 2012–2019. The rates would therefore be 1.6 (1.3–2.3) fatalities and 23 (21–31) serious injuries per 100,000 h of paragliding, or 1.4 (1.1–1.9) deaths and 20 (18–27) serious injuries per 100,000 flights among UK paraglider pilots.

The database offered some additional insight into incidents, which corroborated the findings of the survey. Of the 1000 incidents reviewed, 26 (3%) had fatal, 387 (39%) serious, and 248 (25%) minor injuries, and in 339 (34%) cases the pilot was unhurt. Most injuries occurred during local flights (n=587, 59%) and training flights (n=245, 25%), rather than cross-country flights (n=62, 6%). The landing approach and landing were the flight phases most likely to lead to injury (n=393, 39%), followed by takeoff (n=206, 21%) and ridge soaring (n=189, 20%). Fatal injuries were predominantly those to the head and body, whereas serious and minor injuries were typically to the lumbar spine or limbs. The type of glider instability that led to injury was only specified in 440 reports (44%), but asymmetric collapse was most common (n=165, 38%), followed by spin (n=61, 14%) and frontal collapse (n=52, 12%).

Information on reserve parachute deployment was available for 270 (n=65%) of 413 incidents in which the pilot sustained serious or fatal injuries. The reserve

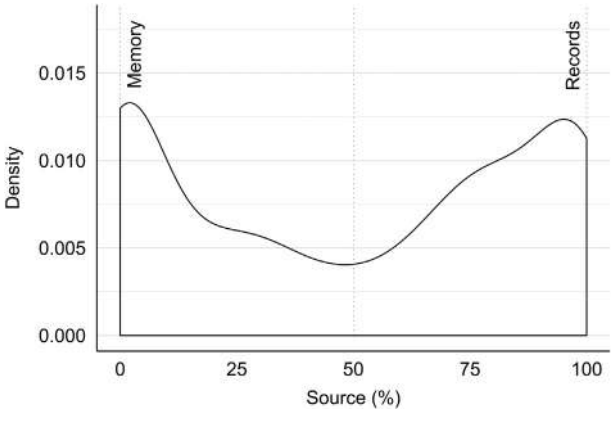

Figure 5. Density plot to demonstrate the distribution of how responses to questions were generated (n=1344), from 0% ("memory") to 100% ("from records").

Table 3. Comparison of paragliding errors to those from commercial and general aviation

Error	*Survey n=480*	*Commercial aviation[12] n=1020*	*General aviation[13] n=14,436*
Decision-based error	213 (44%)	374 (37%)	3854 (30%)
Skill-based error	379 (79%)	576 (57%)	11,433 (79%)
Perceptual error	42 (9%)	66 (7%)	823 (6%)
Routine violation	27 (6%)	236 (23%)	

Frequencies (percentages) of causal factors in incidents, alongside analyses from commercial and general aviation classified by HFACS categories. Because the incidents often included more than 1 causal factor, the percentages in the table do not add up to 100%.

parachute was not deployed in 256 incidents (95%). In the remaining 14 incidents, the reserve was deployed accidentally twice (causing injury) and deliberately on 12 occasions. Where deliberately deployed, it opened successfully on 6 occasions; there was insufficient altitude on 5 occasions but only a single "failure to open." It could not be gathered from the data whether any of the fatally injured pilots had attempted to deploy their parachutes but failed.

Only 48 (5%) of the 1000 incident reports were due to equipment failure, of which 20 related to the harness, 15 to the wing, and 5 to the reserve parachute deployment handle. Seven incidents related to a failure of tow launching equipment. Eight hundred sixty-three (86%) reports alluded to some aspect of pilot error.

Discussion

In the survey, most flights were local, and most flying hours were spent in cross-country flight. (Cross-country flying involves leaving the vicinity of the takeoff hill to search for lift elsewhere, typically leading to a longer flight.) This distinction was important when considering "exposure" to paragliding, as exposure to the risks of launch and landing might be best expressed by numbers of flights, whereas exposure to the risks of flying would be better represented by time spent in the air.

There is a school of thought that pilots who fly regularly, building up hours while being acutely aware of risk, are less likely to be involved in incidents than those who fly rarely and perceive the sport to be very safe. These data suggested the contrary: that the likelihood of reporting an incident or near miss increased fractionally for every additional hour flown and decreased fractionally with every additional week's gap between flights. This ran counter to the "currency" model ("the more flying, the safer the pilot") and instead pointed to an exposure model ("the more flying, the greater the risk exposure"). Intriguingly, with every percentage-point increase in perceived risk, the odds of having an incident or near miss were fractionally increased by a factor of 1.013. The distribution of hours in the sample was skewed heavily toward beginners, and it may be that these effects would no longer persist once pilots reached mastery, which might require 2000 h or more.[9]

Paragliding appeared almost twice as risky as general aviation in the United Kingdom, which recorded 0.73 deaths and 11.2 serious injuries per 100,000 flights between 2012 and 2016.[10] It also had approximately twice the fatality rate of sport parachuting (skydiving), where the rate of fatal injuries was 0.74 per 100,000 in an analysis of 539,885 jumps (excluding tandem jumps, military parachuting, and airplane crashes).[11]

The overwhelming majority of reports, in both the survey and incident database, alluded to some form of pilot error. As with the aforementioned incident rates, to make paragliding more comparable to other forms of aviation, these errors were grouped in the results using HFACS terminology. Despite the differences between paragliding and other forms of aviation, and the shortcomings of self-reported paragliding data, the results were like those from past analyses of commercial and general aviation in the literature (Table 3), classified by HFACS categories.[12,13] Skill-based errors, in particular control input errors, were the most common. The higher number of decision errors and the lower number of violations in paragliding spoke of the greater responsibility placed on paraglider pilots to choose how, when, and where it was appropriate to fly, as well as the lack of rules, regulations, and procedures compared to other forms of aviation. The higher number of perceptual errors was again expected, given the reduced reliance on instrumentation in paragliding, although it was interesting to note that misjudgment of distance was also the most common perceptual error in general aviation.[13] Equally, in a detailed analysis of 125 sport parachuting fatalities, the most frequent causes of incidents were control errors, mid-air collisions, loss of altitude awareness, poor choice of equipment, and medical incapacitation.[14]

Unlike errors, which had much in common with other disciplines, the distribution of injuries was specific. This reflected both the energy and orientation of the impacts involved. With the pilot suspended in a harness and likely to impact vertically or with legs down, forces would be

transmitted up through the lower limbs, pelvis, and back, with fractures at points of biomechanical vulnerability. Isolated limb and spinal injuries were the most common serious injuries reported. Indeed, lower limb fractures and compression fractures of the vertebrae at the junction of the thoracic and lumber spine (T12–L2) have also been reported in other canopy sports.[15,16]

Thirty percent of the incident and near miss reports included a wing collapse. This emphasized the importance of rotational forces and asymmetry in paragliding incidents, the necessity for pilots to train in the prevention and timely management of collapses, and for emergency equipment and procedures to be considered in that context. Strikingly, the reports in the EHPU incident database indicated that the reserve parachute was rarely deployed, even where it might have prevented injury or death. The foundation laid by this study, including the mapping of error to HFACS, is a bridge to more rigorous causal analysis in paragliding incidents. Traditional responses to incidents have focused on the unsafe acts themselves, be they errors or violations of procedure. These, by definition, were always too late.

LIMITATIONS

The study had 3 key limitations: low response rate, self-reporting, and limited generalizability. The response rate was low (25%, n=1788). This may have been due to the short window for response, the subject matter, or the perceived relevance or arduousness of the survey.

The organization and reporting structures in paragliding are patchy, reflected in the limited information available from the EHPU incident database. Reliance on self-reporting was therefore necessary (except for fatal incidents). Self-reporting is particularly problematic in extreme sports, given the limited training and the complex, dynamic environment.[17] Pilots may be unaware of all the factors that led to an incident, may misattribute their significance or their interactions, or may fail to correctly recall their own reactions. When flying solo, impairment due to environmental stress or medical incapacitation may go unrecorded. Because respondents were surveyed retrospectively rather than prospectively, their responses to the risk perception question may have been directly colored by whether they had an incident during 2019. Equally, any incidents or injuries had not caused respondents to leave the sport or stop their subscription to the magazine.

Finally, the results may not have been generalizable to the broader community of pilots. The respondents were a self-selected cohort who had already chosen to subscribe to a free flight publication. *Cross Country* magazine was written in English and the invitation and survey were delivered in English, so the study was biased toward English speakers. Respondents may have been more engaged with the sport (by virtue of subscribing to a magazine) than other pilots. Equally, they have been more or less experienced. There was no accepted definition of the "average pilot" against which to compare. Concerns around the generalizability of the sample were particularly acute for the incident rate estimates because they used the BHPA subset and the 368 BHPA respondents only equated to 7% of the 5385 members of the BHPA in 2019.

Given these limitations, both the incident rates and the conclusion of the logistic regression should be treated with caution and refined through continued study. Future approaches might include making a pilot's annual flying license renewal contingent on providing activity and incident data or, alternatively, following a cohort of pilots prospectively. The cohort would be asked to log all flying activities, incidents, and near misses over several flying seasons (to account for variation in weather conditions). Then their reports could be corroborated with data from their flight instruments. This approach would also help assess the effectiveness of existing reporting systems.

Conclusions

It appeared that increased flying hours exposed pilots to a slight increase in risk, contrary to the belief that more flying reduces risk. Pilot errors in glider control and in decision-making, not in equipment malfunction, were the key issues, consistent with other disciplines. Future safety interventions should focus on improving back protection, pilot training, and glider control and encouraging timely reserve parachute deployment.

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WILDERNESS MEDICAL SOCIETY CLINICAL PRACTICE GUIDELINES

Wilderness Medical Society Clinical Practice Guidelines on Anaphylaxis

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The Wilderness Medical Society convened a panel to review the literature and develop evidence-based clinical practice guidelines on the treatment of anaphylaxis, with an emphasis on a field-based perspective. The review also included literature regarding the definition, epidemiology, clinical manifestations, and prevention of anaphylaxis. The increasing prevalence of food allergies in the United States raises concern for a corresponding rise in the incidence of anaphylaxis. Intramuscular epinephrine is the primary treatment for anaphylaxis and should be administered before adjunctive treatments such as antihistamines, corticosteroids, and inhaled β agonists. For outdoor schools and organizations, selecting a method to administer epinephrine in the field is based on considerations of cost, safety, and first responder training, as well as federal guidelines and state-specific laws.

Keywords: epinephrine, autoinjector, food allergy, insect bites and stings, wilderness medicine

Introduction

Accounts of anaphylaxis date back to the earliest recorded history. Hieroglyphs from 2640 BC depict the pharaoh Menes dying after a wasp sting.[1] Today, anaphylaxis continues to be a serious medical issue. An estimated 2 to 5% of the US population has experienced anaphylaxis. In addition, between 1999 to 2010, there were a total of 2458 anaphylactic deaths, a figure that may reflect underreporting. Although such deaths appear to be rare, estimated at 0.1% of all emergency department (ED) visits and 1% of all hospital admissions for anaphylaxis, the potential for sudden and unpredictable fatality is an ever-present concern for at-risk individuals and their families.[2]

In remote areas or wilderness settings, access to standard medical care may be limited or delayed. To help increase the availability of life-saving treatment, the Wilderness Medical Society published clinical practice guidelines in 2010 and 2014 supporting the concept that nonmedical professionals whose duties include providing first aid or emergency medical care in the field also be trained to administer epinephrine for anaphylaxis.[3,4] Examples of such professionals include expedition leaders, outdoor instructors or guides, park rangers, and camp directors.

The current guidelines expand the focus from the administration of epinephrine by trained nonmedical professionals to the broader field treatment of anaphylaxis, with consideration for hospital-based treatment.

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Methods

Anaphylaxis, with its potentially drastic course, does not lend itself to study in randomized, controlled trials. The authors reviewed the literature for the best available evidence, including observational studies, case series, limited controlled trails, extrapolation from physiological data, and expert consensus. Practice recommendations were assigned a level of evidence according to the

methodology proposed by the American College of Chest Physicians (online Supplemental Table).[5]

Definitions and Scope

Emphasis in the guidelines is placed on the field treatment of anaphylaxis. Treatment of asthma and various non-anaphylactic allergic reactions are beyond the current scope. Nonetheless, the field practitioner will note some overlap in pathophysiology and treatment along the spectrum of allergic, asthmatic, and anaphylactic reactions.

- *Allergen.* An environmental substance that triggers an abnormal or heightened immune response in susceptible individuals. Common sources of allergens include foods, plant or animal elements, and medications.
- *Allergy.* An abnormal or heightened immune response against an allergen.
- *Anaphylaxis.* An acute, potentially-life threatening response to an allergen that progresses to involve multiple organ systems and is described in further detail later.
- *Asthma.* Abnormal bronchial constriction and inflammation arising from exposure to an inciting allergen, infection, extremes of temperature, or physical exertion.
- *Anaphylactoid reaction.* An acute inflammatory or anaphylaxis-like response without prior exposure to the inciting allergen.
- *Angioedema.* Subcutaneous or submucosal swelling and inflammation arising from exposure to an allergen or deficiency of an inflammatory inhibitor.
- *Antigen.* A substance or agent that incites an immune response with antibody production. An antigen may be environmental, as in allergens, bacteria, and viruses, or intrinsic to the body, as in autoimmune diseases.
- *Hypersensitivity reaction.* A heightened immune response against an antigen leading to inflammatory damage to the body. A hypersensitivity reaction may be immediate or delayed and is classified according to the specific types of antibodies or immune cells involved (Table 1).[6]

Epidemiology

In the United States, accurately determining the epidemiology of anaphylaxis is limited by the lack of a comprehensive national registry. National estimates of anaphylaxis are based on extrapolation from regional epidemiological surveys, with considerably varying results. A midrange estimate of the anaphylaxis risk in the US population is 1.6 to 2%.[7,8] Based on a population of 326 million (2020 US Census), 5.2 to 6.5 million individuals are theoretically at risk.

Exact numbers for anaphylactic deaths are also difficult to determine. In the United States, yearly estimates range from 205 (based on death certificate diagnoses) to 1500 (based on extrapolation from regional surveys).[9,10] Although the accuracy of individual estimates is questionable,[11] the range of 205 to 1500 deaths per year casts fatal anaphylaxis as a tragic but much smaller subset of the total incidence of anaphylaxis.

In children and adolescents, food allergies cause the majority of anaphylactic deaths, and asthma is a risk factor for fatal anaphylaxis.[12] The most common food allergies are to peanuts, milk, shellfish, and tree nuts. In adults and the elderly, medications and radiocontrast media cause the majority of anaphylactic deaths, often in hospital or healthcare settings. Penicillin and cephalosporin antibiotics are the medications most often implicated.[10] In both children and adults, *Hymenoptera* stings are the second leading cause of anaphylactic fatalities.[13] Overall, an estimated 45 to 150 deaths per year have been attributed to food allergies and insect stings, and 121 deaths to medications and radiocontrast media.[10,14]

The incidence of anaphylaxis specifically in wilderness settings is unclear, given the lack of a national reporting system. Illustrative examples, however, may be found in the injury and illness databases of 2 large, well-established schools in outdoor education and recreation.

NATIONAL OUTDOOR LEADERSHIP SCHOOL

Enrolling 5093±190 (mean±SD) students each year, the National Outdoor Leadership School (NOLS) teaches outdoor skills and leadership through wilderness-based courses and expeditions (https://nols.edu/en/). The majority of students have been young and healthy (median age: 20 y, IQR: 10). From 2005 to 2019, NOLS recorded 21 anaphylaxis cases in the field, occurring in approximately 0.03% of all students. In addition, 3 cases occurred among NOLS instructors and expedition leaders. Including both students and instructors, the total incidence of anaphylaxis was 24 cases per 2,431,591 person-days, or 10 per million person-days (1 student or 1 instructor in the field for 1 d=1 person-day) (Table 2).

Specific causes for the 24 cases are listed in Table 3. Overall, 13 cases (54%) were attributed to food-based allergies and 8 (33%) to *Hymenoptera* stings. Of note, 5 cases (21%) were first-time reactions in persons without a

Table 1. Hypersensitivity reactions

Reaction type	*Type I*	*Type II*	*Type III*	*Type IV*
Name	IgE-mediated hypersensitivity	IgG-mediated cytotoxic hypersensitivity	Immune complex–mediated hypersensitivity	Cell-mediated hypersensitivity
Mechanism	IgE antibodies activate mast cells	IgG antibodies activate T cells and complement	Antigen–antibody complexes activate complement and neutrophils	Antigens activate T cells and macrophages
Onset	Immediate (within minutes)	Intermediate (minutes to hours)	Intermediate (hours)	Delayed (48–72 h)
Clinical example	Anaphylaxis	Blood transfusion reaction	Serum sickness	Contact dermatitis, poison ivy

Adapted from Punt et al.[120]

known history of allergy. No anaphylactic deaths occurred among students or instructors. (All NOLS data provided by TS and DL)

OUTWARD BOUND—UNITED STATES

Enrolling 41,732±2427 (mean±SD) students each year, Outward Bound (OB) charters 11 regional schools across the United States that lead expeditions and outdoor-based courses in varied settings (https://www.outwardbound.org). The median student age is 16 y (IQR: 4). From 2005 through 2019, OB recorded 39 anaphylaxis cases in the field, occurring in approximately 0.01% of all students. In addition, there were 7 cases among field instructors. However, only the student incidence of anaphylaxis is available, which was 39 cases per 1,839,727 person-days, or 21 per million person-days (Table 2).

Specific causes for the 46 anaphylaxis cases among students plus instructors are listed in Table 3. These include 4 cases (9%) of students with a history of asthma who received epinephrine for respiratory distress, because distinguishing anaphylaxis with respiratory involvement from a severe asthma exacerbation may be difficult in the field. Overall, 11 cases (24%) were attributed to food-based allergies and 20 (43%) to *Hymenoptera* stings and insect bites. Of note, 10 cases (22%) were first-time reactions in persons without a known history of allergy. No anaphylactic deaths occurred among students or instructors. (All OB data provided by CBS)

Table 2. Anaphylaxis at the NOLS and OB, 2005 through 2019

	NOLS	*OB*
Students		
Annual enrollment (mean±SD)	5093±190	41,732±2427
Age, y (median) [IQR] (range)	20 [10] (6–84)	16 [4] (10–78)
Anaphylaxis cases in all students, n (%)	21 (0.03%)	39 (0.01%)
Field time (p-d)	1,945,057	1,839,730
Anaphylaxis incidence (per p-d)	1/96,622	1/46,173
(per million p-d)	10	21
Instructors		
Age, y (median) [IQR] (range)	32 [10] (20–72)	27 [7] (18–77)
Anaphylaxis cases in all instructors, *n*	3	7
Field time (p-d)	486,534	NA
Anaphylaxis incidence (per p-d)	1/162,178	NA
(per million p-d)	6	
Totals (students + instructors)		
Anaphylaxis cases, n	24	46
Field time (p-d)	2,431,591	NA
Anaphylaxis incidence (per p-d)	1/101,316	NA
(per million p-d)	10	
Cases per year (mean±SD) (range)	2±2 (0–10)	3±2 (0–8)
Anaphylaxis deaths	0	0

IQR, interquartile range; NA, not available; NOLS, National Outdoor Leadership School; OB, Outward Bound; p-d, person-day (1 student or 1 instructor in the field for 1 d).

Table 3. Causes of anaphylaxis at NOLS and OB, 2005–2019

Allergen	*Cases at NOLS n (%)*	*Cases at OB n (%)*
Hymenoptera/Insect stings	8 (33)	20 (43)
Peanuts/Tree nuts	8 (33)	7 (15)
Other foods	5 (21)	4 (9)
Plants/Pollen/Grasses	1 (4)	1 (2)
Marine life/Jellyfish	0 (0)	1 (2)
Asthma trigger	0 (0)	4 (9)
Unknown	2 (8)	9 (20)
Total	24 (99)	46 (100)

NOLS, National Outdoor Leadership School; OB, Outward Bound.

INCREASE IN THE FIELD REPORTING OF ANAPHYLAXIS BY NOLS AND OB

Since 1984, NOLS has noted a 12-fold increase in its field reporting of anaphylaxis (Table 4). Similarly, OB also has noted an increase, although exact comparative numbers are not currently available. To explain this, NOLS and OB leaders have proposed 3 theories, all of which may be contributing.

First is an increased incidence of anaphylaxis in this population, which is supported by a corresponding 3-fold increase at NOLS of nonanaphylactic, acute allergic reactions (Table 4). In particular, the number of NOLS and OB students reporting a history of food allergy has increased in recent years (Table 5 illustrates NOLS data). It is plausible that as the number of students with food allergy has increased, so has the number at risk for anaphylaxis. The rise in food allergy at NOLS and OB parallels an increased prevalence of food allergy in the general US population. Currently, an estimated 11% of adults and 8% of children in the United States have a food allergy, with an increased prevalence in children by 50% over a 15-y period.[15]

Table 4. Increase in incidence of anaphylaxis and allergic reactions at NOLS

NOLS injury and illness database	*2005–2019*	*1984–2004*
Total field time (p-d)	2,431,591	2,446,159
Anaphylaxis cases	24	2
Incidence per p-d	1/101,316	1/1,223,080
Allergic reactions	467	149
Incidence per p-d	1/5207	1/16,417

NOLS, National Outdoor Leadership School; P-D, person-days.

Table 5. Increase in food allergies at NOLS

Year	*NOLS students with food allergy, n (%)*	*Nut allergy (% of total allergies)*
2015	37 (0.7)	65
2016	65 (1.2)	71
2017	116 (2.2)	71
2018	192 (3.6)	58
2019	140 (2.7)	54

NOLS, National Outdoor Leadership School.

Second, the increase in anaphylaxis may be due to increased recognition by instructors, perhaps as the result of specialized training, as well as the indirect effect of heightened public awareness of food allergies and anaphylaxis. On the other hand, internal quality-assurance reviews of field reports by each school have suggested that instructors appropriately identified and generally did not overdiagnose anaphylaxis.

Third, veteran OB leaders have observed that more parents and physicians are allowing students with a food allergy or anaphylaxis history to participate in remote wilderness-based courses and expeditions compared to the past. Caretakers may have found reassurance in the increased public awareness of food allergy and anaphylaxis, as well as emergency care plans for the immediate availability of prehospital epinephrine, whether administered by trained, onsite first responders or self-injected by students. As more individuals at risk for food allergy and anaphylaxis feel comfortable enrolling in NOLS and OB courses, a slight selection bias may be contributing to the increased field incidence of these conditions.

Pathophysiology

The vast majority of allergic and anaphylactic responses are IgE-dependent, immediate (Type I) hypersensitivity reactions (Table 1). An allergen exposure in a susceptible host stimulates B lymphocytes to produce specific IgE antibodies that bind to receptors on mast cells and, to a lesser extent, basophils. If the same allergen is later reintroduced into the body, it binds to the previously formed IgE-receptor complex, triggering a release of multiple preformed mediators such as histamine, tryptase, and proteases. Neutrophils, eosinophils, monocytes, and platelets can also be activated in the process.[16]

Limited, focal release of preformed mediators such as histamine leads to the relatively minor expressions of allergy such as allergic conjunctivitis and rhinitis, as well as urticaria.

In acute asthma, allergens that enter the airways induce T-helper cells to produce cytokines, stimulate B cells to release IgE, and cause mast cells to release leukotrienes and histamine—all of which trigger bronchoconstriction and initiate airway inflammation. Ongoing, subacute release of inflammatory mediators occurs in

chronic asthma, with associated mucus hypersecretion as well as airway edema and remodeling.[17]

Large-scale release of preformed mediators, together with the synthesis and release of inflammatory mediators such as prostaglandins, cytokines, and leukotrienes, lead to the clinical signs and symptoms of anaphylaxis. Histamine induces vasodilation and increases capillary vascular permeability, causing fluid to extravasate from the intravascular to the extravascular space. Tryptase stimulates additional mast cell degranulation and downstream activation of the complement and coagulation pathways, as well as the kallikrein-kinin system, triggering an amplified, overwhelming inflammatory cascade.[18]

The role of IgE-independent pathways in anaphylaxis is less clearly understood, but it may involve the release of preformed mediators by mast cells and basophils through direct activation of the complement system. Reactions involving IgM and IgG antibodies also have been demonstrated in animal models.[16]

Histamine and other inflammatory mediators can be released directly from mast cells without an antigen-antibody interaction or prior exposure to an antigen. Such direct release has been called an anaphylactoid reaction, although some experts now discourage use of this term because the clinical symptoms and severity may be identical to IgE-mediated anaphylaxis.[19] Non-IgE anaphylaxis or anaphylactoid reactions are most commonly associated with certain pharmaceuticals, such as nonsteroidal anti-inflammatory drugs and radiocontrast agents.[20]

Histamine also contributes to allergic angioedema, a form of submucosal tissue swelling typically affecting the face, oropharynx, and larynx and sometimes presenting as extremity or abdominal wall edema. Angioedema also can develop in nonallergic conditions, including hereditary angioedema, acquired C1-inhibitor deficiency, and drug-related angioedema, such as that caused by angiotensin converting enzyme (ACE) inhibitors. In these conditions, the cause is excess production or decreased clearance of bradykinin, a vasoactive peptide.[19,21]

Certain cofactors augment allergy symptoms in susceptible individuals who ingest a food-based allergen. These cofactors include exercise, alcohol, and nonsteroidal anti-inflammatory drugs, all of which are thought to enhance intestinal permeability and allergen absorption. In addition, exercise has been associated with 2 subtypes of anaphylaxis: exercise-induced anaphylaxis (EIA) and food-dependent, exercise-induced anaphylaxis (FDEIA). A hypothesis for the pathophysiology of EIA is that physical activity, in the absence of an environmental allergen, increases plasma osmolarity, which in turn causes mast cells to degranulate, releasing histamine and other cell mediators. FDEIA is an IgE-mediated food allergy that occurs when exercise is performed shortly after ingestion, activating the histamine-based cellular cascade.[22-24]

A food allergy to red meat such as beef, pork, or lamb may develop in individuals who form an IgE antibody response to the mammalian glycoprotein component galactose-α-1,3-galactose after a tick bite. This is associated with the Lone Star tick (*Amblyomma americanum*) in the United States and other species worldwide. Symptoms of α-gal allergy characteristically develop 3 to 5 h after ingestion as the meat is digested and absorbed through the gastrointestinal tract and range from mild to severe, including anaphylaxis.[25]

Clinical Manifestations

Anaphylaxis is a systemic reaction, usually of rapid onset, that progresses to affect multiple organ systems. Medical organizations have published clinical algorithms to aid in diagnosis, emphasizing early recognition. In 2020, based on evidence review and expert consensus, the World Allergy Organization proposed that anaphylaxis be diagnosed when 1 of 2 criteria is met, in the context of known or highly probable allergen exposure[26]:

1. Cutaneous or mucosal signs that occur suddenly, progress within minutes to hours, and are accompanied by respiratory compromise, hypotension, or persistent gastrointestinal symptoms.
2. Acute onset of hypotension or respiratory compromise, including severe bronchospasm or laryngeal involvement, even in the absence of skin involvement.

As exceptions, and described in the previous section, the diagnosis of EIA does not require exposure to an environmental allergen, and symptoms of α-gal allergy typically develop 3 to 5 h after ingestion.

Cutaneous or mucosal involvement is the most frequent sign of anaphylaxis and includes urticaria, flushing, pruritus, oropharyngeal swelling, or angioedema.[27] Urticaria (hives) is a blanching, erythematous rash with transient wheals that is typically pruritic. However, skin involvement is not required for the diagnosis; up to 10 to 20% of anaphylaxis cases have absent or unrecognized skin and mucosal findings.[28]

Respiratory involvement can present as sneezing, nasal congestion, cough, hoarse voice, angioedema, bronchospasm, wheezing, stridor, dyspnea, and hypoxemia. The most common gastrointestinal symptoms are nausea, vomiting, abdominal pain or cramping, and diarrhea. Cardiovascular signs and symptoms include tachycardia, dysrhythmias, lightheadedness, syncope, chest pain, and hypotension. Neurologic symptoms such

as lightheadedness, impending sense of doom (*angor amini*), and confusion may be present. Patients may also report a metallic taste or appear extremely anxious.[26]

Anaphylactic fatalities result from respiratory or cardiovascular effects. Angioedema and bronchospasm lead to airway obstruction and ventilatory failure. In the cardiovascular system, a sudden and massive increase in capillary permeability causes hypotension and shock. Up to 35% of intravascular fluid may extravasate within 10 min of allergen exposure, leading to decreased venous return and cardiovascular collapse.[27]

The majority of anaphylactic reactions resolve with appropriate treatment. Certain patient characteristics, however, increase the risk of severe or fatal anaphylaxis.[29] Infants and young children are less able to communicate symptoms and may have unrecognized abnormal vital signs. Their narrower airways are more susceptible to obstruction by swelling and increased secretions. Elderly patients are at increased risk owing to underlying comorbidities or medication use. Those with cardiovascular or pulmonary disease, for example, may not tolerate the increased stress on these organ systems. In addition, patients with heart disease have more mast cells in their coronary arteries, which may lead to increased coronary vasoconstriction during anaphylaxis.[30] For patients on antihypertensive medications, beta blockers may blunt the therapeutic response to epinephrine and ACE inhibitors may interfere with the degradation of inflammatory mediators.[19,26]

The biphasic reaction is an anaphylaxis variant, reported to occur with a wide range of incidence from <1 to 15%, although larger cohorts have reported 4 to 6%.[31-35] After treatment and apparent resolution of anaphylaxis, symptoms can recur within 1 to 78 h without antigen re-exposure. A multinational registry found that the second phase of symptoms occurred within 12 h in 60% of patients; from 12 to 24 h in 24% of patients; and >24 h in 16% of patients.[34] Risk factors associated with the development of a biphasic reaction have included history of anaphylaxis, elderly age, cardiovascular disease, regular use of beta-blockers, onset of symptoms >30 min from allergen exposure, nut allergy, unknown allergen, medication allergy in children, severe initial reaction with multiorgan involvement, delay in epinephrine administration, and requirement of multiple epinephrine doses.[13,34]

Exercise-induced anaphylaxis occurs less commonly than other causes of anaphylaxis, but it is relevant to the physical activities of outdoor recreation. Activity of any intensity may induce symptoms in the absence of an environmental allergen; however, jogging and aerobic exercise appear to be the most common causes.[23] Symptoms occur shortly after the onset of exercise and include rhinorrhea, pruritus, flushing or urticaria, abdominal cramping with nausea, vomiting or diarrhea, cough, wheezing, and shortness of breath. Symptoms of EIA (and FDEIA) typically resolve with rest and prompt cessation of activity. Patients who have persistent or worsening signs and symptoms, including respiratory compromise or hypotension, should be treated with the standard therapies discussed later.[23,24,26] Mortality attributed to EIA has been reported only in a handful of cases, although it is speculated that EIA may be underreported as a cause of sudden death with exercise.[24]

Anaphylaxis is a syndrome with a variable presentation, and therefore the differential diagnosis is broad. It includes conditions affecting the respiratory system, such as asthma, acute pulmonary edema, foreign body aspiration, pulmonary embolism, and vocal cord dysfunction. Acute coronary syndrome, cardiogenic shock, sepsis, hypoglycemia, hyperventilation, panic attack, and vasovagal reactions should also be considered. Hereditary angioedema, ACE-inhibitor induced angioedema, and diffuse urticaria may share similar skin findings with anaphylaxis. Less common mimics involve excess histamine release such as scombroid, mastocytosis, and drug-related red man syndrome. Pronounced flushing of the skin also may be caused by rare syndromes such as pheochromocytoma and carcinoid.[20,26]

Treatment

GENERAL CONSIDERATIONS AND DECONTAMINATION

As with any potentially serious illness or injury in the field, care begins with an assessment of scene safety followed by a primary evaluation of the patient with interventions as needed to support airway, breathing, and circulation. Epinephrine should be given as soon as possible once anaphylaxis has been identified. Additional interventions depend on first responder training as well as local resources and equipment.

Removal of the inciting allergen is appropriate in certain circumstances. For example, immediately removing an insect stinger from the skin may prevent additional injection of venom (avoiding pressure, if possible, on the venom sac). Exposure to aerosolized, food-based allergens should be stopped by discontinuing on-site cooking of the associated food (eg, steaming of shellfish) and, if feasible, distancing of the patient. Inducing vomiting for food-based allergens, however, has not been proven effective and may delay treatment with epinephrine.[35]

EPINEPHRINE (ADRENALINE)

Ideally, the treatment of anaphylaxis should stabilize mast and other immune cells, reverse vascular dilation and increased permeability, and relieve airway constriction. Epinephrine accomplishes all these tasks through agonist effects at α_1 receptors in the vascular system and β_2 receptors in the lungs and mast cells.[36] Its worldwide acceptance as the primary anaphylaxis treatment is based on years of clinical experience and theoretical mechanisms of action rather than controlled human trials.[13,26,37,38] Unfortunately, epinephrine is still viewed by some as a temporizing rather than definitive treatment and is withheld while other medications are given first.[39-42] Delay in epinephrine administration has been repeatedly associated with fatal anaphylaxis.[43]

Recommendations: Epinephrine is the essential, primary treatment that should be given once anaphylaxis has been diagnosed (1A). If possible, separating the patient from the inciting allergen is prudent, but vomiting should not be induced to eliminate a food-based allergen (1C).

Routes of Administration

Intramuscular (IM) injection of epinephrine is used in the prehospital and hospital settings to treat anaphylaxis immediately before intravenous (IV) access is established. Based on experimental IM studies, injection into the anterior lateral thigh delivers the highest serum levels of epinephrine in the shortest time and is strongly preferred.[44] If the anterior lateral thigh is inaccessible (eg, because of body position, injury, thick clothing, or protective gear), then the deltoid is acceptable. Subcutaneous (SQ) deltoid injection has been proposed as an alternative to IM injection, although current evidence favors muscle tissue for its greater vascularity, which enhances medication absorption.[45]

Although they are widely available, over-the-counter, metered-dose inhalers of epinephrine have not been found to be a practical or effective treatment for anaphylaxis. In a pharmacological study on children, achieving weight-based doses of epinephrine required a high number of puffs (11±2 [mean±SD]) and was hampered by the adverse effects of bad taste, cough, and dizziness. As a result, most of the children were not able to achieve therapeutic plasma levels.[46]

The US Food and Drug Administration (FDA) has given expedited review to intranasal formulations of epinephrine based on preliminary trials that demonstrated effective absorption equivalent to IM injection.[47] In the future, sublingual administration also may become an option.[48]

Epinephrine may be given IV as a continuous infusion or intermittent boluses when anaphylactic shock is refractory to repeated IM injections (see "Dosage" and "Refractory Anaphylaxis" sections).

Recommendations: Given its effectiveness and rapid administration, IM epinephrine is the first-line treatment for anaphylaxis. The preferred injection site is the anterior lateral thigh, followed by the deltoid (1B). Over-the-counter, metered-dose inhalers of epinephrine have not been found to be a practical or effective treatment for anaphylaxis (1B).

Epinephrine Injection Devices

Various devices are available to inject epinephrine (Table 6), each with advantages and disadvantages. NOLS outfits its trips with a preassembled kit containing an insulin-type syringe with needle and a 1-mL ampule of epinephrine. OB primarily uses epinephrine autoinjectors (EAIs) in addition to prefilled syringes and the syringe-plus-ampule or vial method. Regardless of the device used, with regular training, instructors from both schools have had an excellent safety record on correctly administering epinephrine.

Autoinjectors; Prefilled Syringes

Fixed-dose EAIs have become widely available in hospitals, clinics, emergency medical services, certain public venues, and the field.[49] They are effective, convenient to carry, and eliminate the need to draw medication into a syringe, which may decrease the risk of incorrect dosage.[50] Many can be discharged through clothing, although thicker clothing would likely decrease the depth of delivery. Currently, EAIs are available in 0.1, 0.15, 0.3, and 0.5 mg doses, outfitted with a variety of needle lengths, and manufactured in different styles, depending on the country of origin (Table 6). Their disadvantages include cost, which is compounded by a limited shelf life. The manufacturer's wholesale list price of EpiPen, a widely used brand in the United States, is $609 for a package containing 2 autoinjectors (0.3 or 0.15 mg doses). The list price for a generic dual-pack is $300.[51] (Medical insurance and other factors, however, may greatly decrease the final consumer cost.) In addition, without proper training that is periodically reinforced, both prescribers and patients may use the devices incorrectly[52,53] or cause unintended medication discharge and needle injury.[54-57]

Epinephrine is also available in sterile, prefilled syringes (0.3 and 0.15 mg doses) with a manual plunger and preattached needle. The list price is $250 for a package containing 2 syringes (Symjepi).[51]

To help prevent accidental needle sticks, manufacturers have developed safety features, such as needles that automatically retract after medication discharge (eg, Auvi-Q)

Table 6. Sample epinephrine delivery devices

Name	*Mechanism; medicine container*	*Dose (mg)*	*Needle length (mm)*	*Safety features post-injection*	*Manufacturer*
Autoinjectors					
EpiPen (G)	Spring; cartridge	0.15 0.3	12.7 15.2	Automatic needle guard	Meridian Medical Technologies, United States
Auvi-Q (United States), Allerject (Canada)	Compressed gas; cartridge	0.1 0.15 0.3	7.4 12.7 15.7	Automatic retractable needle	Kaléo, United States
Adrenaclick (G)	Spring; syringe	0.15 0.3	12.7 12.7	Carrier case for syringe + exposed needle	Meridian Medical Technologies, United States
Emerade	Spring; syringe	0.15 0.3 0.5	16 23 23	Automatic needle guard	Medeca Pharma, Sweden
Jext	Spring; cartridge	0.15 0.3	13 15	Automatic needle guard	ALK-Abelló, Denmark
Anapen	Spring; syringe	0.15 0.3 0.5	12.7 12.7 12.7	Manual sliding needle guard	Bioprojet, United Kingdom
Manual injectors					
Symjepi	Plunger; fixed-dose syringe	0.15 0.3	15.9 15.9	Manual sliding needle guard	Adamis Pharmaceuticals, United States
Epi Kit	Plunger on 1 mL syringe; 1 mg epinephrine in 1 mL vial	Variable, up to 1 mg	25.4	Manual sliding needle guard	Curaplex, United States

G, generic versions available.
In a syringe-based autoinjector, the medicine container is connected directly to the base of the needle. In a cartridge-based autoinjector, the medicine container is propelled during injection to connect with the base of the needle. Product specifications subject to change.

and guards that slide over the needle either automatically (eg, EpiPen) or manually (eg, Symjepi).[51]

Vials or Ampules

Epinephrine manually drawn into a syringe at the time of injection is a viable and less expensive alternative to EAIs. The list price for generic epinephrine (1 mg·mL^{-1} concentration) is approximately $3 for a 1 mL vial and $10 to $18 for a 30 mL multidose vial.[58] For cost savings, the vial or ampule plus syringe method is becoming more commonly used among basic life support practitioners in emergency medical services.[59,60] A possible disadvantage to this method is operator error or time delay while calculating the dose and drawing epinephrine into a syringe, especially during an anaphylaxis emergency.[50] The risk of operator error can be lessened by prefilling syringes with epinephrine before a trip or field deployment. With proper technique and storage, the risk of medication inactivation and contamination is reported to be minimal for up to 90 d.[61] In addition, stocking field medical kits with ampules or vials containing 1 mL of epinephrine rather than 30 mL multidose vials limits the total amount of medication that may be given in overdose.

Recommendations: An organization's choice of an epinephrine delivery device depends on considerations of cost, operator training, and safety for both patient and first responder (1C). Autoinjectors may be less prone to dosage error, but they require periodic training to use correctly and avoid injury. With regular training, the ampule or vial and syringe method has been safely used for decades in field conditions by NOLS and OB. A third option involves prefilled or fixed-dose syringes with epinephrine.

Dosage

In the United States, epinephrine is available in 2 concentrations, 1 mg·mL^{-1} and 0.1 mg·mL^{-1} (formerly 1/1000 and 1/10,000 concentrations, respectively).[62] The standard initial adult dose for anaphylaxis is 0.3 to 0.5 mg IM in the United States and 0.5 mg in Europe (1 mg·mL^{-1} concentration) (Table 7). Clinical experience has confirmed the

Table 7. Summary of pharmacological treatments for anaphylaxis, with examples in each drug class

Medication	*Route*	*Dosage*	*Indication*	*Recommendation*
Epinephrine (Adrenaline)	IM: anterior lateral thigh > deltoid	0.01 mg·kg^{-1}, up to 0.3-0.5 mg per dose Q 5-15 min PRN	Initial treatment	1A (Epinephrine) 1B (choice of anterior lateral thigh)
	IV	Infusion: 0.1 microgram·kg^{-1}·min^{-1}, titrate to clinical effect Bolus: 50–100 microgram·min^{-1}	Refractory cases	1C
H_1 antihistamines	Diphenhydramine PO, IM, IV	25–50 mg Q 4–6 h Peds: 1 mg·kg^{-1} per dose	Secondary treatment; cutaneous manifestations (rash, edema, pruritis)	1C
	Certirizine PO, IV	10 mg QD Peds: <6 y: 2.5 mg; 6–11 y: 5–10 mg QD		
H_2 antihistamines	Famotidine PO, IV	20 mg BID Peds >3 mo: 0.25 mg·kg^{-1} dose BID	Possible synergistic effect with H_1 antihistamines	2B
β2 agonist	Albuterol		Secondary treatment; bronchospasm	1C
	Metered-dose inhaler, 90 microgram·actuation^{-1}	2 inhalations; frequency varies with severity		
	Nebulizer solution	≥12 y: 2.5–5 mg 5–12 y: 1.25–2.5 mg; 1–5 y: 1.25 mg		
Corticosteroids	Prednisone PO	1-2 mg·kg^{-1}, up to 50–60 mg QD Peds: ÷ Q12–24 h	Secondary treatment; bronchospasm; asthmatic patient; possible prevention of biphasic reaction	1C
	Methylprednisolone PO, IM, IV	1–2 mg·kg^{-1}, up to 40-60 PO/IM QD, 80-125 mg IV QD Peds: ÷ Q12–24 h		
	Dexamethasone PO, IM, IV	6–9 mg QD Peds: 0.3 mg·kg^{-1} QD		
Glucagon	IV	Initial dose: 1–5 mg Peds: 0.02–0.03 mg·kg^{-1}, up to 1 mg per dose Subsequent infusion at 5–15 microgram·min^{-1}, titrate to clinical effect	Refractory cases in patients on β blockers	2C
Desensitization therapy	SQ, PO	Protocol of sequentially increasing antigen dose.	Prior anaphylaxis to *Hymenoptera* venom or peanuts	1B

BID, twice per day; IM, intramuscular; IV, intravenous; Peds, pediatric dose; PO, orally; PRN, as needed; Q, every; QD, per day; SQ, subcutaneous.
Unless otherwise specified, the maximum pediatric dose is the adult dose. Duration of treatment with antihistamines and corticosteroids is generally 3 to 5 d (1–2 d with dexamethasone). Dosing reference: Kleinman et al.[121]

Table 8. Recommended minimum needle length (22–25 gauge) for IM injection of patients according to body weight

Weight kg (lb)	*Minimum needle length for IM injection (mm)*
Female or male <60 (130)	16
Female or male 60–70 (130–152)	25
Female 70–90 (152–200)	25
Male 70–118 (152–260)	25
Female >90 (200)	38
Male >118 (260)	38

Adapted from: National Center for Immunization and Respiratory Diseases.[122]

safety and efficacy of this dose range.[13] The pediatric IM dose is 0.01 mg·kg^{-1} of body mass until the adult dose is reached.[63] For pediatric EAIs, 0.15 mg is an accepted dose for patients weighing 7.5 to 25 kg.[64] For both children and adults, there is no cumulative maximum dose. Repeat IM doses can be given as needed every 5 to 15 min if there has been no improvement.[13]

Although the majority of cases resolve after 1 dose, a reported 8 to 12% of ED adult and pediatric anaphylaxis patients have required 2 or more epinephrine doses during initial treatment (not for a biphasic reaction).[65,66] In urban pediatric EDs, 6 to 19% of anaphylaxis patients have required 2 or more initial doses.[63]

For adult IV administration, generally in hospital settings for refractory anaphylaxis (see the following), 1 mg of epinephrine may be added to 1 L of normal saline, producing a concentration of 1 microgram·mL^{-1} and started at a drip of 0.1 microgram·kg^{-1}·min^{-1}, with careful hemodynamic monitoring. Alternatively, 1 mg of epinephrine may be added to 10 mL of normal saline, producing a concentration of 0.1 mg·mL^{-1}, and given slowly via IV bolus starting at 50 to 100 microgram·min^{-1} (0.5–1 mL·min^{-1}). Subsequent rates and doses are titrated to effect. In infants and small children, the concentrations of IV epinephrine solutions are weight-based and adjusted so as to not infuse an excess amount of fluid.[67]

Recommendations: Standard IM doses of epinephrine may be repeated every 5 to 15 min for an inadequate response to initial anaphylaxis treatment or hours later for a biphasic reaction (1B).

Needle Length

In the United States, the EpiPen needle length is 16 mm for adults and 13 mm for pediatrics. In Europe, needle length in adult EAIs may reach 23 mm (eg, Emerade). Studies using ultrasound to measure adipose thickness in the adult lateral thigh have questioned whether the 16 mm needle is long enough to deliver epinephrine into the muscle layer of many female patients (who may have a thicker adipose layer at the thigh compared to males) as well as patients with obesity.[45,68,69] These concerns, however, are based on a surrogate marker of clinical effectiveness (thickness of adipose tissue) rather than on actual clinical outcomes or pharmacological studies. In actual use, EAIs compress the SQ tissue and also deliver medication with propulsive force, both of which contribute to the depth and effectiveness of injection.[70] Therefore, the available pharmacological studies suggest that EAIs administered into the anterior lateral thigh deliver an effective medication dose in the majority of patients.[47] Although certain adult patients with obesity or thick adipose tissue in the thigh may benefit from a longer needle (23–25 mm), current evidence does not exactly characterize such patients. On the other hand, a 16 mm or longer needle may be too long for small children and risk penetrating bone.[71]

For injections using a manual syringe with plunger, published guidelines for needle length needed to reach muscle tissue are based on vaccine administration in outpatient settings. The recommended needle length varies from 16 to 38 mm depending on body weight and sex (Table 8). A 22- to 25-gauge needle is acceptable for all needle lengths. A 16 mm needle may deliver an effective IM dose of epinephrine in fit adolescents and young adults, but 25 mm should be considered in large adults or obese patients. Compressing the SQ tissue and pushing the plunger with propulsive force may render the 16 mm needle effective in large adults or obese patients, but this has not been definitely studied in anaphylaxis.

Recommendations: In general, an EAI with a 16 mm needle delivers an effective dose of medication in adult patients (1B), although obese patients may benefit from a longer needle (2B). For manual syringes, a 16 mm needle delivers an effective IM dose in fit adolescents and young adults, though a 25 mm or longer needle should be considered in large adults or obese patients (1C).

Storage

Environment

Manufacturers recommend keeping epinephrine at temperatures between 20 and 25°C (Meridian Medical Technologies, Columbia, MD). These conditions cannot always be met in the field. Limited research, however, suggests that temperatures exceeding this range will have little impact on potency over short durations. As an extreme example, EAIs experimentally stored at 70°C for 5 d delivered 97±4% of labeled dose compared to room temperature controls.[72] Conversely, freezing neither inactivates epinephrine[73,74] nor damages an EAI (EpiPen) for use after thawing.[75]

Insulated carrying cases designed to protect medications from high temperatures are available (Frio, Walnut Creek, CA) but require independent field testing and additional corroboration of efficacy.

Expiration Dates

For disaster or austere conditions, multiple reports suggest that acceptable epinephrine potency is retained as long as 24 mo beyond the expiration date.[76-79] The US Army Health Command in Europe has extended the expiration dates of EAIs by 6 mo due to manufacturing shortages.[80] In the United States, the FDA has extended the expiration dates of EAIs by 4 mo and prepackaged epinephrine syringes by 9 mo due to similar shortages.[81]

Recommendations: During manufacturing shortages, US government agencies have approved the use of epinephrine for up to 9 mo past the expiration date. This extension provides a potential rationale, but not regulatory approval, for the use of recently expired epinephrine in shortages associated with austere or disaster conditions (2C). In addition, uncontaminated epinephrine may retain its potency despite short excursions to high or low temperatures as may occur in the field (2B).

Complications

National and international guidelines note the lack of absolute contraindications for epinephrine in anaphylaxis.[13,26,63] Serious adverse events with therapeutic dosing, including arrhythmias, stroke, and myocardial infarction, are rare and generally have affected the elderly or individuals with a history of coronary artery or cerebrovascular disease.[82,83] A few cases of myocardial infarction in young, healthy patients have been reported, presumably due to coronary artery vasospasm.[84] On the other hand, cardiac complications attributed to epinephrine may result instead from the effects of anaphylaxis itself.[85,86]

Cases of cardiac dysrhythmias and myocardial infarction have been associated with IV epinephrine and attributed to accidental overdose, rapid administration, or insufficiently diluted medication.[87-90]

Complications also may result from mechanical operation of the EAI. Lacerations and embedded needles have occurred in children who have forcefully withdrawn from the autoinjector needle. To decrease the risk of such injury, caregivers should receive education on firmly immobilizing the limb during injection. In addition, the EAI should be pressed against the skin only with the force required to deploy the spring or cartridge-loaded needle (1-3.5 kg [2-8 lb] of pressure) and only for the time required to deliver the medication (about 3 s).[91] Inadvertent digital injection of epinephrine has occurred from handling the needle end of the autoinjector after safety lid removal. The resulting local vasoconstriction and ischemia have been treated effectively with warm compresses, topical nitroglycerin, or, in severe cases, phentolamine injected into the affected finger. Digital necrosis or permanent injury from inadvertent epinephrine injection has not been reported.[55]

Recommendations: Absolute contraindications to epinephrine in anaphylaxis are lacking; however, IV administration carries additional risks and generally requires advanced medical expertise and monitoring (1C). Proper limb immobilization and injection technique may decrease the risk of EAI-associated injuries, especially in children. For inadvertent digital injection of epinephrine, treatment options include warm compresses, topical nitroglycerin, and, in severe cases, local phentolamine injection (1C).

Legal Considerations

Historically, training nonmedical first responders to inject epinephrine has involved controversy and uncertainty, especially in light of different state regulations. Controversies have included whether such training promotes practicing medicine without a license, as well as the liability implications of a provider writing, and a pharmacist dispensing, a prescription to an organization rather than an individual. To some extent, these concerns have been addressed by the Federal School Access to Emergency Epinephrine Act of 2013, which supported trained lay providers administering epinephrine for anaphylaxis in elementary through secondary schools. Expanding upon this act, individual states have passed legislation (commonly known as "stock epinephrine entity laws") to include other locations where anaphylaxis may occur, including daycare centers, recreational camps, theme parks, and sporting events.[92] In general, state laws on stocking emergency epinephrine in public venues have favored EAIs or FDA-approved prefilled syringes to help reduce the risk of a dosing error or needle injury that may occur when manually drawing medication from a vial into a syringe. An exception is illustrated by the state of Alaska, which has approved the use of vials and syringes by lay providers who have obtained state-approved certification.[93]

Regardless of the epinephrine delivery device chosen, the US Occupational Safety and Health Administration has issued guidelines requiring the use of engineering controls and standard procedures to protect worker safety by reducing the risk of needle injury and transmission of bloodborne pathogens.[94] Outdoor schools and organizations that carry epinephrine into the field therefore must consult and follow both the relevant federal guidelines as well as state-specific laws.

SUPPLEMENTARY TREATMENTS

National and international guidelines list antihistamines, corticosteroids, and inhaled β agonists as acceptable secondary treatments for anaphylaxis that should not substitute or delay epinephrine administration.[13,26,63,95] Table 7 lists representative medications, administration routes, and dosages.

Antihistamines

H_1 antihistamines such as diphenhydramine bind to and block H_1 histamine receptors in mast cells, smooth muscle, and endothelium. They improve the cutaneous manifestations and pruritus of allergic reactions.[96] Early administration of antihistamines with epinephrine has been associated with blunting the overall severity of anaphylaxis and reducing the total number of epinephrine doses needed.[66] On the other hand, antihistamines do not reverse vascular dilation and airway constriction or edema, nor do they inhibit the release of other inflammatory mediators.[97] Their role in preventing a biphasic reaction is possible but uncertain.[13,66]

Potential side effects include sedation and anticholinergic reactions, such as dry mouth, tachycardia, and urinary retention. Second generation antihistamines such as loratadine are less likely to cause sedation. In hospital and certain prehospital settings, IV administration of H_1 antihistamines may cause vascular dilation if injected too rapidly.[98]

H_2 antihistamines such as famotidine or ranitidine have been used in combination with H_1 antihistamines to treat allergic reactions with improved outcomes compared to H_1 use alone.[99] Evidence to support an additive therapeutic effect specifically in anaphylaxis, however, is inconclusive.[100]

Recommendations: Antihistamines may help blunt the overall severity of anaphylaxis when given early with epinephrine (1C). Non-sedating antihistamines may be preferred in the field to help keep the patient alert and potentially able to walk (2B). The addition of an H2 antihistamine to an H1 antihistamine has improved outcomes in allergic reactions and may be beneficial in anaphylaxis, but the exact incremental benefit is unknown (2B).

Inhaled β Agonists

This class of medications is a mainstay for the treatment of asthma exacerbations but an adjunct to treating the lower airway constriction and wheezing that may occur in anaphylaxis.[63,95,101] Possible side effects include tachycardia and a temporary decrease in serum potassium (which shifts into cells) and increase in serum glucose.[102]

Recommendations: Inhaled β-agonists may be administered as adjunctive treatment for wheezing, especially in a person with a history of asthma (1C).

Corticosteroids

The anti-inflammatory effects of corticosteroids stabilize mast cells and blunt the cascade of inflammatory mediators. Corticosteroids have been used in anaphylaxis based on their efficacy in asthma[103] as well as theoretical mechanisms of action. The onset of their anti-inflammatory effect may not occur for several hours, regardless of oral or parenteral administration.[104] Studies have suggested that corticosteroids diminish the possibility of a biphasic reaction, although this finding has not been consistently replicated.[31,105,106] In 1 review, prehospital administration of corticosteroids for anaphylaxis was associated with increased hospital or ICU admission; however, confounding variables and selection bias may have contributed.[66]

The optimal route and dose for steroids in anaphylaxis have not been established. Common practice in the ED is 50 to 60 mg of oral prednisone for stable adult patients who are not vomiting, or 80 to 125 mg of methylprednisolone IV for severely ill patients. The pediatric dose is 1 to 2 $mg{\cdot}kg^{-1}$ orally or IV until the adult dose is reached. The total duration of steroid treatment (commonly with antihistamines) is generally 3 to 5 d (1–2 d for dexamethasone, given its long half-life) with the theoretical aims of reducing the risk of biphasic reaction and offsetting any lingering allergen effect, especially with gastrointestinal absorption that may continue past the day of ingestion.[35] Side effects are uncommon with short-term use and may include blood glucose elevation in diabetic patients, agitation in the elderly, exacerbation of peptic ulcer disease, and increased infection risk in immunosuppressed patients.[104]

Recommendations: Evidence of benefit for corticosteroids in anaphylaxis is inconsistent; however, pending conclusive evidence, continued empiric use is reasonable given the potential for benefit paired with a low side-effect profile (1C). In particular, corticosteroids should be given for anaphylaxis with a respiratory component in asthmatic patients (1C).

FIELD PROTOCOLS

Both the NOLS and OB field protocols (see online Appendix 1 and 2) stipulate that individuals who have been treated with epinephrine for anaphylaxis be evacuated from the field. The actual decision to evacuate, however, as well as the modality (eg, air or ground) and

timing of evacuation depend on multiple factors. These include environmental and safety considerations, such as the local terrain, weather, visibility, and distance to definitive care. Medical factors should also be considered, including the severity of the reaction; patient comorbidities and risk factors for a biphasic reaction; medical training of field providers; availability of medical control; and contents of the medical kit, including additional doses of epinephrine. Preparations for litter transportation should be made for patients with ongoing cardiovascular or respiratory symptoms. Patients should be transported in a position of comfort—those with hypotension may benefit from recumbency, whereas those with breathlessness may not tolerate a supine position.[2]

As a potentially life-saving measure in the field, off-label techniques have been described for disassembling an EAI after IM administration to obtain an additional epinephrine dose when no other source is available.[107,108] This procedure involves a significant risk of injury from the spring or cartridge-loaded needle, which remains under tension even after medication discharge, and requires practice under controlled conditions. Knowledge of this procedure, however, should never replace proper planning for an adequate, reliable supply of medication in the field.

Recommendations: In the field, medical evacuation is generally recommended after treatment of anaphylaxis (2C). The actual decision to evacuate, however, may be influenced by case-specific factors, such as geography, weather, field capabilities, and patient characteristics and response to treatment. These factors also may influence the timing and method of evacuation. For austere or disaster conditions, off-label techniques for disassembling an EAI after IM administration to obtain another epinephrine dose are available and should be considered an inherently risky but potentially life saving measure when no other source is available (2C).

REFRACTORY ANAPHYLAXIS

Refractory anaphylaxis has been defined as requiring 3 or more epinephrine doses during initial treatment and occurs in approximately 1% of cases.[65,66] In contrast, persistent anaphylaxis has been defined as lasting 4 or more hours despite initial treatment.[109] Epinephrine may be given every 5 to 15 min IM to treat refractory anaphylaxis, along with the secondary treatments of antihistamines and corticosteroids, as well as inhaled β agonists for patients with bronchospasm. Where available, supplemental oxygen should be given for hypoxia and crystalloid solutions for volume replacement and hypotension. Persistent shock or hypoxia requires critical-care measures including positive-pressure ventilation, intubation, and advanced cardiovascular monitoring, generally in hospital settings. In these instances, epinephrine may be given IV as a continuous infusion or with slow boluses. Other vasopressors should also be considered.[20,26,35,95,110] For patients on β blockers, a few case reports have suggested that glucagon may be beneficial in refractory anaphylaxis.[111,112]

Recommendations: Epinephrine may be given every 5 to 15 min IM to treat refractory anaphylaxis, along with the secondary treatments of antihistamines and corticosteroids, as well as inhaled β agonists for patients with bronchospasm (1C). For hypotension after epinephrine administration, IV crystalloids may be given with additional doses of IM epinephrine (1C). For persistent hypotension, IV epinephrine or an alternative vasopressor may be considered, in addition to standard critical-care measures (1C). For patients on long-term β blocker medication with refractory hypotension, glucagon is an option (2C).

POST-TREATMENT OBSERVATION PERIOD

The length of observation for patients after successful treatment of anaphylaxis is not clearly established, although there is consensus that it should vary with the severity of the initial reaction and risk factors for a biphasic reaction.[31] Ideally, observation should occur in a hospital or setting able to treat recurrence of symptoms with additional epinephrine, as well as the ability to address respiratory or hemodynamic decompensation. A recent meta-analysis concluded that 1 h of observation post-treatment achieved a 95% negative predictive value for detecting a biphasic reaction, and 6 and 8 h of observation achieved a 97% and 98% negative predictive value, respectively.[113]

Based on these findings, the American Academy of Allergy, Asthma, and Immunology has proposed that patients with nonsevere presentations, a prompt response to treatment, and low risk for a biphasic reaction be observed in a medical center for 1 h before discharge. Patients with a more severe presentation, significant comorbidities, or requiring multiple doses of epinephrine may benefit from a prolonged observation period up to 6 h or longer.[13] In addition, European guidelines have suggested a minimum monitoring period of 6 to 8 h for patients presenting with respiratory compromise and 12 to 24 h for patients with hypotension.[110] After observation and discharge, patients should receive an epinephrine prescription and be advised to follow up for allergy testing and consideration of immunotherapy.[114]

Recommendations: The length of observation after treatment of anaphylaxis depends on the severity of the initial reaction and risk factors for a biphasic reaction (1C). In patients with nonsevere reactions, a prompt

response to treatment, and low risk for a biphasic reaction, observation for 1 h may be sufficient (2B). Patients with more severe presentations, significant comorbidities, or requiring multiple doses of epinephrine may benefit from a minimum observation period of 6 h, or 12 to 24 h for presentations that involve cardiovascular compromise and hypotension (2B). Before discharge from a medical center, patients should receive an epinephrine prescription and be advised to follow up for allergy testing and consideration of immunotherapy (1C).

Prevention

The most important preventive strategy is avoidance of known allergens. This is not always possible, especially with *Hymenoptera* encounters in the outdoors. Desensitization protocols with SQ insect venom injections have been effective for at-risk individuals with a history of moderate to severe reactions.[115,116] For food-based allergies such as peanut, desensitization may be based on gradually increasing doses of prescription oral antigen. Of note, all desensitization protocols may reduce rather than eliminate allergic symptoms.[117]

Avoidance of food-based allergens is achieved by careful pre-trip medical screening of participants followed by appropriate selection of group provisions. All participants should be informed of the allergies present and advised of the importance of not exposing those at risk. Even with such measures, however, avoidance of allergens is challenging because many foods are produced in facilities that process a range of ingredients and may contain traces of potential allergens—an effect known as cross-contamination. The FDA does not require manufacturers to declare potential cross-contamination on food labels, although many do.[118]

Although pretreatment with antihistamines and steroids is widely used to prevent an allergic reaction to radiocontrast injection in the short term, a similar protocol has not been developed for the prevention of anaphylaxis to environmental or food-based allergens. In particular, the use of antihistamines to prevent allergic reactions or anaphylaxis in asymptomatic individuals prior to allergen exposure has shown inconsistent results.[119]

Recommendations: Desensitization protocols to *Hymenoptera* venom and peanuts are available and should be considered in patients with prior anaphylactic reactions to these antigens (1B).

Conclusions

Based on the injury and illness databases of NOLS and OB, anaphylaxis occurs in 0.01 to 0.03% of students in outdoor education courses, but its incidence appears to be increasing in recent decades. This increase is associated with a rise in the prevalence of food allergies among students in both schools, as well as children in the general US population. Since 2010, the Wilderness Medical Society has supported the concept that nonmedical professionals such as outdoor educators and guides whose work responsibilities include providing emergency medical care in the field also be trained to appropriately administer epinephrine for anaphylaxis. This position is strengthened by the finding that over 20% of the anaphylaxis cases in the NOLS and OB databases were first-time reactions in individuals without a known history of allergy or need to carry their own epinephrine. The top causes of anaphylaxis were food-based allergens or insect stings and bites.

The primary prehospital or field treatment for anaphylaxis is IM epinephrine. An organization's choice of an epinephrine delivery device depends on multiple factors, including cost, safety, provider training, as well as federal and state regulations. Antihistamines, corticosteroids, and inhaled β agonists are supplemental treatments for anaphylaxis that should not delay epinephrine administration. Formulations of intranasal and sublingual epinephrine are currently under development and may provide alternatives to needle-based devices in the future.

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Supplemental Material(s)

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BRIEF REPORT

Illness Incidence, Psychological Characteristics, and Sleep in Dogsled Drivers During the Iditarod Trail Sled Dog Race

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Introduction—Every March, dogsled drivers (mushers) compete in a 1569-km race across Alaska, involving physical exertion, mental exertion, and sleep deprivation for up to 2 wk. These factors may increase mushers' vulnerability to illness, making them a relevant study population for acute infection risk factors. Specifically, the influence of psychological factors on illness risk during prolonged physical exertion has rarely been investigated. The aim of this study was to examine the relationship between psychological characteristics, sleep deprivation, and illness incidence in Iditarod mushers.

Methods—Fourteen mushers completed 4 psychological instruments to assess state and trait anxiety, resilience and perceived stress, and self-reported upper respiratory symptoms (URS) in the month before the race. Mushers self-reported sleep duration and URS during the race.

Results—State and trait anxiety, resilience, and perceived stress did not differ between mushers with and without pre- and in-race URS ($P>0.05$). However, all mushers who reported in-race URS had reported URS ≤9 d before the race, and the onset of symptoms during the race typically occurred shortly after a rest period. Sleep duration was higher in mushers who reported in-race URS, both before (4.9±0.3 h, $P=0.016$) and during illness (5.9±1.3 h, $P=0.006$), vs mushers without in-race URS (3.4±0.8 h).

Conclusions—This study highlights recent illness, rest periods, and greater sleep requirements as potential risk factors for URS onset during a multiday endurance challenge, whereas psychological factors were not associated with URS.

Keywords: endurance, immunity, psychological stress, upper respiratory symptoms

Introduction

Prolonged physiological and psychological stress can negatively influence immune function.[1] For athletes, military personnel, and others working in extreme environments, understanding risk factors for illness could help to prevent illness and facilitate performance and completion of essential training. Decrements in normal immune function and increased incidence of upper respiratory symptoms (URS) and gastrointestinal symptoms may arise as a result of prolonged training sessions, exposure to extreme environments, inadequate nutrition, and disrupted sleep.[1] Recently, the potential moderating effect of psychological factors, such as state anxiety and perceived stress, on immune responses to exercise has been highlighted.[2] Continued research efforts are needed to understand how both physiological and psychological factors influence immune disturbances associated with physical stress.

Dogsled drivers, known as mushers, are a unique group of endurance athletes who compete in multiday races in extremely cold environments, undertaking heavy and prolonged physical exertion[3] and forgoing normal sleep.[4] These factors may contribute to an increased vulnerability to illness and thus makes this population an interesting model to study risk factors for common infections. In tandem, recommendations for performance

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Table 1. Overview of key variables and time point of collection

Variable measured	*Instrument used*	*Time point collected*	*N*	*Mean±SD*
Trait anxiety	STAI-Y2[6]	1 mo before race start	10	34±6
Resilience	BRS[7]	1 mo before race start	10	3.8±0.6
State anxiety	STAI-Y1[6]	Day before the race start	11	40±11
Perceived stress	PSS-14[8]	Day before the race start	9	27±4
URS in month before race	Modified JCCQ[9]	Day before the race start	Yes: 6 No: 6	Yes: Symptom score=6.5±5.2, duration=4±1 d
Sleep in month before race	Estimated sleep duration	Day before the race start	12	6.3±1.2 h
URS during race	Common cold symptoms? (yes/no)	Daily during race	Yes: 3 No: 7	Yes: Duration range=2–6 d
Gastrointestinal symptoms during race	Upset stomach? (yes/no)	Daily during race	Yes: 0 No: 10	
Sleep duration during race	Hours of sleep, reported to nearest half hour	Daily during race	10	3.9±1.2 h
Environmental temperature during race	Sensor attached to sled	Continuously during race (rests excluded)	4	Mean: −7.6±3.5°C Min: −39.4±1.0°C Max: 10.5±7.0°C

Data are reported as mean±SD unless otherwise stated. N refers to number of participants with complete data on a given variable.

optimization could benefit the sled dog racing community. Previous studies have typically focused on the occurrence of illness after endurance challenges.[5] However, during multiday dogsled races, it is likely that illness may occur during the race, which may affect mushers' ability to remain alert to the needs of their dogs and stay competitive in the race.

The Iditarod is one of the best-known long-distance sled dog races, traversing 1569 km across interior Alaska. Mushers and their dog teams typically complete the course in 9 to 14 d,[3] resting as needed at checkpoints every 30 to 130 km. Mushers must take one 24-h and two 8-h breaks during the race at checkpoints of their choosing. On arrival at a checkpoint, mushers' first priority is to take care of the dogs. The musher then attends to personal needs and may manage to sleep for a few hours before departure on the next segment.[3] The physical and psychological demands of traversing the trail combined with the responsibility to take care of the dogs means that fatigue and sleep deprivation are almost inevitable during the event. This setting provides an opportunity to investigate the influence of psychological factors on the ability to remain healthy during a multiday wilderness race involving multiple environmental, psychological, and physical stressors.

The main aim of this study was to investigate the influence of state and trait psychological characteristics and sleep quantity on the incidence of URS among competitors in the Iditarod sled dog race.

Methods

All 57 mushers who started the 2020 Iditarod were invited to participate in the study via email from the organizing committee. Participants contacted the research team for further information and provided informed consent to participate. The study was approved by the University of Alaska Fairbanks institutional review board and was conducted in accordance with the Declaration of Helsinki. The study employed a prospective cohort study design, where participants completed questionnaires before, during, and after the race. The main outcome measures were URS incidence before and during the race; participants were classified into groups based on these variables.

One month before the race, participants completed 2 questionnaires pertaining to trait psychological characteristics, intrinsic aspects of one's character that are relatively stable over time. These measurements included the trait aspect of the state-trait anxiety inventory (STAI-Y2) to measure trait anxiety, defined as a general tendency to perceive situations as threatening,[6] and the brief resilience scale, which measures resilience, or the ability

Table 2. State and trait psychological characteristics in mushers who reported pre-race and in-race upper respiratory symptoms (URS) versus those who remained symptom-free (healthy)

Variable: scale (score range)	*Pre-race*			*In-race*		
	URS Mean±SD (n)	*Healthy Mean±SD (n)*	*P*	*URS Mean±SD (n)*	*Healthy Mean±SD (n)*	*P*
State anxiety: STAI-Y1 (20–80)	43±12 (6)	36±8 (5)	0.29	47±16 (3)	40±5 (6)	0.54
Perceived stress: PSS (0–56)	27±4 (4)	27±4 (5)	0.88	27±5 (3)	27±3 (4)	0.86
Trait anxiety: STAI-Y2 (20–80)	32±6 (5)	35±7 (5)	0.53	28±6 (2)	36±6 (6)	0.18
Resilience: BRS (1.00–5.00)	3.90±0.65 (5)	3.67±0.52 (5)	0.55	4.42±0.83 (2)	3.64±0.47 (6)	0.13

to recover or bounce back after stress.[7] The day before the race start, participants completed 3 further questionnaires. Two instruments were used to assess pre-race state characteristics, or transient emotional states usually caused by external factors, after participants had arrived at the race start. The state aspect of the STAI (STAI-Y1) was used to assess state anxiety, transient anxiety experienced in a specific situation.[6] Perceived psychological stress was assessed using the 14-item perceived stress scale, which reflects the degree to which life situations were considered stressful by the mushers in the month leading up to the race.[8] Mushers then completed a modified version of the Jackson common cold questionnaire.[9] The first question asked whether the respondent had experienced a common cold in the past month. If the respondent answered yes, they were asked when the illness began, how many days it lasted, and to rate the severity of 8 symptoms (headache, sneezing, chilliness, sore throat, nasal discharge, nasal obstruction, malaise, and cough) on a 4-point scale (0=not at all; 1=mild; 2=moderate; 3=severe). Respondents who answered yes to the first question were categorized as having pre-race URS. Mushers were also asked to estimate their "average" sleep duration during the past month.

Participants carried a booklet on the trail that included daily questions regarding hours of sleep per 24 h, whether they thought they were suffering from a common cold that day (yes/no) or had an upset stomach (yes/no), and space for comments. Mushers answered these questions once every 24 h. Selected participants also carried a temperature logger (Thermochron 22L, iButton Link, Whitewater, WI), hung from the sled, that logged environmental temperature every 5 min throughout the race. Owing to last-minute COVID-19 restrictions at the finish line, mushers were requested to mail booklets and temperature loggers back to the research team; 10 of 14 booklets and 4 of 5 loggers were returned.

Mushers were categorized based on whether they reported pre-race and/or in-race URS. However, low participant numbers in the cohort and subgroups (Table 1) led to an a posteriori decision to report only descriptive statistics (mean±SD unless otherwise stated) and unpaired *t*-test results of differences between groups that did and did not report URS. Thus, no models to predict illness risk were constructed. Significance was accepted at $P=0.05$.

Results

The 2020 Iditarod race traversed Alaska via the 1569 km Northern route. Only 34 of 57 teams finished—the highest drop-out rate since 1974. Fourteen mushers provided informed consent to participate (10 male, 4 female, aged 39±11 y). Five participants were competing in the race for the first time, whereas others were experienced veterans. Seven participants completed the race in 9 to 14 d. Five participants dropped out owing to illness among the dogs, 1 was pulled from the race, and 1 dropped out owing to illness. On the trail, participants faced temperatures from −39.4 to 10.5°C (Table 1). We had limited success in obtaining complete data sets from all participants (Table 1).

Five male mushers and 1 female musher reported pre-race URS (Table 1). Five cases of URS presented 5 to 9 d before the race start, and 1 musher had ongoing URS at the race start.

Nine mushers completed the pre-race Jackson common cold questionnaire before the race start and returned data at the end of the race. Five remained healthy both pre-race and in-race. Of the 4 mushers who reported URS beforehand, 3 also reported URS during the race. Two episodes of URS occurred within 24 h after the compulsory 24-h break. One episode of URS occurred around the race finish. URS duration ranged from 2 to 6 d. Comments regarding URS that arose during the race mentioned "cough" (n=3), "sore throat" (n=1), "shortness of breath" (n=1), and "low energy" (n=3). Other mushers who did not self-report URS nevertheless reported "low energy" (n=2), "cough" (n=1), and "headache" (n=1) on occasion

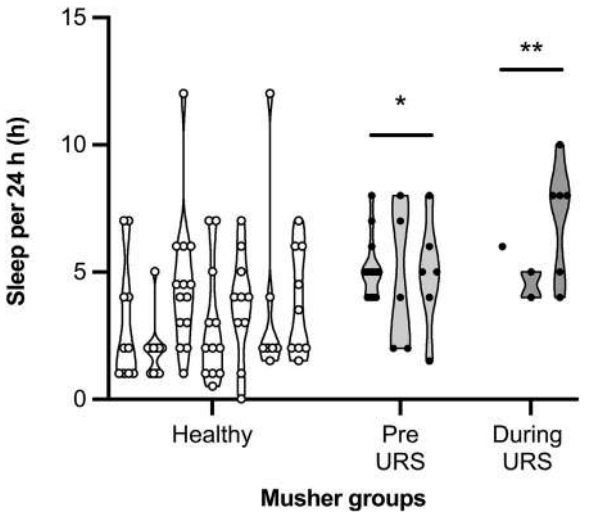

Figure 1. Sleep duration per 24-h period during the race. Individual violins represent nights of sleep for healthy mushers (white) and mushers with upper respiratory symptoms (URS) (gray), minimum to maximum. Individual dots represent individual 24-h periods during the race. The URS group is further divided into pre- and during-URS periods. Post-URS sleep data are not shown because only 1 participant's URS resolved before the race finish. Significant differences between URS and healthy group indicated: *P<0.05; **P<0.01.

(1 entry in all cases). No participants reported gastrointestinal illnesses during the race.

There were no significant differences in state (STAI-Y1 and perceived stress scale) and trait (STAI-Y2 and brief resilience scale) characteristics between mushers with or without URS either before or during the race (Table 2).

Sleep duration during the race was lower than that before the race (P<0.01; Table 1). Mushers without URS slept less than mushers with URS both before (3.4±0.8 vs 4.9±0.3 h, P=0.016) and during (3.4±0.8 vs 5.9±1.3 h, P=0.006) symptom onset (Figure 1).

Discussion

Field studies such as this provide insight into the demands of multiday wilderness races.[3,4] With a mean of only 3.9 h sleep per 24 h, our participants experienced sleep deprivation and environmental temperatures as low as −39.4°C. Illness was frequent both before and during the race, with half of the participants reporting URS before the race, 3 of 10 during the race, and one-off comments that could indicate illness reported by 3 further mushers during the race. We observed no significant differences in state anxiety, resilience, trait anxiety, or perceived stress between participants who self-reported illness before or during the race and participants who remained healthy. Sleep deprivation was more common among participants who remained healthy during the race and may have facilitated faster finishing times; the 2 fastest finishers among our cohort did not report URS either before or during the race.

The month before Iditarod involves numerous potential stressors for mushers, including selecting their dog team, training, developing their race plan, and traveling, often long distances, to the race start. Recent studies demonstrate that risk factors for developing URS include intensified training, long-haul travel, low energy availability, psychological stress, and depression.[1,10] Such risk factors could perhaps explain why half of our participants experienced URS in the month before the race. Unsurprisingly, the mean state anxiety was considered moderate[11] and perceived stress was higher than normative values[12] in the days before the race.

That all of the mushers who experienced in-race illness had also experienced illness in the month leading up to the race was an interesting observation, and pre-race illness was associated with in-race URS in 3 of 4 pre-race cases. We could speculate that in-race symptoms may have occurred via recurrence of symptoms from a pre-race common cold virus, given that participants likely had relatively little exposure to novel pathogens during the race, especially with COVID-19 restrictions. Nevertheless, during rests, participants could have been exposed to other mushers, volunteers, and locals who could be pathogen carriers, and mushers may have opted to rest after particularly arduous race segments. Of particular interest is the observation that mushers who experienced in-race illness in most cases developed URS symptoms shortly after their required 24-h break or after finishing the race. This may coincide with the classical post-exercise "open window" for opportunistic infections, whereby immune function is proposed to be temporarily compromised for a few hours after strenuous exercise.[13,14] Similarly, we could draw parallels with the phenomenon termed "leisure sickness," in which illness often strikes during weekends and vacations, times generally associated with relaxation, the mechanisms of which remain unclear.[15]

One previous study investigated sleep deprivation in mushers during Europe's longest sled dog race and similarly found that participants typically slept for 3 to 4 $h \cdot d^{-1}$.[4] Although chronic sleep deprivation is known to negatively affect immune function,[16] in the present study, mushers who remained healthy obtained less sleep than mushers with in-race URS, also before URS onset. One explanation may be that mushers who subsequently developed URS increased their sleep duration during the incubation period, which for a common cold is typically 2 to 5 d.[17] However, given that the mushers with in-race URS had also been ill before the race, the increased sleep may also be a lingering effect of prior illness.

LIMITATIONS

As is typical for studies performed in a remote field setting and competition environment, we encountered challenges in the design and execution of this study that limit the scientific rigor and conclusions that can be drawn. First, participant recruitment and retention was poorer than expected, and many participants returned incomplete datasets. This prevented us from making predictive inferences about the association between psychological traits and illness risk. Second, there are limitations with self-reported estimates of sleep duration. Objective monitoring of sleep would have strengthened these data.

We must also be cautious in interpreting symptom data, in particular during the race, because there was overlap in the symptoms noted in the comments between mushers who did and did not self-report illness using the yes/no question. Symptoms such as "shortness of breath" and "cough" could have arisen for reasons other than a common cold, such as cold exposure causing irritation to the upper respiratory tract[18]; likewise, "low energy" could have resulted from sleep deprivation or illness.

Because we did not collect biological samples, we cannot identify specific pathogens that could have explained the symptoms reported. Thus, we cannot assume that the symptoms were of infectious origin (hence, we also do not know if any illness could have been COVID-19). In addition, inclusion of other biological sampling procedures, such as measurement of salivary IgA, plasma cytokine, or leukocyte responses, would have enabled us to draw conclusions regarding biological responses to the stressors faced by participants during the race.

Conclusions

Our study highlights prior recent illness, rest periods, and greater sleep requirements as potential risk factors for symptom onset during a multiday wilderness event. State and trait psychological characteristics did not differ between mushers with and without illness. Future studies may wish to build on the present observations to elucidate critical time points for prevention of in-race illness during multiday endurance challenges and further assess the association between psychological factors, sleep, and illness risk in larger cohorts.

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BRIEF REPORT

Common Health Issues Encountered by Ultraendurance Ocean Rowers

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Introduction—Ocean rowing is an extreme ultraendurance sport in which athletes push themselves to their mental and physical limits while rowing across an ocean. Limited academic attention has meant health issues facing this population are poorly understood. This report provides a descriptive analysis of the injuries and illnesses encountered by ocean rowers at sea and suggests potential preventative measures.

Methods—Retrospective self-reported data were collected from ocean rowers via an online 29-question survey, classified by medical system, and totaled to produce a report of the most frequently encountered symptoms.

Results—Seventy-one ocean rowers, accounting for 86 ocean rowing attempts, completed the survey. Dermatologic symptoms formed 52% (n=169) of all reported issues, followed by musculoskeletal injuries (14%; n=45), mental health symptoms (11%; n=36), gastrointestinal symptoms (5%; n=16), and neurologic symptoms (2%). Gluteal pressure sores were the most common dermatologic symptoms (24%; n=40), hallucinations the most common mental health symptoms (69%; n=25), hand and finger issues the most reported musculoskeletal problems (36%; n=16); vomiting (38%) and headaches (50%) were the most common gastrointestinal and neurologic issues, respectively. Seasickness was reported in 42% of expeditions (n=33).

Conclusions—This report presents the physiological, mental, and medical challenges facing ocean rowers. Dermatologic and musculoskeletal issues were most common and varied greatly in severity. Over 90% of reported infections occurred as a dermatologic complaint, demonstrating the importance of preventative measures such as hygiene and wound care. Continued work with a larger population is required to further understand the physiological stress and medical complaints associated with transoceanic rowing.

Keywords: ocean rowing, rowing, ocean medicine, expedition medicine, dermatology, mental health

Introduction

Ocean rowing is a burgeoning sport where athletes row unsupported across an ocean, encountering some of the world's harshest and most unpredictable environments. Participants row in teams or solo across the Indian, Pacific, and Atlantic oceans, living isolated for months while facing unique physical and mental challenges.

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Ultraendurance events are emerging from the fringes of sporting endeavor, with the founding of ultraendurance races increasing public awareness and participation.[1,2] Ocean rowing is an extreme ultraendurance event, with expeditions lasting weeks or months, far longer than the 6 h generally considered to mark ultraendurance.[3] For those rowing in teams, individual shift patterns can be 2 h on, 2 h off,[1] with each crew member rowing for 12 h in every 24, whereas solo rowers average 10.3±1.1 h.[1] These athletes live and row in specially designed boats, carrying all supplies needed to survive for months at sea. Should rescue or assistance be necessary, emergency beacons and satellite phones provide means to access help.

As this sport's popularity grows, so must our understanding of the medical challenges involved. Previous research largely focused on individual case studies[4-7] or

small sample sizes,[1] ranging from nonfreezing cold injuries experienced by long-distance polar rowers[5] to physiological changes,[4] physical and psychological experiences,[6] and nutrition.[7] Only 1 study has examined the spectrum of medical issues encountered during a transatlantic race.[1] In contrast, extensive research highlights the common injuries experienced in sailing,[8,9] river rowing,[10,11] and kayaking/canoeing.[12]

This report's main objectives are to further introduce the wilderness medical community to the growing ultra-endurance sport of ocean rowing and provide a descriptive analysis of the medical conditions these rowers face.

Methods

Approval was gained from the King's College London's research and ethics committee before data collection for this retrospective descriptive study (reference LRU-19/20-14601).

The Ocean Rowing Society Facebook page is a platform where ocean rowers share knowledge and experience with individuals planning transoceanic crossings. Information on study objectives, use of personal data, and risks of participation (including publication of potentially identifiable information) was made available to page members with the online questionnaire.

Additionally, 88 ocean rowers who had recently attempted an ocean crossing, either as part of a transatlantic race or a publicized independent expedition, were contacted directly via their public expedition email addresses and social media. Only 1 attempt to make contact via email and social media was permitted per rower. As part of the questionnaire, consent was obtained from all participants before completing the survey. All data collected were anonymized and response order randomized to avoid participant identification via date of rower contact and order of survey completion.

Participants completed an online 29-question SurveyMonkey questionnaire covering demographics, weight loss, medical history, medical issues faced at sea and the impact these issues had on their expedition, help sought from external medical teams, medications used, and problems persisting 3 mo post-row.

Data were categorized by medical system and cumulative totals produced, allowing for disease pattern analysis and a descriptive report of common medical issues. Where a participant had completed multiple ocean rows, with no indication allowing for specific row analysis, their data were omitted. Data are presented both graphically and as a total incidence or intracategory percentage, alongside the total frequency where n>10. Where averages were determined, results are noted as mean±SD.

Table 1. Participant and ocean rowing demographics

Characteristics	*Values*
Participants (n)	71
Age, mean±SD (y)	37±12
Sex (n)	
Male	41
Female	16
No response	14
Transoceanic race (n)	58
Independent expedition (n)	16
Successful (n)	67
Rescued (n)	5
Ocean (n)	
Atlantic	69
Indian	4
Pacific	2
Mediterranean Sea	1
Southern	1
Other	1
Total ocean rowing attempts (n)	86
Time at sea, mean±SD (d)	46±18

Results

Approximately 70% of responses resulted from direct messaging with rowers or their team. Remaining responses were gathered from the Ocean Rowing Society Facebook page.

Participant and general ocean rowing demographics are presented in Table 1. Data covered 71 rowers aged 37±12 y, reporting on 86 ocean rowing attempts. Most participants were male (58%, n=41) and competing in a race. Time at sea ranged from 2 to 92 d with an average of 46±18 d, and the Atlantic was the most rowed ocean. Body weight changes indicated a loss of 12±6 kg. The majority rowed either as teams of 4 (34%; n=29), in pairs (27%; n=23), or solo (16%; n=14), with the rest in teams of various sizes ranging from 3- to 14-person boats.

Eight expeditions were excluded owing to participant errors completing the survey, leaving a total of 78. Eight medical categories were noted from the 323 reported medical issues. Dermatologic problems were the most common, accounting for 52% of the total issues reported (n=169), whereas acute traumatic injuries were the least common (1%).

Specific breakdowns of each category are seen in Figures 1 to 3. An average of 2 dermatologic issues per expedition were reported, with gluteal pressure sores and salt sores/chafing/rashes each affecting over half of all rowers (24% [n=40] and 23% [n=39] of all dermatologic cases, respectively). Blisters accounted for

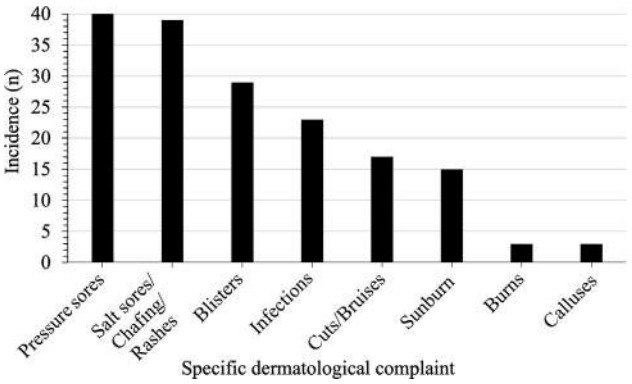

Figure 1. The 169 dermatologic issues experienced by ocean rowers are broken down into 8 subcategories of specific complaints.

17% (n=29), infections 14% (n=23), cuts/bruises 10% (n=17), and sunburn 9% (n=15), Figure 1.

Musculoskeletal problems (Figure 2) encompassed injuries to muscle groups or joints, equating to 14% (n=45) of reported issues, with over half of all rowers affected. Hand/Finger/Wrist symptoms represented 36% (n=16) of cases, knee issues 18%, both back and general musculoskeletal complaints 16%, arm pain 7%, and shoulder and hip pain 4% each.

Mental health issues encompassed 11% (n=36) of all reported complaints, present in 46% of expeditions (Figure 3). Hallucinations were the most common symptom reported (72%; n=26), affecting 32% of expeditions. Panic attacks accounted for 8% of cases, and anxiety and depression each comprised 6%. The remaining 8% were symptoms noncompatible with these subsections and therefore listed as "other."

Seasickness affected 42% of expeditions, representing 10% (n=33) of total medical complaints. Gastrointestinal issues were less common (5% overall; n=16), consisting of vomiting (38%), diarrhea (31%), and constipation (31%) (Figure 3). Neurologic cases accounted for 2% of total issues reported, composed of headaches (50%), specific limb numbness (25%), and dizziness/vertigo (25%) (Figure 3). Four injuries fell into the acute trauma category, including substantial ligament damage, broken bones, and trauma to the face/eye, mainly due to capsizes. Remaining health issues not categorized are classed under "other," including dehydration (n=4), sleep deprivation (n=3), heatstroke (n=3), loss of appetite (n=1), and other infections (n=1).

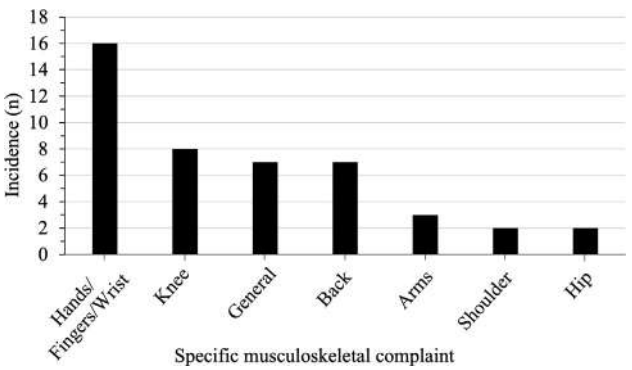

Figure 2. The 45 musculoskeletal self-reported issues could be further grouped into 7 main types. The category specific for general musculoskeletal issues refers to a generalized pain in most joints or muscle groups.

Discussion

This report details self-reported medical issues encountered by ocean rowers during these extreme ultra-endurance expeditions, providing insight into the demands on both body and mind.

Dermatologic problems ranged from minor to potentially debilitating. Abrasions, cuts, and blisters provided common routes for infection, with over 95% of reported infections occurring secondary to dermatologic complaints. Previous research also confirms infection rates to be as high as 92% in ocean rowers.[1] Confined and humid living conditions may predispose rowers to illness and infection, with gluteal pressure sores potentially at increased risk.[13,14] Improved hygiene advice and skin barrier protection may help mitigate risks. Burns from boiling water in turbulent seas could be prevented by the mandatory use of gimbles for stoves.

Regular shift patterns endured by transoceanic rowers result in protracted periods of physical effort, stress, and strain that likely contribute to musculoskeletal issues. Hand symptoms were common, often described as finger dislocation, stiffness, pain, swelling, difficulty with finger extension, or a "claw-hand." In-shore rowers also report this tightness in grip, where a carpel tunnel syndrome is often seen.[11] All rowers commonly report injury to back, knee, hip, arms, wrists, and shoulders[10]; however, condition severity in relation to the incidence reported in river rowers remains unclear.

Mental health symptoms occurred in just under half of ocean crossings, in keeping with previous research.[1] Physiological stress, severe sleep deprivation, and extreme exercise have been linked to emotional instability, hallucinations, and psychological issues.[15,16] Symptom severity and associated risk factors should therefore be a future research priority. Interestingly, a positive correlation between exertion and positive emotion has been previously reported in ocean rowers [6] and other endurance events,[2] conflicting with the frequency of mental health issues reported here. Further

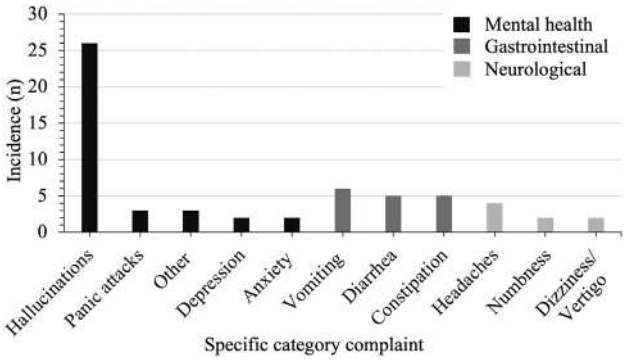

Figure 3. Thirty-six mental health self-reported issues were subcategorized into 5 specific issues, and 16 gastrointestinal and 8 neurologic complaints were differentiated into 3 specific categories, respectively.

investigation into ocean rowing-specific stresses, such as isolation and physical demands, may indicate whether psychological coping techniques have a place in rower preparation.[6]

Surprisingly, seasickness was only reported in just under half of all rowers despite small boats feeling the pitching of the sea more than larger vessels. However, an overlap with gastrointestinal or neurologic symptoms should not be excluded. Further work examining how debilitating seasickness is could aid understanding of its impact on rowers and provide support for familiarizing rowers with recommended medication regimes before departure.

Ocean rowers live on diets of 22.6 to 33.5 MJ (5400−8000 kcal) a day at sea.[1,7] This increased daily consumption, together with physiological stresses, constant exercise, and freeze-dried foods,[1] might explain the gastrointestinal issues reported. However, without definitive testing, infective causes such as gastroenteritis cannot be excluded.[17] Further studies may examine the potential benefits of rowers training while eating types and quantities of food similar to ocean diets before departure.

Various neurologic issues, including numbness in the leg/foot, headaches, and dizziness/vertigo, were noted. Leg numbness due to nerve compression is commonly seen in river rowers owing to impingement of the sciatic nerve and poor seat fitting.[11] Ocean rowers have limited space for stretching exercises, so attention should be paid to ensuring seat holes are spaced sufficiently for the ischial tuberosities, preventing nerve entrapment.

Chronic headaches were also reported, backing up previous findings in this population.[1] Both headaches and dizziness/vertigo have various potential etiologies, with stress, tiredness, motion sickness, and dehydration all possible contributors. One hypothesis is that dizziness may be due to the constant rolling motion disrupting inner ear calcium carbonate crystals, but further research is required.

Physiological stress may be demonstrated by the average weight loss reported. Research has suggested that low body mass index is an injury risk factor in those undertaking heavy exercise, owing to increased physical strain through weakened muscle groups.[18] Rapid decreases in muscle mass and body habitus may therefore contribute to a lack of joint stability and abnormal kinetic loading, leading to muscular/tendon pain. However, further research is needed to confirm this association. A previous study looking at 4 transatlantic rowers also noted weight loss, together with heightened proinflammatory cytokines and C-reactive protein, whereas anti-inflammatory markers were suppressed.[4] Although below levels indicative of infection, these markers might suggest background inflammation similar to other ultra-endurance events,[19] potentially contributing to reported musculoskeletal and neuropathic pain.

LIMITATIONS

These conclusions are limited by several important factors. First, only a small proportion of the total ocean rowing population were surveyed, and conclusions drawn at best indicate trends and reveal areas for future research. Likewise, the implications of variables such as age and sex are yet to be determined, with larger population samples required. Furthermore, the study population was contacted exclusively online, representing only those with access to the ocean rowing Facebook group or race websites. This limited the population and could create a bias toward more recent races and an overrepresentation of a younger demographic, who may also have a greater social media presence.[20] Additionally, participant first language was not considered, with only an English-language version available, and may have interfered with both participation and understanding. Self-reported data could lead to reporting biases wherein similar experiences are reported differently, depending on rower perceptions, or information is forgotten. Some individuals may list all issues encountered, whereas others may only note their most troubling issue. Further work is needed to understand which injuries/illnesses have the greatest impact and to contextualize physiological changes. This could involve linked pain scores, records of medical intervention, determining weight loss as a percentage change from a rower's departure weight, and sleep data collection. Pre-existing medical conditions, medications, and a comparison control group should also be analyzed.

Conclusions

This report catalogs the experiences of 71 ocean rowers participating in one of the most grueling ultraendurance sports. The study seeks to address the limited discourse about ocean rowing, particularly with respect to understanding the medical conditions encountered. Dermatological, musculoskeletal, and mental health issues most commonly affect ocean rowers, all with the potential to become debilitating. Areas where improved advice and understanding may protect athletes were also highlighted. Future studies have potentially wide-ranging implications for our understanding of ultraendurance events and the long-term effects of intense exercise in extreme environments on mental and physical health. Further research is needed to develop evidence-based advice to reduce risks and provide better medical care and treatment to athletes.

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CONCEPTS

Palliative Care's Role in Austere Medicine

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The integration of palliative care across multiple domains has increased in recent years, particularly in austere medical settings such as humanitarian crises and low- and middle-income countries. Providing relief from suffering through a multifaceted approach for patients in austere environments is both an ethical imperative and perfectly suited to palliative care's ethos. Practical resources have been developed that can guide many levels of medical practitioners in the administration of palliative care techniques in these settings. Further education and advocacy continue to be needed both for the promotion of primary palliative care and for access to certain classes of medications by which to deliver this care.

Keywords: symptom management, primary palliative care, wilderness medicine

Introduction

The rapid growth of the global population has seen a concurrent increase in the number of people living, working, traveling, and recreating in areas that are considered austere, or resource limited, environments, requiring the medical community to adapt and grow with the challenge of caring for patients in these settings. Palliative care is an integral aspect of quality medical care that has been expanding into austere medical settings over the past decade. The information presented in this article will clarify what palliative care is and how its inclusion in care delivery in austere settings such as the wilderness, humanitarian crises, and low- and middle-income countries (LMICs) is beneficial for both patients and providers.

Background

Palliative care is a practice approach that improves the quality of life for patients with both acute and chronic life-threatening or life-limiting conditions by preventing and relieving suffering, whether it be physical, psychosocial, or spiritual.[1,2] Key elements of palliative care include relief from pain and other distressing symptoms, holistic care, and an approach that affirms life yet does not fear death. Palliative care can be implemented in conjunction with therapies intended to prolong life and can therefore be offered at any point along the continuum of care.[3]

Palliative medicine has experienced significant growth in the past few decades, expanding from its roots in the hospice movement to a designation as a separate medical subspecialty with growing global recognition as international organizations acknowledge palliative care as an essential component of healthcare.[3] As the field of palliative medicine grows, its role and integration into various settings, such as patients' homes, outpatient clinics, emergency departments, and telemedicine, have been explored. Recently, the role and benefits of palliative care in austere medicine, defined as the practice of medicine in resource-limited environments,[4] has been increasingly discussed, particularly in the context of humanitarian crises and LMICs. Although some palliative care techniques, such as pain control, are not unique and are used by various levels of practitioners in wilderness environments, palliative care as a specialty has yet to be discussed in expeditionary or search and rescue contexts. However, these wilderness settings are another austere environment in which certain aspects of palliative care, such as a holistic approach and thorough management of pain and other distressing symptoms, can benefit patients.

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Benefits of Integrating Palliative Care in Austere Medical Settings

Because palliative care can be provided with a modest amount of medications and equipment, most of which are inexpensive and globally accessible,[1] it is feasible in resource-limited settings and offers benefits to both patients and providers.

WILDERNESS SETTINGS

Wilderness medicine is characterized by the provision of care in remote settings with limited resources, the need for improvisation, reliance on clinical examination and judgment, and delayed evacuation to definitive care.[5,6] By definition, wilderness medicine can occur in many contexts, but for the purposes of this paper, references to wilderness medicine and wilderness environments will be referring to medical care in settings such as search and rescue operations and expeditions. In these situations, taking a palliative care approach of focusing on thoroughly managing symptoms such as pain and anxiety can be beneficial to patients. This is especially important during technical extrications and prolonged evacuations to definitive care. Negative effects of undertreated pain include a significant stress response, increased sensitivity to pain the longer the pain remains uncontrolled, and an increased risk of developing posttraumatic stress disorder.[7] Negative effects of undertreated anxiety may also include hyperalgesia, in addition to the development of other distressing symptoms such as palpitations, nausea and vomiting, and shortness of breath.[7-9]

Medical providers in wilderness settings can use the Wilderness Medical Society guidelines for analgesic care in remote environments to effectively manage pain.[7] This stepwise approach encourages all providers to offer at least some form of pain management, starting with the safest and most accessible interventions before escalating care.[10] This involves using nonpharmacological interventions, such as comforting techniques and PRICE therapy (protection from further injury, rest, ice, compression, elevation) when appropriate before advancing to nonopioid medications, followed by opioid administration by providers with appropriate licensing. Implementing PRICE therapy in austere settings can be accomplished through improvisation. Splints can be created with items such as trekking poles to protect areas from further injury, and snow, ice, or cold water can be used to cool an injured extremity to relieve pain and reduce swelling. Compression can be attained using a compression bandage, if available, or an article of stretchy clothing wrapped around the extremity and tied securely, checking for continued perfusion beyond the wrap. For managing anxiety, nonpharmacological interventions, encompassed in the principles of psychological first aid, should be the mainstay of treatment. These include providing a safe physical space, offering reassurance and support, listening to the patient, reviewing the plan of care, and using relaxation techniques such as distraction and meditation.[7,8]

HUMANITARIAN CRISES

Palliative care has an important role in humanitarian crises. This has been acknowledged in the *Sphere Handbook*—one of the most widely used publications on principles and standards for humanitarian response—where palliative care is listed as an essential element of healthcare, and by the World Health Organization (WHO), which states that humanitarian responses that do not include palliative care are incomplete and unethical.[1,11] There are multiple humanitarian scenarios in which palliative care should be considered: in mass casualty events where resource limitations affect care delivery; in protracted crises where care for life-limiting conditions has been interrupted; and in outbreaks of disease where mortality rates are high and therapeutic interventions are limited.[12] In each of these scenarios, palliative care can be provided using the palliative care package from the *Lancet* commission on global access to palliative care and pain relief or the WHO essential package of palliative care medications and equipment (Table 1). These packages are simple enough to be used by physicians and nurses with basic symptom management training.[1]

Mass Casualty Events

In mass casualty events, healthcare providers frequently care for patients who are actively dying or whose poor chance of survival has precluded them from receiving limited life-sustaining resources. In these cases, implementing palliative care measures to ensure comfort and dignity during death fulfills the ethical imperative to aid these highly vulnerable patients.

Research investigating what patients and families consider to be a "good death" has shown that the key elements are 1) clear communication from healthcare providers, 2) adequate symptom management, 3) time with loved ones, and 4) preparation for death.[8] Although achieving these elements may be difficult in a humanitarian crisis, steps can be taken to further these aims. When possible, healthcare providers should promptly and clearly communicate prognosis to patients and families, with identification of a lead decision maker in situations where there are multiple

Table 1. Essential package of palliative care for humanitarian emergencies and crises

Interventions	*Inputs*			
	Social supports	*Medicines*[a]	*Equipment*	*Human resources*[b]
Prevention and relief of pain or other physical suffering,[c] acute or chronic, related to humanitarian emergencies or crises		Amitriptyline, oral Bisacodyl (senna), oral Dexamethasone, oral and injectable Diazepam, oral and injectable Diphenhydramine (chlorpheniramine, cyclizine, or dimenhydrinate), oral and injectable Fluconazole, oral Fluoxetine, oral Furosemide, oral and injectable Haloperidol, oral and injectable Hyoscine butylbromide, oral and injectable Ibuprofen (naproxen, diclofenac, or meloxicam), oral Lactulose (sorbitol or polyethylene glycol), oral Loperamide, oral Metaclopramide, oral and injectable Metronidazole, oral, to be crushed for topical use Morphine, oral immediate release and injectable Naloxone, injectable Omeprazole, oral Ondansetron, oral and injectable[d] Oxygen Paracetamol, oral Petroleum jelly	Pressure-reducing mattresses Nasogastric drainage and feeding tubes Urinary catheters Opioid lock boxes Flashlights with rechargeable batteries (if no access to electricity) Adult diapers or cotton and plastic	Doctors (with basic palliative care training) Nurses (with basic palliative care training) Community health workers (if available)
Prevention and relief of psychological suffering,[e] acute or chronic, related to humanitarian emergencies or crises		Amitriptyline, oral Dexamethasone, oral and injectable Diazepam, oral and injectable Diphenhydramine (chlorpheniramine, cyclizine or dimenhydrinate), oral and injectable Fluoxetine, oral Haloperidol, oral and injectable Lactulose (sorbitol or polyethylene glycol), oral	Adult diapers or cotton and plastic	Doctors (with basic palliative care training) Nurses (with basic palliative care training) Social workers or psychologists Community health workers (if available)
Prevention and relief of social suffering, acute or chronic, related to humanitarian emergencies or crises	Income and in-kind support[f]			Social workers Community health workers (if available)
Prevention and relief of spiritual suffering related to humanitarian emergencies or crises				Local spiritual counselors

[a]Based on WHO Model List of Essential Medicines 2015. Acceptable alternative medicines are in parentheses.

[b]Doctors may be local or foreign and may be surgeons, anesthesiologists, intensivists, infectious disease specialists, pediatricians, general practitioners, palliative care specialists, or others. Nurses may include nurse-anesthetists.

[c]Other physical suffering includes breathlessness, weakness, nausea, vomiting, diarrhea, constipation, pruritus, bleeding, wounds, and fever.

[d]Only at hospitals that provide cancer chemotherapy or radiotherapy.

[e]Psychological suffering includes anxiety, depressed mood, confusion or delirium, dementia, and complicated grief.

[f]Only for patients living in extreme poverty and for 1 caregiver per patient. Includes cash transfers to cover housing, children's school tuition, transportation to healthcare facilities or funeral costs; food packages; and other in-kind support (blankets, sleeping mats, shoes, soap, toothbrushes, toothpaste).

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caregivers.[8,13] Adequate management of common end-of-life symptoms such as pain, dyspnea, and agitation is crucial and can be achieved with opioids such as morphine, benzodiazepines such as lorazepam, and haloperidol.[8] Because symptom control is possible in almost any setting by using various routes for medication administration such as rectal, sublingual, and subcutaneous, dying patients can be cared for outside of the hospital; when paired with having nonmedical personnel stay with patients and their families, this can help offload medical staff and divert dying patients from overburdened hospitals.[8,13]

Although spending time with loved ones and preparing for death can be especially challenging in disaster contexts when the environment is chaotic and patients may die rapidly, efforts should be made to locate family members and help patients and families prepare for death with interventions such as culturally appropriate spiritual care and memory making, particularly if the patient is a child.[8]

Although palliative care may be of highest priority for patients triaged as imminently dying, patients triaged to immediate life-saving interventions also frequently need symptom management and psychosocial support, as do patients lingering with nonsurvivable injuries and complications. Palliative care is particularly valuable when surgical and life-sustaining resources are inadequate, as is often seen in sudden-onset disasters.[1] Preparing disaster response teams with the adequate training and supplies to relieve suffering and to recognize and treat end-of-life symptoms is a necessary step toward a comprehensive disaster response plan.

Protracted Crises

In protracted crises, healthcare workers have traditionally focused on treating patients with acute and curable conditions owing to limited resources, whereas the needs of those with life-limiting chronic and incurable conditions are largely, or even completely, unmet.[14,15] Many of those unmet needs could be addressed by palliative care. An illustration of this is seen in the Rohingya refugee crisis in Bangladesh, where many refugees have been found to have substantial neglected suffering, such as pain and caregiver strain, that could be mitigated by palliative care services provided by community health workers with palliative care training.[14,16] These services could include routine pain assessments; treatment of severe pain with locally manufactured oral morphine; access to essential medical supplies such as catheters and pressure-reducing mattresses, which are also manufactured locally; and caregiver training and psychosocial support.[14]

Disease Outbreaks

Palliative care can benefit patients during outbreaks of disease, where the management of distressing symptoms is at times neglected in the haste to save lives, although palliative care has more to offer beyond just the relief of suffering. This is seen with intensive control of symptoms such as diarrhea and vomiting, which not only reduces suffering but may also improve survival rates and provide better infection control.[1] Furthermore, in situations where therapeutic interventions are limited and curative treatments may not exist, palliative care may be the only acceptable option.[17] This was seen during the Ebola epidemic, where palliative care was frequently the only form of care available to patients.[18] Treatment of psychosocial suffering, which can be caused by both illness and the response to it, is another domain of palliative care that can be applied during outbreaks of disease. Fear, anxiety, depression, loss of self-esteem, a sense of powerlessness, and feelings of dehumanization may be experienced by patients in relation to isolation, the threat of a potentially fatal illness, and stigmatization during epidemics.[1,13] Psychosocial support can be given to patients by training healthcare providers and volunteers in psychological first aid, providing regular information to patients about their condition, organizing isolation wards so that patients can communicate with family at a distance or via technology, recruiting local mental health providers to offer culturally appropriate care, and ensuring access to antidepressants and benzodiazepines as appropriate. Forming partnerships with local religious leaders with infection control training who can visit patients on request and conducting education in the community about the disease to reduce fear and stigma are also helpful.[1] Lastly, pandemics can produce ethically challenging situations regarding the allocation of scarce resources, and providers trained in palliative care are well prepared to discuss preferences and advance care plans with patients and their caregivers, which can reduce resource strain.[13]

LOW- AND MIDDLE-INCOME COUNTRIES

One of the broadest austere settings where palliative care can benefit patients is LMICs, where medical care frequently occurs in resource-constrained environments with significant unrelieved suffering. More than 80% of the 25.5 million patients who died with serious-health related suffering in 2015 were in LMICs with limited to nonexistent access to palliative care and pain relief.[2] As such, improving access to palliative care in these countries can reduce significant end-of-life suffering. By providing pain and symptom management, palliative care

can also help patients suffering from preventable, infectious, and poverty-associated conditions related to health systems that fail to provide access to preventative services and life-saving treatments and interventions.[2] A prime example of this is the use of palliative care for cancer patients in LMICs, where late detection is common and there is insufficient access to specialists and treatments.[19,20]

Another unique role of palliative care in LMICs is as a poverty-reduction strategy, as the cost of care for incurable diseases often results in entire communities struggling to absorb medical expenses. Homes, livestock, and other assets are often sold to pay for medical care, which can quickly cause a family to slip into poverty or destitution. Added to this are indirect costs such as missed wages from family members caring for the patient and lost opportunities for children who are pulled out of school because the family can no longer afford the fees owing to medical expenses. These can all contribute to financial losses that are passed to future generations.[21] An extended cost-effectiveness analysis of the *Lancet* commission's palliative care package found that universal implementation of the package can reduce the risk of catastrophic medical expenses, which is one of the major causes of impoverishment in LMICs. The *Lancet* commission's palliative care package costs between $2 and $16 US per capita per year and is one of the least expensive components of the essential universal health coverage package.[2] As such, palliative care can help to reduce the financial burden and progression into poverty caused by incurable diseases by offering patients and families a more cost effective alternative than continuing to look for a cure that is not available.[21,22] Palliative care in the community may also offer cost savings in LMICs by reducing end-of-life hospital admissions.[2]

BENEFIT TO PROVIDERS

It is important to note that the provision of palliative care can be beneficial not just for patients, but for healthcare providers as well. In interviews conducted with healthcare providers who had responded to natural disasters, the inability to alleviate patient suffering was believed to exacerbate the trauma of the event.[23] Similarly, interviews with healthcare providers who had cared for Ebola patients revealed that providers continued to be deeply disturbed by their experiences of witnessing patients suffering immensely without relief. Therefore, a strong palliative care response in any situation likely to have significant suffering may help healthcare providers mitigate their own moral distress and vicarious trauma by empowering them to provide effective symptom management.[24]

Barriers to Implementation

There are barriers to providing palliative care in austere settings, and although some have been addressed, others continue to be a challenge. In wilderness settings, barriers are generally related to pain management and include lack of medications, challenges related to carrying and administering opioid analgesics, insufficient pharmacologic knowledge, concerns about addiction or adverse side effects from medications, and the potential of masking clinical deterioration with the administration of certain medications.[10,15] In LMICs, limited access to opioid medications is one of the most significant barriers; although nonopioid medications are an integral aspect of symptom management, opioid medications are often required to provide quality palliative care. Currently, 83% of countries have limited to no access to opioids, with LMICs accounting for just 10% of global opioid use.[3] Other barriers to palliative care in LMICs include lack of policies that support the provision of palliative care, little to no knowledge of palliative care among healthcare professionals, and cultural differences in the approach to serious illness or death that affect the delivery and acceptance of palliative care.[3] Providing palliative care in humanitarian emergencies may include the same challenge of access to opioids depending on location, in addition to the difficulty of triaging patients to determine who should receive curative-focused care versus a comfort-focused approach. A lack of palliative-specific training is a barrier to the implementation of palliative care in all austere settings.

Although the barriers to integrating palliative care into austere medicine may seem daunting, strategies exist to minimize or eliminate these barriers. First, efforts are being undertaken on a global scale to develop policies that support the provision of palliative care via laws that acknowledge palliative care as part of the healthcare system, clinical guidelines for the delivery of palliative care, and national strategies for palliative care implementation. Second, the palliative care community and related human rights organizations have been advocating for an appropriate balance between the prevention of illicit use of controlled substances and the human right to access medications for pain relief—in particular, opioids for palliative care in LMICs.[2,3] Improving the availability of licit opioids for palliative care can benefit patients in all of the previously discussed austere environments. For humanitarian relief efforts, responders can refer to the WHO model guidelines for transporting controlled medications across international borders for emergency medical care. Cultural barriers can be addressed through self-reflection on personal cultural values, an attitude of

humility and respect, and consulting and collaborating with local healthcare providers to deliver culturally sensitive palliative care.[25] One of the greatest ways to improve access to palliative care in any austere environment is to provide education on primary palliative care (PPC). This is defined as palliative care provided by primary healthcare workers in settings prior to specialized palliative care access.[26] Efforts are being undertaken globally to provide this education through international partnerships and even remote telehealth education. By furthering PPC education for wilderness first responders, humanitarian aid workers, and medical providers in LMICs, the likelihood increases that there will be a knowledgeable individual nearby when needed.

Available Resources

Organizations and healthcare providers across multiple specialties have developed some excellent resources that are useful for both PPC education and anyone interested in learning more about the integration and implementation of palliative care in austere medicine. For palliation in wilderness environments, the Wilderness Medical Society practice guidelines for acute pain management in remote settings provides guidance on ideal medication selection for wilderness use, along with a stepwise pain management pyramid for providing analgesic care for the typical backcountry patient. For palliative care in humanitarian emergencies and LMICs, the WHO guide to palliative care in humanitarian emergencies and the report by the *Lancet* commission on global access to palliative care and pain relief are replete with useful information. Both include an essential palliative care package of medications, equipment, and human resources developed specifically for use in LMICs, along with practical considerations for implementation. The WHO guide also contains a sample palliative care curriculum for training humanitarian responders, which can help to further PPC education. For more in-depth guidance, *A Field Manual for Palliative Care in Humanitarian Crises* is a tremendous resource focused on providing instruction to those not specifically trained in palliative care. A network of global partners working to further palliative care in humanitarian aid settings through research, advocacy, and education has also been developed by the Palliative Care in Humanitarian Aid Situations and Emergencies organization. All of these resources and more, such as virtual webinars, are available to help further PPC education and, where needed, address cross-cultural understanding of factors that affect the delivery of palliative care.

Conclusions

It is important to note that the need to integrate palliative care in austere medical settings in no way replaces the importance of providing mortality-reducing care when available. Instead, both types of care should be delivered conjointly whenever possible. This can be achieved by taking steps to educate all medical practitioners in PPC techniques. Gaining this knowledge base should result in both increased quality of care in austere settings and achievement of the ethical imperative to reduce suffering in all care settings. This transformation will take the concerted global effort of individuals and organizations to advocate for PPC education and the resources necessary for its implementation.

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CASE REPORT

Atypical Hemolytic Uremic Syndrome in a Patient With *Bothrops asper* Envenomation

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Bothrops asper envenomation is common in Colombia and is characterized by local tissue injury and venom-induced consumption coagulopathy (VICC). Rarely, thrombotic microangiopathy is associated with envenomation by this species. The case of a 57-y-old man with *B asper* bite and envenomation on the left foot is presented. The patient was admitted 8 h after the event and progressively developed edema, hemorrhage at the site of the bite, and hemorrhagic blisters. His coagulation test results (prothrombin and partial thromboplastin times) were prolonged, and his fibrinogen levels were severely reduced. The diagnosis of VICC was made. Administration of Colombian polyvalent viper antivenom controlled the VICC within a few hours. Subsequently, the patient developed severe microangiopathic anemia, thrombocytopenia, and acute kidney injury. A diagnosis of thrombotic microangiopathy was made, and the patient met the criteria for hemolytic uremic syndrome. Management with hemodialysis in addition to therapeutic plasma exchange and replacement with fresh frozen plasma was indicated. The patient's condition resolved 14 d later. To the best of our knowledge, this is the first case of *B asper* envenomation in which the patient presented with hemolytic uremic syndrome after VICC. A proposal is made regarding the pathogenesis of this chain of events.

Keywords: snakebite, coagulopathy, thrombotic microangiopathy, acute kidney injury

Introduction

Acute kidney injury (AKI) occurs in approximately 5% of cases of envenomation by snakes of the genus *Bothrops* in Colombia.[1–3] In other areas of the world, such as Asia, AKI can occur in up to 29% of patients after snakebite, and *Echis carinatus*, *Daboia russellii*, *Cryptelytrops* spp, *Trimeresurus* spp, *Protobothrops* spp, *Hypnale hypnale*, and *Hydrophinae* spp are the most frequently involved in this complication.[4] AKI is mainly related to myotoxic or hemotoxic effects in addition to hypotension.[5–7] Acute tubular necrosis is the most common cause of AKI (80%), followed by interstitial nephritis (15%) and, more rarely, thrombotic microangiopathy (TMA) (approximately 5% of cases).[8,9] The species involved in the development of TMA are *Hypnale hypnale*,[10–13] *Pseudonaja* spp,[14] *Notechis scutatus*,[15] *Daboia russellii*,[16,17] *Bothrops jararaca*,[18,19] *Echis coloratus*,[20,21] *Bothrops erythromelas*,[22] and *Bothrops lanceolatus*.[23] Some cases of TMA may progress to renal cortical necrosis.[24]

The triad of AKI, thrombocytopenia, and hemolytic anemia with fragmented erythrocytes (schistocytes) is the essential criterion for the diagnosis of hemolytic uremic syndrome (HUS) and has been maintained since HUS was first described.[25,26] HUS is extremely rare and has only been reported in isolated cases or in a small series of cases of envenomation by *Daboia russellii*,[27] *Oxyuranus scutellatus*,[28] and *Hypnale hypnale*.[29]

Neither TMA in general nor HUS in particular has been reported in association with *Bothrops asper* envenomation. The present case study is the first published description of this condition.

Case Report

A 57-y-old male military professional, while on the banks of the Anchicayá River in the Colombian Pacific region (southwestern Colombia), was bitten on his left foot by an adult *B asper*. The species was identified by a ranger.

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Figure 1. *Bothrops asper* (Garman, 1884), "Terciopelo." Local name in Colombia: "Taya equis." It is a species of venomous snake of the Viperidae family with a wide distribution in southwestern Colombia. *B asper* causes the highest number of snake envenomations in Colombia. Its venom is proteolytic, histiolytic, vasculopathic, cardiotoxic, nephrotoxic, and coagulopathic.

The snake was approximately 1.5 m in length; it was collected from the ground and relocated to the surrounding area. *B asper* is endemic to the area and is widely known as "Taya X" (Figure 1).[3] The patient was transferred to our hospital, where he was admitted 8 h after the event. He had history of mild arterial hypertension and was receiving chronic management with hydrochlorothiazide and losartan.

On admission, the patient was in apparent good condition, with blood pressure of 135/82 mm Hg and heart rate of 78 beats·min^{-1}. His head, neck, and sensory organs were normal, with normal cardiopulmonary, abdominal, and neurologic examination. Extremities were normal, except the dorsum of the left foot, where there was a puncture wound corresponding to a bite with a fang, with active bleeding and the development of an adjacent hemorrhagic blister 1 cm in diameter; moderate edema was also observed (Figure 2).

Admission laboratory examination results showed creatinine at 2.28 mg·dL^{-1} (normal 0.67−1.17), blood urea nitrogen (BUN) at 40 mg·dL^{-1} (normal 6−20), hemoglobin at 16.5 g·dL^{-1} (normal 13.7−17.5), and platelets at 225,000 µL (normal 163,000−337,000). Leukocytes were 8940/mm^3 (normal 4230−9070) with 6600/mm^3 neutrophils (normal 1780−5380), 1840/mm^3 lymphocytes (normal 1320−3570), 400/mm^3 monocytes (normal 300−820), and 100/mm^3 eosinophils (normal 40−540). A noncoagulating prothrombin time (PT) and thromboplastin time (TPT), undetectable fibrinogen levels, and D-dimer concentration of 3.5 µg·dL^{-1} (normal <0.50) led to the diagnosis of venom-induced consumption coagulopathy (VICC).

A total of 15 vials of polyvalent antivenom was required; the initial dose was 8 vials, according to the recommendations given by national guidelines, depending on the classification of the envenomation,[30] and subsequent doses were added every 6 h until the patient's coagulation tests were normalized. At 6 h, 3 vials were used (PT: noncoagulating, TPT: noncoagulating, fibrinogen: 50 mg·dL^{-1}); 12 h later, 2 vials (PT: 30 s, TPT: 60 s, fibrinogen: 150 mg·dL^{-1}), and 18 h later, 2 vials (PT: 14, TPT: 34, fibrinogen: 175 mg·dL^{-1}). Each vial of polyvalent antivenom (Laboratorios Probiol, Bogotá, Colombia) contains 10 mL of equine polyvalent antivenom, which neutralizes at least 25, 10, and 5 mg of the venom of *B atrox/asper*, *Crotalus durissus,* and *Lachesis muta*, respectively. The VICC was controlled by 18 h.

Forty-eight hours after admission, the patient presented with oliguria. He was pale and jaundiced. Erythema and increased local heat began to be observed on the back of the left foot. Given suspicion of the onset of an infectious process, piperacillin-tazobactam at a dose adjusted to his kidney function was initiated. Magnetic resonance imaging of the left foot was performed

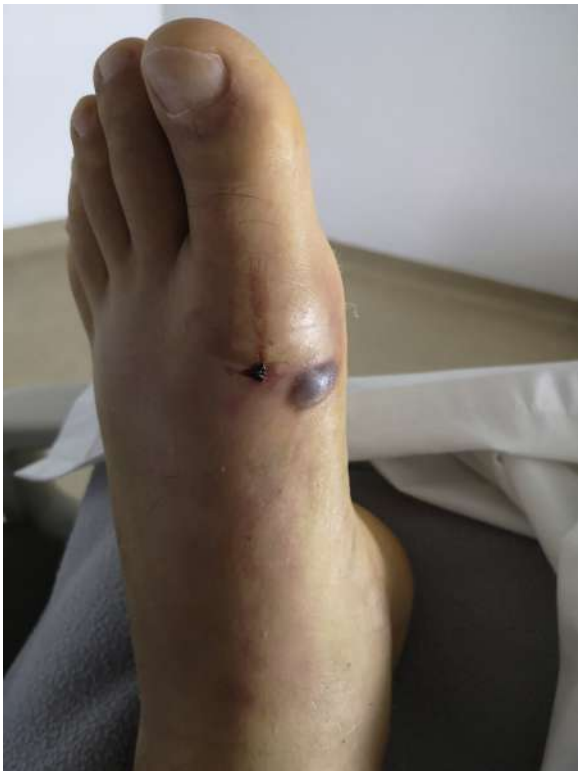

Figure 2. Punctate wound corresponding to a bite with a fang on the dorsum of the left foot, with evidence of bleeding and the development of an adjacent hemorrhagic blister 1 cm in diameter; moderate edema was also observed.

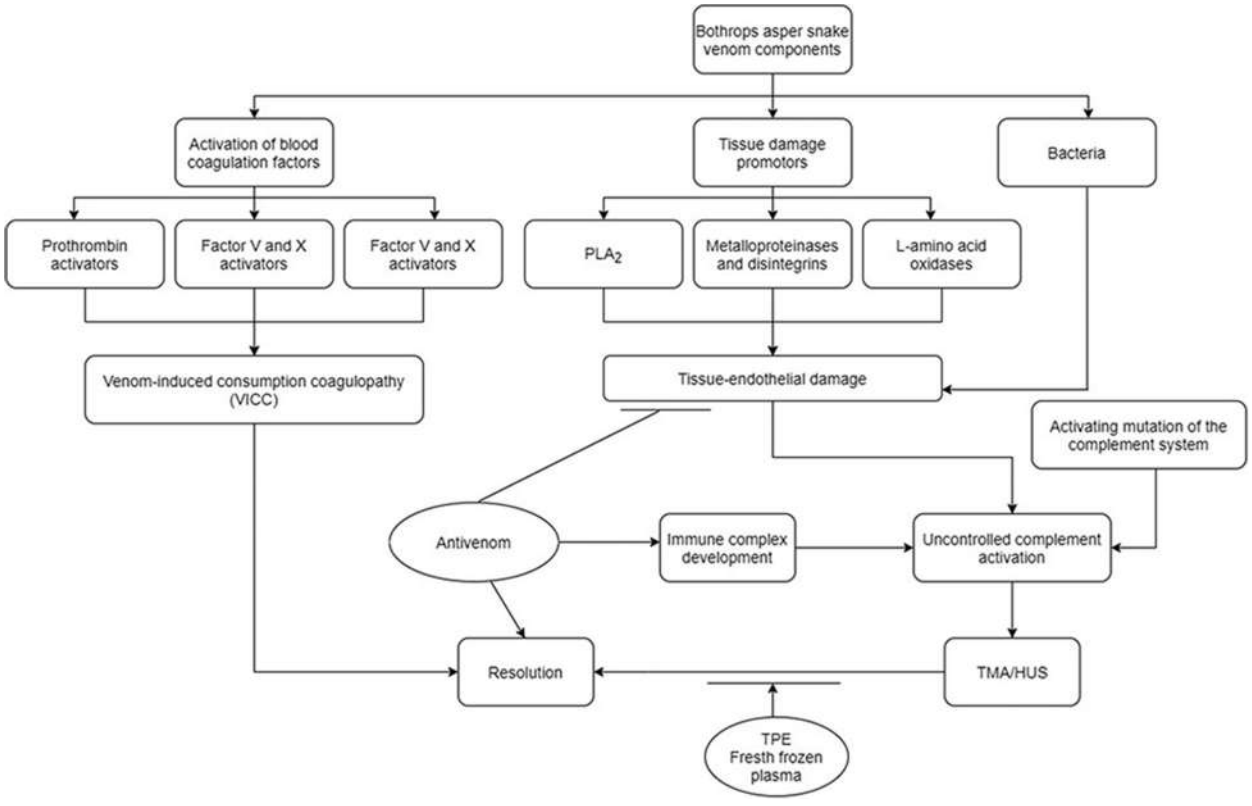

Figure 3. The possible sequence of events that occurred in our patient. Treatment actions are indicated by horizontal line. Typical venom-induced consumption coagulopathy (VICC) developed due to the effect of various proteins that activate clotting factors. He improved with the application of antivenom. The patient then developed thrombotic microangiopathy (TMA), probably as a consequence of a defect in the regulation of complement that could be activated by the effects of endothelial damage caused by other protein elements of the venom, the presence of an infection, and the use of antivenom. TMA with the presence of clinical and laboratory elements consistent with hemolytic uremic syndrome (TMA/HUS) was successfully controlled with hemodialysis and therapeutic plasma exchange (TPE) with fresh frozen plasma.

and showed considerable soft tissue edema without evidence of fluid collection. There was a decrease in hemoglobin to 10 $g \cdot dL^{-1}$. Lactate dehydrogenase (LDH) was 1100 $U \cdot L^{-1}$ (normal 135–225), haptoglobin was undetectable, and indirect bilirubin was 17 $mg \cdot dL^{-1}$ (normal 0.3–1.9). These findings are diagnostic of hemolytic anemia. Peripheral blood smears showed schistocytes, a decrease in platelets to 9000 μL, BUN 78 $mg \cdot dL^{-1}$ (normal 7–20), creatinine 5.8 $mg \cdot dL^{-1}$ (normal 0.59–1.04), complement component C3 98 $mg \cdot dL^{-1}$ (normal 90–180), and C4 8 $mg \cdot dL^{-1}$ (normal 10–40). The findings of hemolytic anemia in conjunction with thrombocytopenia and kidney injury are highly suggestive of TMA. Due to hyperkalemia (potassium 5.49 $meq \cdot L^{-1}$), acidosis (pH 7.26 and HCO_3 17.1), and edema, we decided to start daily hemodialysis and hemofiltration as needed. Given the suspicion of HUS, we decided to start concomitant therapeutic plasma exchange (TPE) with replacement with fresh frozen plasma. The patient required 4 TPE sessions. The hemolytic anemia was controlled, and his platelet levels gradually increased.

Twenty days after admission, the patient had significant improvement of the left foot injury. He presented with normal urine output and improved laboratory parameters: hemoglobin 9.8 $g \cdot dL^{-1}$; leukocytes 4752/mm^3, with 2450/mm^3 neutrophils, 1250/mm^3 lymphocytes, 50/mm^3 monocytes, 2/mm^3 eosinophils, and 234.000/mm^3 platelets; BUN 24 $mg \cdot dL^{-1}$; creatinine 1.6 $mg \cdot dL^{-1}$; LDH 110 $mg \cdot dL^{-1}$; indirect bilirubin 0.9 $mg \cdot dL^{-1}$; sodium 139 meq/L^{-1}; and potassium 4.7 $meq \cdot L^{-1}$. Complement components were C3 102 $mg \cdot dL^{-1}$, C4 14 $mg \cdot dL^{-1}$, creatine phosphokinase (CPK) 98 $U \cdot L^{-1}$, aspartate aminotransferase (AST) 24 $UI \cdot L^{-1}$, and alanine aminotransferase (ALT) 18 $UI \cdot L^{-1}$.

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. This report was approved for publication by the ethics committee of Fundación Valle del Lili.

Discussion

VICC is a coagulopathy that occurs in envenomation by snakes whose venom contains proteases that act by stimulating coagulation factors or simulating their structure and function. Clotting factors are rapidly consumed, clotting times are prolonged, hypofibrinogenemia develops, and D-dimer increases.[31] In the specific case of snakes of the genus *Bothrops,* prothrombin activators,[32,33] factor X activators,[34] and thrombin-like enzymes[35] have been identified. Similar to thrombin, thrombin-like enzymes share the property of the transitioning fibrinogen to fibrin, but they do not activate factor XIII, which is responsible for stabilization of fibrin clotting, and thus lead to VICC. VICC is resolved by the synthesis of new coagulation factors and neutralization of toxins. The main complication is bleeding. VICC differs from disseminated intravascular coagulation in several ways: It does not present with any evidence of systemic microthrombi and end-organ failure[36] and has other etiologic, pathophysiological, and prognostic factors.[37]

Our patient presented with classic VICC with fibrinogen consumption, prolonged coagulation tests with clinical evidence of a bleeding tendency based on bleeding from the bite orifice, and the presence of hemorrhagic blisters. The patient responded adequately when treated with Colombian polyvalent viper antivenom, and his coagulation tests were normalized within a few hours of treatment. After recovery from VICC, the patient presented low urine output with uremia, his platelet levels began to decrease, and severe hemolysis developed, with evidence of schistocytes in the peripheral blood smear. This indicated the presence of TMA in the absence of criteria for thrombotic thrombocytopenic purpura that met the criteria for HUS. Similar cases have been reported that initially presented as VICC and were followed by the development of thrombocytopenia, TMA, and AKI.[14] The predilection toward kidney involvement seems to be characteristic of TMA associated with HUS caused by snake envenomation, a finding that should be differentiated from occasional initial prothrombotic phenomena in the presence of envenomation by vipers, which tends to affect the cerebral arteries, specifically those at the base of the skull.[38]

In HUS, there is endothelial damage, which in the kidney manifests as thickening of the vascular walls with detachment of the endothelial cells of the glomerular basement membrane and the formation of microthrombi with variable degrees of vessel occlusion, a condition that causes erythrocytes to rupture when passing through this partially occluded microvasculature.[39] Two forms of HUS have been described: typical and atypical. The most frequent (90% of cases) is typical (or classic) HUS, which is associated with a diarrheal syndrome caused by *Escherichia coli* O157:H7, a producer of Shiga toxin, which binds to Gb3 receptors (globotriaosylceramide) on the surface of endothelial cells and causes their lysis directly or through activation of proinflammatory and/or procoagulant mechanisms.[40] The majority of patients with typical HUS progress satisfactorily; however, approximately 10% progress to chronic kidney disease.[41]

In atypical HUS (less than 10% of HUS cases), the disease develops with no relation to infection with *Escherichia coli* O157:H7 and has been associated with mutations of various complement proteins that favor its activation against different stimuli. Some of these mutations associated with atypical HUS are activators, such as those described in factor B[42] or C3,[43] as well as mutations of regulatory proteins such as factor H,[44,45] factor I,[46] and MCP (membrane cofactor protein-CD46).[47,48] This knowledge has led to the development of therapeutic strategies aimed at inhibiting the complement cascade, as in the case of eculizumab, a monoclonal antibody directed against the C5 protein.[49] Patients with atypical HUS have a less favorable prognosis and more frequently develop chronic kidney failure,[50] which justifies the use of inhibitory treatment strategies in patients with associated mutations.[51] The etiologic factors that can trigger atypical HUS in a genetically susceptible individual include various viral or bacterial infections, autoimmune disease, the use of oral contraceptives, chemotherapeutic agents, pregnancy, and postpartum status, among others.[52,53]

In the particular case of snake envenomation leading to the development of atypical HUS, we are not aware of a study of any mutations in the related complement cascade proteins. The outcomes of these patients and the results of different proposed treatments, such as the use of fresh frozen plasma[10] and TPE,[54] have been reported mainly in observational studies,[55] such as that of Wijewickrama et al. in a series of 103 patients with *Hypnale* spp envenomation.[56] A randomized controlled study did not demonstrate the utility of fresh frozen plasma.[57] There is no experience with the use of eculizumab.

Thus, we present a patient with *B asper* envenomation who developed typical VICC that responded adequately to polyvalent antivenom for Colombian vipers and who, 56 h after the bite and after initiation of established treatment, presented with TMA associated with HUS. By definition, it was atypical HUS, which has a variable etiology, including a genetic predisposition toward unregulated complement activation in response to different events that cause vascular endothelial damage. In this case, there could be a genetic predisposition and an external condition related to the venom components,

including phospholipase A2,[58] metalloproteinases/disintegrins,[59,60] and L-amino-acid oxidases,[61] among others. Microorganisms that were inoculated during the bite could also be involved in the development of atypical HUS; less likely is an adverse reaction to the antivenom, which can cause serum sickness, an immune complex–mediated immune reaction that clinically manifests as arthritis, rash, nephritis, and consumption of complement.[62] Our patient presented a slight decrease in the C3 component of complement, which could be related to its activation in the presence of HUS and/or a slight immune complex–mediated reaction to the use of antivenom. Figure 3 schematically shows the pathogenic events that may be related to the patient's clinical presentation.

VICC in our patient was managed with antivenom, and he had an adequate clinical response. The TMA and HUS were managed with TPE with fresh frozen plasma despite the fact that there is no evidence of a consistent response in this type of case. The disease resolved 3 weeks later. Piperacillin-tazobactam was added to his treatment after suspicion of the onset of a local infection. To the best of our knowledge, this is the first case of *B asper* envenomation in which the patient presented with TMA with manifestations related to HUS after VICC typical of this type of envenomation. Although our patient had an adequate response with fresh frozen plasma and TPE, further studies need to be performed to assess the significance of these treatments in these types of patients.

In 2009, cases of VICC were retrospectively analyzed[63] and showed that the use of coagulation factors (fresh frozen plasma and/or cryoprecipitate) after the use of antivenom is associated with earlier improvement of coagulation function. However, it is important to note that there is evidence that the use of fresh frozen plasma did not reduce coagulopathy in Russell's viper envenomation,[57] and some low-quality studies (especially in animal models)[64] show that the use of fresh frozen plasma can be deleterious, increasing the presence of thrombotic complications; for this reason, antivenom is the mainstay of the treatment of VICC and should always be administered.

Author contributions: CAC, MJ-V, and IP-O wrote the manuscript and contributed to the medical observations. All authors read and approved the final manuscript.

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CASE REPORT

A Tropical Kiss by a Malabar Pit Viper

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Snakebite in India is often attributed to the "big 4," for which polyvalent anti-snake venom is effective. Also significant and less known is the burden of other venomous snakes, one of which is *Trimeresurus malabaricus*. We report a bite to the face of a tree climber by *Trimeresurus malabaricus* in the Western Ghats of India, which caused severe local envenomation in the form of facial edema and systemic signs of envenomation, including coagulopathy and hypotension. We discuss the role of thromboelastogram, infrared thermography, and routine diagnostics in this case, which led to the administration of Indian-made polyvalent anti-snake venom. The patient recovered and was discharged without any clinically evident physiological or physical dysfunction.

Keywords: Trimeresurus malabaricus, envenomation, thromboelastogram, venom induced consumptive coagulopathy, snakebite, antivenom

Introduction

Snakebite is a neglected tropical disease, with India burdened by the highest number of deaths globally. Snakebite is attributed most commonly to the "big 4," namely, the Russell's viper (*Daboia russelli*), spectacled cobra (*Naja naja*), common krait (*Bungarus caeruleus*), and the saw-scaled viper (*Echis carinatus*). However, many other snakes of medical importance have been described over the years.[1-5] In the Western Ghats of India, the malabar pit viper (MPV) (*Trimeresurus malabaricus*), bamboo pit viper (*Trimeresurus* gramineus), and large-scaled pit viper (*Trimeresurus macrolepis*), known *Trimeresurus* spp, are venomous and predominantly arboreal.[4] No specific anti-snake venom (ASV) exists for these species in India.

MPV envenomation remains largely unreported from the Western Ghats and coastal regions of southwestern India. In a hospital-based registry from the hospital that treated this case, MPV envenomation was more common than *Echis carinatus* and *Bungarus caeruleus* envenomation. We report a case with multiple bites to the face, potential upper airway obstruction due to severe cellulitis caused by the envenomation, coagulopathy caused by MPV, the potential neutralizing capacity of poly-specific Indian ASV in the treatment, and the role of thromboelastography in assessing the coagulopathy of MPV envenomation.

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Case Report

A 44-y-old male, with no known comorbid conditions, from Honnavara, North Karnataka presented to the emergency department with swelling of the left side of the face after experiencing a snakebite while climbing a coconut tree at 1100. He was bitten twice, after which he immediately withdrew, killed the specimen, and presented to a local hospital where he was given tetanus immunization, diclofenac, and chlorpheniramine maleate and transferred to our hospital.

The patient arrived 6 h after the bite to the emergency department. The dead specimen was identified as an MPV (Figure 1) based on morphologic characteristics and the physician's knowledge of species distribution in the region. On examination, he was conscious, oriented, and hemodynamically stable with a pulse rate

Figure 1. (A, B) Killed specimen, identified as malabar pit viper (*Trimeresurus malabaricus)*: green morph from Honnavara district-Near Sharavati, 14.2°N, 74.5°E. (C) Oral cavity of the specimen showing 2 fangs on the right with the left fang broken off. (D) Killed specimen. Full length of 57 cm with the typical black/brown spots over the dorsum arranged in a bold zig-zag pattern.

of 60 beats·min^{-1}, blood pressure of 120/70 mm Hg, hemoglobin oxygen saturation of 100% on room air, and respiratory rate of 18 breaths·min^{-1}. An airway examination indicated no immediate threat to his airway. The local examination was painful with a diffuse swelling of the left cheek with multiple fang

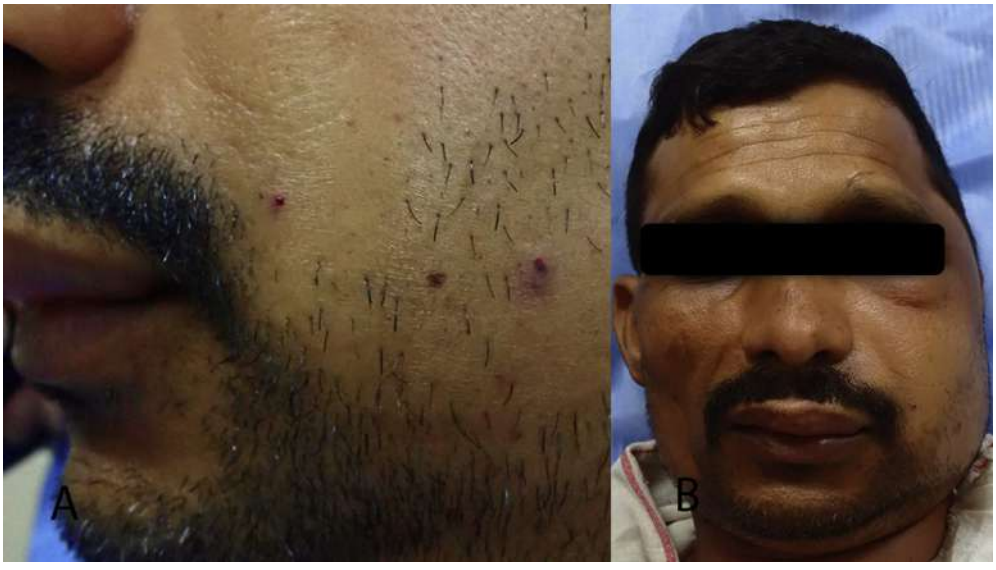
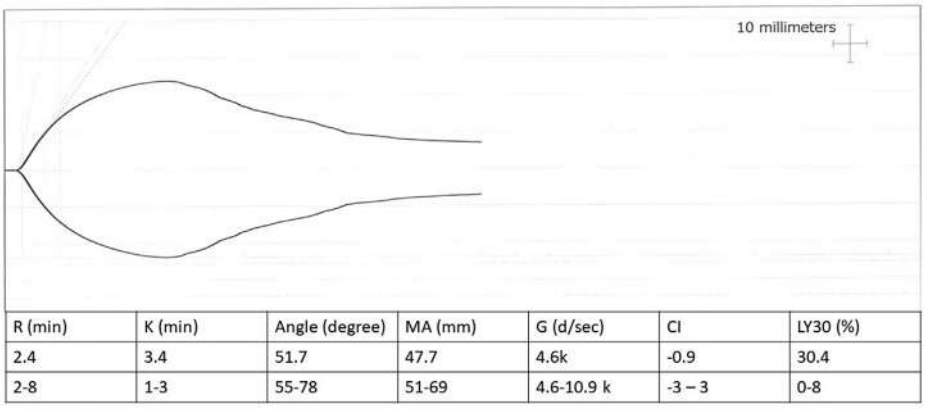
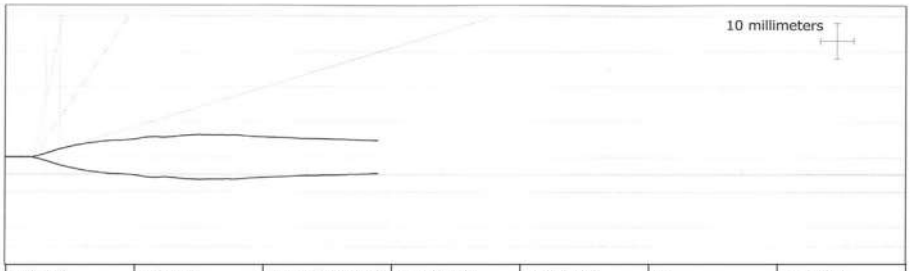


Figure 2. (A) Fang marks: 2 sets of fang marks with 1 indistinct over the cheek. (B) Victim's face showing fang marks on the left cheek with edema extending to the neck.



R (min)	K (min)	Angle (degree)	MA (mm)	G (d/sec)	CI	LY30 (%)
2.4	3.4	51.7	47.7	4.6k	-0.9	30.4
2-8	1-3	55-78	51-69	4.6-10.9 k	-3 – 3	0-8

A



R (min)	K (min)	Angle (degree)	MA (mm)	G (d/sec)	CI	LY30 (%)
5.9	Not Recorded	16.2	9.8	0.5	NR	0
2-8	1-3	55-78	51-69	4.6-10.9 k	-3 – 3	0-8

B

Figure 3. (A) TEG at the time of admission showing a mild hypocoagulable state with primary hyperfibrinolysis. (B) TEG after 24 h showing a severe hypocoagulable state, probably due to fibrinogen deficiency and no fibrinolysis.

marks (Figure 2). Other systemic examinations were unremarkable.

A 20-min whole blood clotting test on admission was positive. A prothrombin time with international normalized ratio (PT-INR) was normal, and the thromboelastogram (TEG) revealed a mild hypocoagulable state with primary fibrinolysis with a normal R time (Figure 3A). ASV was not started. He was admitted in the emergency intensive care unit for further evaluation and observation and because of the high possibility of the need for airway intervention. A serial PT-INR done 14 h after the bite was prolonged, and TEG revealed a hypocoagulable state

Table 1. Laboratory findings

	Reference range	*6 h (Day 1)*	*18 h (Day 2)*	*22 h (Day 2)*	*42 h (Day 3)*	*66 h (Day 4)*
Hb	13–17 g·dL^{-1}	14	12	12	11	na
TLC	4–10 μ·L^{-1}	9500	8800	na	4600	na
PLT	150–400x10^{3} μ·L^{-1}	183x10^{3}	177x10^{3}	na	122x10^{3}	119x10^{3}
Urea	10–40 mg·dL^{-1}	22	32	na	24	20
Cr	0.7–1.2 mg·dL^{-1}	1.3	1.1	na	1.0	1.0
CPK	20–300 U·L^{-1}	577	na	na	na	na
LDH	125–220 U·L^{-1}	232	na	na	na	na
Urine pH	4.5–7.5	6.0	na	na	na	na
Protein	<30 mg·dL^{-1}	30	na	na	na	na
Sugar	2–20 mg·dL^{-1}	250	na	na	na	na
RBC	0–3/hpf	0.2	na	na	na	na
WBC	0–5/hpf	0.1	na	na	na	na

CPK, creatine phosphokinase; Cr, creatinine; Hb, hemoglobin; LDH, lactate dehydrogenase; na, not done/not available; PLT, platelet count (lakh); TLC, total leukocyte count; hpf, high power field.

Table 2. Coagulation parameters

	Reference range	*6 h (Day 1)*	*14 h (Day 2)*	*35 h (Day 2)*	*43 h (Day 3)*	*67 h (Day 4)*
20WBCT	20 min	Positive	na	Negative	na	na
PT	9.6–12.5 s	12.6	24.2	18.1	14.9	11.9
INR	Reference ranges as per Figure 3	1.2	2.3	1.7	1.4	1.1
aPTT	26.8–33.2 s	25.2	25.1	21.9	22.2	22.6
TEG		Mild hypocoagulable state with primary fibrinolysis	Hypocoagulable state			

aPTT, activated partial thromboplastin time; INR, international normalized ratio; na, not done/not available; PT, prothrombin time; TEG, hromboelastography; 20WBCT, 20-min whole blood clotting test.

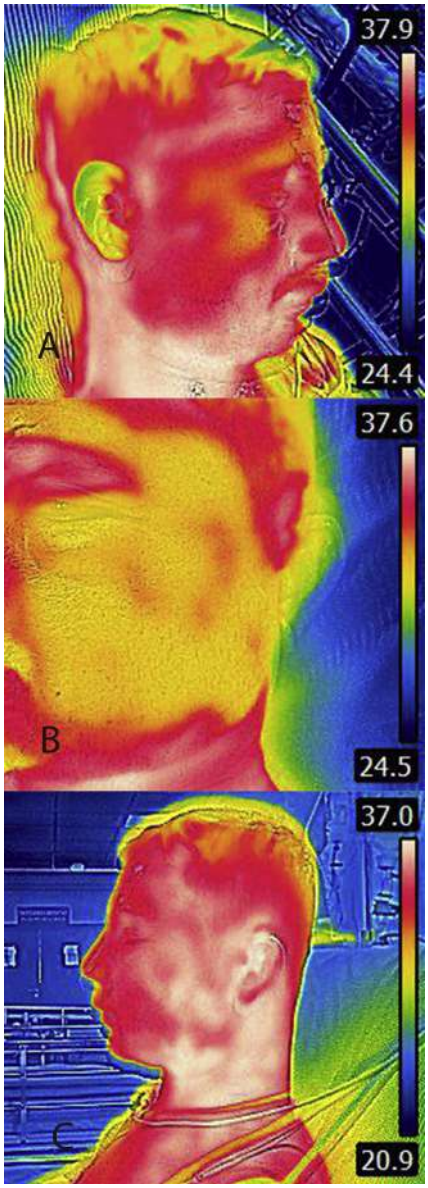


Figure 4. (A) Image of the opposite side of the face at the time of presentation. (B) Thermographic image of the bitten site showing lower temperature at the edematous area and higher temperature in the surrounding areas. (C) Image of the bitten side of face showing decrease in edema and swelling after ASV.

without hyper fibrinolysis (Figure 3B). He was also hypotensive with a blood pressure of 70/50 mm Hg. Ten vials of ASV (Bharath serums) were administered and a fluid bolus was given, to which he was found to be responsive. Intravenous amoxicillin-clavulanic acid, trypsin chymotrypsin, and paracetamol were given. A serial decline in platelets and hemoglobin was managed conservatively (Table 1). The elevated creatine kinase levels, serial serum creatinine, and urine analysis did not confirm renal injury. A peripheral smear done on Day 1 showed normal red blood cell morphology. He maintained normal urine output throughout the hospital stay. Serial PT-INR tests showed an improvement over the next 4 d (Table 2). Serial infrared thermography indicated a lower body surface temperature of the bitten cheek as compared to the left cheek and a normal temperature bilaterally on Day 3 (Figure 4). He did not develop any bleeding manifestations, renal injury, or airway compromise. He improved and was discharged on Day 4.

Discussion

The Western Ghats of India, one of 10 biodiversity hotspots, span over 1600 km, from Gujarat to Kerala, with an average elevation of 1200 m and the highest peak being 2695 m above mean sea level. The area is host to a wide range of endemic species.[6] The MPV is found in Maharashtra through Goa, Karnataka, and Kerala. It is arboreal, well camouflaged, and preys on amphibians and small reptiles.[4,7] It is locally known as *happatte havu.* These bites are often unreported owing to the perception of their venom as being nonlethal.

Our patient was engaged in agricultural activity when he was bitten, after which he developed local edema that spread over the bitten half of his face and neck, impairing eye opening. In this situation, a potential airway compromise was anticipated; however, he maintained his airway and the edema subsided. Chymotrypsin and topical glycerin magnesium sulphate were applied, but it remains unclear as to how much benefit they offered in

resolution of symptoms. Venom ophthalmia is caused by ocular contamination with venom (eg, by spitting cobras),[8] but facial bites by other venomous snakes may cause severe local envenomation and may secondarily compromise vision.[9] There was no evidence of venom ophthalmia in this patient.

Embedded fang fragments may be retained and result in infection or ulceration and hence should be looked for using x-ray or ultrasound.[10] In this case, ultrasound did not show evidence of the embedded fang.

Envenomation is mediated by phosphodiesterases, metalloproteinases, L-amino acid oxidases, and cysteine rich proteins, which may cause myonecrosis, hemolysis, platelet dysfunction, endothelial damage, and ion channel blockage.[11,12] This patient's creatine phosphokinase, renal function, and electrolytes were monitored; creatine phosphokinase was elevated. His hemoglobin dropped from 14 $g{\cdot}dL^{-1}$ on Day 1 to 11 $g{\cdot}dL^{-1}$ on Day 4, but there was no apparent bleeding or hemolysis, as evidenced by the work-up. The only attributable causes were blood sampling for diagnostic testing, rehydration, and mild extravasation at the bite site. MPV venom, as compared to *Naja naja* and *Daboia russelli*, can cause severe local tissue degeneration.[12] Ion channel dysfunction and myonecrosis may cause electrolyte imbalances.

Infrared thermography has been studied and used as a diagnostic and monitoring tool in many situations,[13,14] including local envenomation. The device used was an FLIR E8 camera with an image resolution of 320×240 pixels and a thermal sensitivity of <0.05°C (0.09°F)×<50 mK. Although there was a significant difference in temperature between the normal and the affected side, the temperature was actually lower around the bite site, which is attributed to the edema fluid and low ambient temperature.

The mild coagulopathy caused by MPV venom is primarily fibrinolytic, mediated by phosphodiesterases, metalloproteinases, and serine-protease like proteins such as malabarase.[15] Although the patient had a positive 20 minute whole blood clotting test (MWBCT), the INR was only marginally elevated and TEG showed a mildly hypocoaguable state.[16] MPV venom has potent thrombin-like enzyme (TLE).[11] TLE acts mostly on the alpha and to a lesser extent on the beta domain of fibrinogen, leading to consumption of fibrinogen without fibrin production. The fibrinogen consumption was evident from the first TEG; the graph showed evidence of ongoing clot lysis and decreased clot strength with normal clot initiation (normal R time). TEG assesses the coagulation cascade in clot initiation (R time), clot kinetics (K time and alpha angle), clot dynamics (maximum amplitude [MA], G and coagulation index), and clot resolution (Ly 30) and hence provides a clear picture of the dynamics of in vivo clot formation, unlike PT or activated partial thromboplastin time (aPTT), which are static tests. The patient had signs of coagulopathy at the time of admission (Figure 3A), which later completely evolved into a severe hypocoaguable tracing in TEG because of the fibrinogen consumption by TLE (Figure 3b). The fibrinogen-fibrin-platelet crosslinking is traced by the parameters K time and alpha angle, whereas 80% of MA is contributed by platelets and the remainder by fibrinogen.[17]

K time and angle were in the normal or low normal range in TEG (3.4 and 51.7 min) and MA was slightly reduced (47.7 mm) at the time of admission, with a fibrinolysis value of (Ly30−45%) indicating fibrinogen consumption due to venom-induced consumptive coagulopathy (Figure 3a). The repeat TEG had revealed more evolved coagulopathy in terms of unrecorded K time and a severely reduced angle (16°) as well as MA (9.8 mm) without evidence of ongoing clot resolution, indicating that venom-induced consumptive coagulopathy had completely evolved by that time (Figure 3b).

A normal R time in TEG meant the clot initiation was normal, which was also evident from a normal PT and aPTT on admission. The hyperfibrinolysis could be attributed to the defibrination by the TLE. Coagulopathy by TLE affects fibrinogen; the early coagulation screen by PT or aPTT may be normal and might lead to unwarranted consumptive coagulopathy if left to evolve fully. The authors also believe that measuring fibrinogen levels in *Trimeresurus malabaricus* bites might prove beneficial in establishing early consumption, where viscoelastic tests are not available considering the Indian landscape and resources.[18]

ASV was initially withheld but, owing to the deranged coagulation profile the next day, 10 vials of Indian polyvalent ASV (Bharat Serums, Navi Mumbai, India) were administered. The ASV as per the manufacturer is only effective against the "big 4," but there is some evidence of paraspecificity (Haffkine's,[19] Virchow, and VINS[11]). In vitro studies show the EC50 of ASV against MPV is significantly higher.[11]

The patient was hypotensive on the first day but gradually improved on the second day. The 5' nucleotidase in the venom is a hypotensive agent.[11] As analgesia, nonsteroidal anti-inflammatory drugs such as diclofenac are often used, but caution is recommended because they are associated with platelet dysfunction, anaphylaxis, and renal injury.[20]

Although species identification is important, it may also lead to complacency. It is therefore important to identify and recognize the possibility of life-threatening envenomation in pit vipers. *Trimeresurus malabaricus* also presents a challenge in identification owing to its shared habitat with other *Trimeresurus* species and its

varied morphologic features (at least 6 known color morphs). Syndromic identification is of limited value; therefore, there is a need for rapid diagnostic kits and species-specific/region-specific/pan-specific ASV.

Conclusions

We recommend that all *Trimeresurus* envenomations be considered medically significant. Thromboelastography is a useful tool for detecting and monitoring coagulopathy. The time to onset of systemic envenomation requires study, and the recommended period of observation should be modified accordingly. The role of Indian polyvalent ASV in these cases requires further in vitro and in vivo studies. Species identification is imperative for recognition of the burden and appropriate management of these cases. Research must focus on rapid diagnostic kits such as Dot-enzyme linked immunosorbent assay (DOT ELISA),[21] and species-/pan-specific antivenom needs to be developed as early as possible.

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Author Contributions: Original draft of manuscript, imaging of snake and bite site, identification of snake (FMS); drafting of manuscript, reviewing, and editing (VL); IR imaging (CAS); TEG analysis (GM); critical review of manuscript (SS); approval of the final manuscript (all authors).

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CASE REPORT

Poisoning After Ingestion of *Mucuna pruriens* Seeds on Reunion Island

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Mucuna pruriens seeds contain levodopa, a dopamine precursor that composes the standard treatment to manage symptoms of Parkinson's disease. Also known as velvet bean, this plant is often involved in cases of intense pruritus after contact with the hair that grows on its beans. This case report describes the effects in a 58-y-old woman after ingestion of 5 raw seeds of *M pruriens* on Reunion Island, a French tropical island territory close to Mauritius, in the Indian Ocean. About 40 min after ingesting the plant, the patient showed adverse digestive symptoms such as nausea, vomiting, and abundant diarrhea followed by a state of confusion, hallucinations, and amnesia. She was brought to the emergency department. After medical examination, her vitals were normal, and only her nausea lingered. The poison control center was contacted by the medical team. The plant, previously well described and known by the patient, was later picked from the garden and reviewed by the Indian Ocean toxicovigilance department. Expert botanists confirmed the plant species to be *M pruriens*. The patient ate the raw seeds after reading articles on the Internet, without confirming whether the seeds should have been cooked before consumption. This case highlights that ingesting raw, unprepared *M pruriens* seeds can cause severe digestive symptoms, possibly accompanied by neurologic disorders.

Keywords: intoxication, plant, velvet bean, Indian Ocean, levodopa, fabaceae

Introduction

Mucuna pruriens is a leguminous plant belonging to the Fabaceae family and is commonly known as velvet bean or cowhage in English and *poils à grater* in French. It is an annual climbing plant that can reach 15 m in length and is widespread in tropical and subtropical regions of the world, such as Reunion Island.

Each 10 to 13 cm pod (Figure 1) is covered by a multitude of stinging hairs when young (less when older) that cause intensely itchy dermatitis after cutaneous contact. The symptom is caused by a histamine-independent itch mediator, mucunain.[1-3] Pods are longitudinal and curved and contain around 4 to 6 seeds each (Figure 2). Seeds contain levodopa, a nonprotein phenolic amino acid that is used as the standard treatment to manage symptoms of Parkinson's disease.[4,5]

Acute poisoning by plant ingestion in humans is rarely described in the literature but can occur, as our case illustrates.

Case Description

A 58-y-old woman with no medical history was driven by her relative to the emergency room with severe vomiting, profuse episodes of diarrhea, mental confusion, a hallucinatory episode, and anterograde amnesia.

Digestive symptoms, quickly followed by neurologic disorders, appeared approximately 40 min after ingestion of 5 freshly picked seeds of *M pruriens*, which the patient meticulously chewed (Figure 2). She was driven by her relative to the emergency room 2 h after the ingestion. The patient's vitals were recorded as blood pressure 160/80 mm Hg, heart rate 63 beats·min^{-1}, a percutaneous oxygen saturation of 99% on room air, and blood sugar 7.9 mmol·L^{-1} (normal range before meal 4.0–7.0 mmol·L^{-1}). Her weight was 56 kg (body mass index 24.9 kg·m^{-2}).

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Figure 1. *Mucuna pruriens* pods. Reunion Island. 2020. Photo by A. Maillot.

A neurologic examination showed a conscious woman with a Glasgow Coma Scale score of 15. She was still nauseated without vomiting on observation. Auscultation was normal. The emergency physician called the poison control center for an expert opinion. Owing to levodopa present in seeds, the toxicologist advised clinical monitoring until the patient became completely asymptomatic, which occurred 3 h after admission. Given her rapidly improving condition, electrocardiogram and blood tests were not performed. She was discharged at the end of the clinical monitoring.

Subsequently, the referring medical toxicologist asked the Indian Ocean toxicovigilance department located on the island to fully investigate the intoxication. One week after the event, the patient was only slightly tired. Some pictures and a piece of the plant that was growing in her garden were directly collected on site the same day we visited her, with the patient's consent. The patient knew how to recognize this plant because it had been growing in her garden since childhood, and she had been told about the risk of pruritus after contact with the beans' hair.

After submission of the photos and sample to a group of French botanical experts working with French poison control centers, *M pruriens* was clearly identified. The patient was questioned regarding the circumstance of exposure, the involved agents, and her symptoms, and all her responses were consistent with the emergency department record. We learned that the patient was tempted to taste the plant, as it was growing in her garden, after reading web pages about *M pruriens* seeds and their alleged benefits in protein and memory boost. She told us the seeds were hard to chew and had an astringent, unpleasant taste.

Discussion

The genus *Mucuna* includes about 150 species that grow easily in a warm and humid climate; thus, Reunion Island is an optimal place with its subtropical climate. In a traditional medicine context, *M pruriens* has many uses around the world, such as in Ayurvedic medicine in India.[6,7] *M pruriens* seed is rich in crude protein, more than other classic legumes.[8,9]

The Internet offers access to a plethora of information. In our case, the patient did not follow the recommended process for removing antinutritional factors that are usually found in raw legumes. Antinutritional factors are defined by chemical compounds synthesized in plants that interfere with the absorption of nutrients (eg, proteins, vitamins, and minerals).[10,11] These chemical compounds compromise digestion and cause adverse events that depend on the part and quantity of the plant ingested. Some animal species (ruminants) are more likely to digest certain raw plants without showing serious adverse reactions owing to their capacity to digest antinutritional factors. This is not the case for humans and this particular plant. The main antinutritional factors in *M pruriens*

Figure 2. *Mucuna pruriens* seeds. Reunion Island. 2020. Photo by A. Maillot.

include tannin, trypsin inhibitors, soponins, and alkaloids. The patient failed to note that although the plant is widely recognized and used around the world, the quantity of levodopa and other toxic antinutritional factors must be reduced by boiling before ingestion because they are water soluble. For example, soaking a raw seed of *M pruriens* for 15 h and boiling for 90 min can reduce the levodopa concentration by 70% compared to the dried seed.[12]

The ingestion of crude *M pruriens* seeds can lead to digestive and neurologic disorders as described in this case report. These symptoms can be induced by levodopa; the same symptoms are currently noted with drugs used to treat Parkinson's disease.[13-16] Digestive disorders can also be induced by other antinutritional toxins, such as tannins and saponins. In 1989, an outbreak of acute poisoning after *M pruriens* ingestion in Mozambique was reported.[17] Water shortage and famine were at the origin of toxic psychosis. Levodopa toxicity was also described in a 27-y-old woman in 2013 after ingestion of *M gigantea*.[18]

Without an antidote, in the event of levodopa overdose, health care remains supportive. Because *M pruriens* seeds contain many antinutritional factors and an estimated concentration of levodopa around 5%,[12] blood pressure should be monitored for a few hours because hypertension or hypotension can occur with levodopa overdose.[14]

One of the major difficulties in toxicology is identification and evaluation of the agent causing the intoxication, especially with plant poisoning. In this particular case, determining the cause of the event was possible owing to available information such as the time of onset of the adverse event combined with suggestive semiotics, pictures of the toxic agent from the patient, and access to the actual plant for later confirmation.

Conclusions

We report a case of moderate poisoning after chewing and ingestion of raw *M pruriens* seeds on Reunion Island in April 2020. Fortunately, even with intense digestive and significant neurologic adverse effects, the intoxication was not fatal. However, blood pressure should be monitored for a few hours given the risk of hemodynamic instability caused by levodopa. It seems useful to keep in mind that raw seeds should be kept out of reach of children and persons with impaired judgment when *M pruriens* is commonly found in fallow fields or less often in private gardens on Reunion Island. This case illustrates, once again, that "natural" does not always mean "safe."

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CASE REPORT

Unusual Sting by a Nonindigenous Caterpillar in Europe

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On the French island of Corsica, a 57-y-old woman without significant medical history was stung on the left thumb while she was taking care of an ornamental *Ficus benjamina* plant. Immediately, she felt intense pain in her hand. She saw a strange caterpillar, later identified by the local poison center as *Acharia stimulea*. The pain in her hand was evaluated as 8 of 10 using the numerical pain rating scale; only a slight erythema was visible on her skin. Symptoms disappeared within 2 h with use of local anti-inflammatory ointment and oral painkillers. Three other caterpillars emerged out of the soil of the potted plant during the following week. This sting by a saddleback caterpillar is exceptional in Corsica. French garden store owners and healthcare professionals should be informed that caterpillars can be imported across the oceans to Europe on different plants.

Keywords: envenomation, toxinology, *Ficus benjamina*, *Acharia stimulea*, saddleback caterpillar, Corsica

Introduction

Caterpillars are butterfly and moth larvae, and several species are equipped with venomous chitinous spines that are capable of penetrating human skin and causing envenomation.[1] Several species of stinging caterpillars are found in the southeastern United States, but the most notorious are the saddleback caterpillar *(Acharia stimulea*) and the puss caterpillar (*Megalopyge* spp).[2] Both have been responsible for clinical descriptions of intense pain with proximal radiation.[3]

The saddleback caterpillar, *A stimulea* (formerly *Sibine stimulea*), has a striking appearance (Figure 1). It belongs to the order of Lepidoptera (butterflies and moths) and the family of Limacodidae (slug-like caterpillars). Larvae are slug-shaped and approximately 2.5 cm (1 in) long and 1 cm (0.4 in) wide when fully grown. They are brown at both ends, with hollow spine-covered tubercles (horns), a green "saddle blanket" middle trimmed with white, and a central purple-brown oval spot like a saddle also edged with white, hence the common name.[1,4,5]

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The saddleback caterpillar is a passive, slow-moving caterpillar that usually remains hidden in foliage.[5] It has tufts of true urticating spines lined with hypodermal cells that secrete toxins, the chemical composition of which remains obscure.[2] Stings have been very rarely reported in European countries because the caterpillar is not native to Europe. We describe an observation after skin contact with an *A stimulea* caterpillar imported to the French island of Corsica on a *Ficus benjamina* plant.

Case Report

A 57-y-old woman without significant medical history was stung on the left thumb while she was taking care of her ornamental plants. Immediately, she felt intense pain in her hand. She saw a strange caterpillar (Figure 1) on a leaf of the exotic *F benjamina* she bought 1 mo earlier in a local garden center in Corsica, France. The plant was still in its original pot.

The sting caused localized pain that radiated to the whole arm, causing skin pallor and generalized discomfort 3 min after the event. She decided to call the emergency department, which contacted the local poison center. The poison center identified the caterpillar as a saddleback caterpillar, *A stimulea* (Figure 1).

Figure 1. *Acharia stimulea* on a *Ficus benjamina* leaf (Torrents).

The pain in her hand was evaluated as 8 of 10 using the numerical pain rating scale; no sting mark was visible on her skin, only a slight local erythema with no edema. Local anti-inflammatory ointment (diclofenac, topical 2%) and oral painkillers (paracetamol 400 mg plus codeine 20 mg) were prescribed with home monitoring. Symptoms resolved within 2 h. According to the patient, 3 more caterpillars of the same species emerged from the soil of the potted plant during the following week. The patient presented no sequelae. Investigation showed that this *Ficus* came from Mexico where *A stimulea* are indigenous, and it was the only case reported from the garden center in Corsica.

Discussion

In a 1-y prospective study of 112 caterpillar envenomations reported in 1990, the caterpillars involved could only be identified in 68% of cases.[6] The authors were from the southeastern United States, which caused bias in the caterpillars involved. In the study, 4 caterpillar species were strongly associated with stings: the buck moth caterpillar (*Hemileuca maia*), the Io moth caterpillar (*Automeris io*), the woolly asp/southern flannel moth caterpillar (*Megalopyge opercularis/Lagoa crispata*), and the saddleback caterpillar.[4,6] *A stimulea* stings are very rare in Europe because the species is not indigenous. One pediatric case was described in southwest France from an imported *Areca* tree, and 2 cases were reported in the United Kingdom: 1 in Wales, from a caterpillar nestling in an ornamental plant, and the other at a garden center in Somerset. In all of these European cases, the caterpillars arrived after being transported across the Atlantic Ocean on imported plants.[5]

In our case, only 1 sting was reported, but the saddleback caterpillar is able to cause multiple stings on the same patient.[2] Cases of ingestion or oral contact have also been reported.[7,8] The most common symptoms associated with this caterpillar's sting are localized pain, erythema, and swelling. Edema is often described with normal vital signs and no fever.[2,5] These symptoms are not always reported. For our patient, nothing was noted except slight erythema. The lack of urticarial lesions with *A stimulea* is known and is similar to other species.[4] However, an urticarial reaction that cleared completely in about 2 h has been reported; 71 h after the caterpillar sting, the same patient presented an acute vesicular eruption in the sting area, which cleared promptly with the use of a topical corticosteroid gel.[2] Biopsy of the urticarial lesion was performed and showed an unremarkable epidermis with marked reticular dermal edema and a sparse superficial perivascular lymphohistiocytic infiltrate.[2] Contact urticaria or, more rarely, an acute eczematous reaction is also possible.[2] The classical clinical presentation of such incidents shows a skin reaction in the shape of an erythematous halo that can become hemorrhagic and vesicular. In our case, no biopsy was performed owing to the lack of local signs. Excruciating pain is characteristic of each envenomation, evaluated at 8 of 10 using the numerical pain rating scale in this case. This pain may be accompanied by systemic manifestations such as skin pallor and discomfort owing to the pain intensity, but generally symptoms resolve within a few hours.

The general management of this caterpillar sting is entirely supportive. It includes immediate soap-and-water washing of the sting site and application of ice packs to reduce the stinging sensation.[5] Spines should be removed with adhesive tape or blackhead-removing pads,[4] and administration of antihistamines may help relieve itching and burning.[5] Topical corticosteroids may reduce the intensity of the inflammatory reaction,[2] and appropriate analgesics should be prescribed for pain. This treatment may be combined with local anesthesia in the emergency department.[9]

This clinical case describes an exceptional sting by a nonindigenous saddleback caterpillar in Corsica. Caterpillars such as *A stimulea* can be imported across oceans on plants such as *Areca* or *Ficus* trees. Garden store owners and health care professionals should be informed about this risk.

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CASE REPORT

Nutritional Strategies of an Athlete with Type 1 Diabetes Mellitus During a 217-km Ultramarathon

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Considering the challenges in meeting the high nutritional demand during ultramarathons, the aim of this study was to analyze the nutritional strategies and glycemic response of an athlete with type 1 diabetes (DM1) during participation in a 217-km ultramarathon. A 36-y-old male athlete who was diagnosed with DM1 15 y earlier was studied during participation in the Brazil 135 ultramarathon. Food consumption and blood glucose were recorded during the race, and nutritional intake was calculated after the race. The athlete completed the race in 51 h 18 min. He consumed a total of 15.0 MJ (3593 kcal), 532 g carbohydrate, 166 g protein, 92 g lipid, and 14 L of water during the race. Glycemic values ranged from 3.6 to 18.2 mmol·L^{-1}. Most glycemic values (47%) ranged from 3.9 to 10 mmol·L^{-1}, whereas 5% were <3.9 mmol·L^{-1}, 16% were >10 to 13.9 mmol·L^{-1}, and 32% were >13.9 mmol·L^{-1}. This case report describes the dietary profile of an athlete with DM1 during a 217-km ultramarathon. Although the athlete implemented strategies that differed from those recommended in the literature, food and nutrient intake and the glycemic management strategy adopted allowed him to successfully finish the race. These results suggest that past personal experiences can be considered and that nutritional recommendations for athletes with DM1 should be individualized.

Keywords: ultraendurance, nutrition, personal experiences, glycemic response, sleep

Introduction

Ultraendurance events have become more popular, and participation in ultramarathons has increased worldwide.[1] Driven by the motivation to reach new achievements and limits, ultramarathons have been undertaken by many individuals, including those with type 1 diabetes mellitus.[2] Current exercise management guidelines provide pragmatic recommendations for individuals with type 1 diabetes, such as adjusting insulin doses and increasing carbohydrate intake before, during, or after exercise.[3]

Although maintenance of on-target blood glucose levels is challenging for individuals with type 1 diabetes, with adequate strategies of exogenous insulin administration and nutritional intake, it is possible to manage glucose homeostasis and obtain good exercise performance.[4] In addition to daily challenges, maintaining normal glucose levels during different training protocols, especially during prolonged exercise events[4] and traveling, may present a major challenge for athletes with type 1 diabetes,[5] considering that factors other than insulin levels have an impact on glycemia, such as macronutrient intake and psychophysiological stress responses.[4]

Some athletes with type 1 diabetes ingest low to moderate levels of carbohydrates during training and rest periods[4,6,7]; however, this strategy becomes inefficient during long-distance events when high levels of carbohydrate (70–90 g·h^{-1}) are ingested, aiming to prevent hypoglycemia and support performance.[4] When carbohydrate is consumed during exercise, there is an alteration of substrate utilization to energy provision, with a lower contribution of endogenous carbohydrate and a higher rate of exogenous carbohydrate oxidation.[8]

Moreover, other macronutrients can also affect blood glucose.[9,10] For example, protein intake can also lead to hyperglycemia delayed by gluconeogenesis, together with increased secretion of glucagon.[10] Considering this sustained postprandial hyperglycemic effect of this macronutrient, protein intake associated with carbohydrate after exercise has been considered an interesting strategy for muscle glycogen replacement associated

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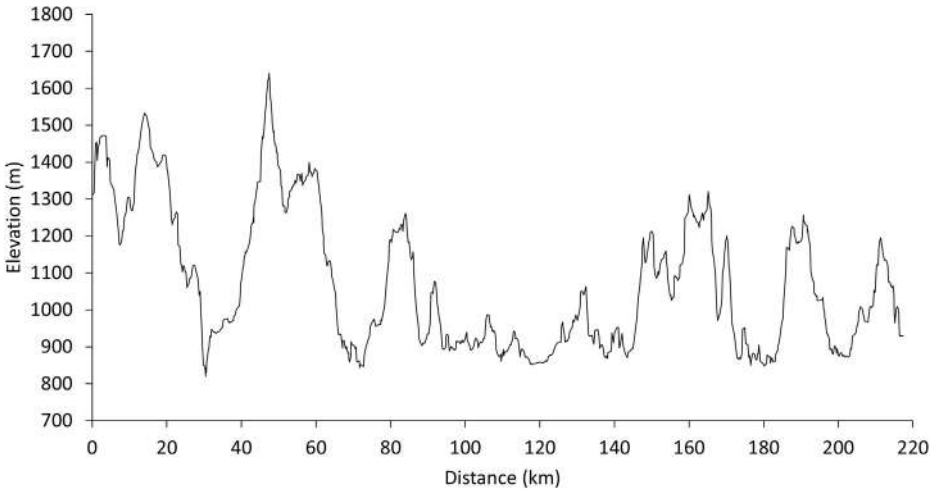

Figure 1. Elevation profile throughout the Brazil 135 Ultramarathon route.

with blood glucose homeostasis.[11] In addition, dietary fat can also affect blood glucose[9,10]; it contributes to initial hypoglycemia owing to a delay in gastric emptying, and to later hyperglycemia induced by impaired insulin sensitivity and increased hepatic glucose production.[9,10]

Although the number of athletes with type 1 diabetes who complete ultramarathon races has increased,[4] knowledge about the dietary pattern of these athletes during competition is scarce in the literature.[12] This study aims to analyze the nutritional strategies and glycemic responses of an athlete with type 1 diabetes during participation in a 217-km ultramarathon.

Case Report

A 36-y-old male athlete who was diagnosed with type 1 diabetes 15 y earlier and had 14 y of running training, including participation in 38 marathons (best marathon time: 3 h 38 min) and 4 ultramarathons (100−217 km), was studied during participation in the Brazil 135 Ultramarathon. This athlete was considered well trained for this sport, having performed a weekly training regimen of 40 to 100 km. He had a body mass of 71.1 kg, height of 1.66 m, and a body fat percentage of 10%. Body mass and height measurements were performed with the participant barefooted and wearing light clothing, using a platform scale (Welmy, Sao Paulo, Brazil) to a precision of 0.1 kg and 0.5 cm, respectively. Skinfold thickness (chest, abdominal, and thigh) was measured using a caliper (Sanny, Sao Paulo, Brazil) to a precision of 1 mm, and body fat percentage was calculated by means of standard equations.[13,14] The study was approved by the research ethics committee of São Paulo State University (no. 037/2008), and the athlete gave written consent to participate.

In this race, runners cover 217 km and arrive at the finish line within 60 h (Figure 1). During the race, athletes can eat and drink ad libitum. To evaluate food consumption during the ultramarathon, the participant completed a form prepared by the researchers that recorded the time, food consumed, and quantity. With these data, the amounts of energy, carbohydrate, lipid, and protein consumed during the race could be calculated using Avanutri software (version 3.1.1) or from the nutritional information on the product labels.

Records of blood glucose levels (Accu-Chek 360 glucose meter, Roche Diagnostics, Sao Paulo, Brazil) and insulin administration were recorded by the athlete during the race. Repeat measures were made of body mass at 78, 175, and 217 km to evaluate hydration status. The athlete was free to sleep whenever he wanted.

Records of split and overall race times were obtained via the official race reports. Thus, we calculated split and overall velocities of the athlete. Moreover, critical velocity was determined 1 wk before the competition.[2] This exercise intensity occurs approximately midway between the anaerobic threshold and maximal oxygen uptake.[15]

Table 1 describes the type and amount of food consumed, in addition to the calorie and macronutrient intake, according to the specific race time. During the competition, 14 L (263 $mL \cdot h^{-1}$) of water was consumed.

As shown in Figure 2, the athlete's blood glucose ranged from 3.6 to 18.2 $mmol \cdot L^{-1}$ during the race. Most glycemic

Table 1. Food and nutrient consumption during the race

Race day	*Race time (h)*	*Food*	*Amount*	*Energy (kcal)*	*Carbohydrate (g)*	*Protein (g)*	*Fat (g)*
	2	Propel Hydractive	500 mL	7	2	–	–
1	3	Mashed potatoes	20 g	24	4	0.9	0.4
		Regular Coke	300 mL	128	32	–	–
	4	Energy gel Carb'Up	30 g	80	20	–	–
		Jelly gummy	40 g	144	36	–	–
		Gatorade	500 mL	120	30	–	–
	6	White wheat light bread	26 g	94	19	4	0.1
		Salami	20 g	60	0.0	5	5
		Regular Coke	300 mL	128	32	–	–
	8	Ruffles potato chips	65 g	367	31	4	25
		Regular Coke	200 mL	85	21	–	–
		Jelly gummy	40 g	144	36	–	–
	10	Energy gel Carb'Up	30 g	80	20	–	–
		Sports Beans	28 g	100	25	–	–
	11	Spaghetti bolognaise	45 g	109	12	4	5
		Regular Coke	200 mL	85	21	–	–
	14	Energy gel Carb'Up	30 g	80	20	–	–
		Protein bar—VO2 chocolate	30 g	122	11	16	3
Total D1 (g)				1957	372	33	38
Total D1 (g·kg^{-1})				28	5	0.5	0.5
	18	Spaghetti with garlic and oil	30 g	66	10	2	2
2		Chicken with tomato sauce	280 g	356	0.0	62	12
	25	Propel Hydractive	500 mL	0	2	–	–
	27	Protein bar—VO2 chocolate	30 g	122	11	16	3
	29	Roll	50 g	135	29	5	0.1
		Salami	20 g	60	0.0	5	5
		Apple	150 g	95	21	0.6	0.8
		Regular Coke	200 mL	85	21	–	–
	32	Roll	50 g	135	29	5	0.1
		Salami	20 g	60	0.0	5	5
	37	Canned tuna fish	120 g	233	0.0	29	13
Total D2 (g)				1347	123	130	40
Total D2 (g·kg^{-1})				19	2	2	0.6
	40	Cream cracker	25 g	108	17	3	3
3		Mayonnaise	15 g	100	0.1	0.3	11
	46	Energy gel Carb'Up	30 g	80	20	0.0	0.0
Total D3 (g)				288	37	3	14
Total D3 (g·kg^{-1})				4	0.5	0.0	0.2
Total (g)				3592	532	166	92
Total (g·kg^{-1})				50	7	2	1
Total (g·kg^{-1}·h^{-1})				1	0.1	0.0	0.0

D1, day 1; D2, day 2; D3, day 3.

values (47%) ranged from 3.9 to 10 mmol·L^{-1}, whereas 5% were <3.9 mmol·L^{-1}, 16% were >10 to 13.9 mmol·L^{-1}, and 32% were >13.9 mmol·L^{-1}. The athlete took a total of 10 U basal insulin (4 U at 2 h postrace and 6 U at 6 h postrace) plus 40 U of bolus insulin (10 U before the race, 10 U at 116 km, and 20 U at 2 h postrace) via insulin pen.

Sixty-two athletes participated in the race. Forty-four runners (66% of participants), including our volunteer, completed the competition. Their race time ranged from 27 h and 59 min to 59 h and 23 min. The studied athlete completed the race in 51 h and 18 min (mean of 4.2 km·h^{-1}, 33% of critical velocity) in 27th place. Figure 2 also

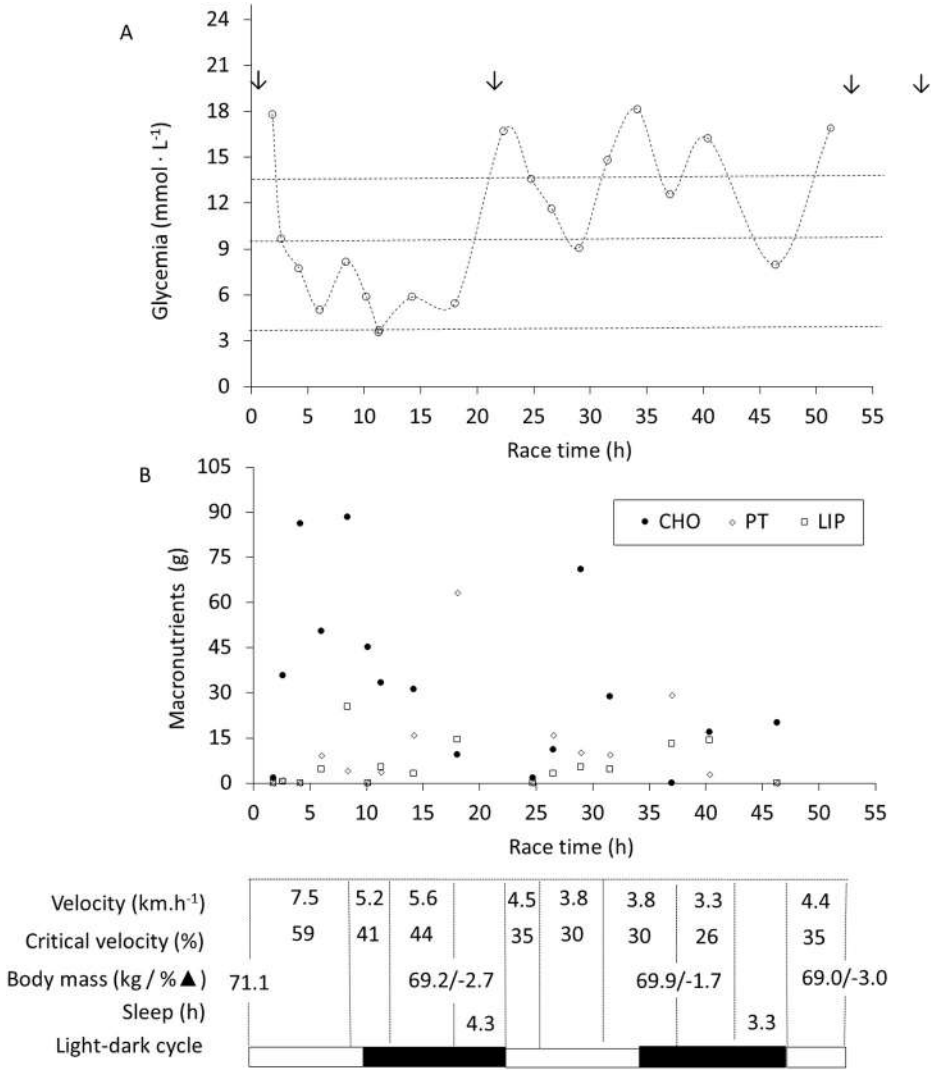

Figure 2. Glycemic response and macronutrients consumed during the race. Dotted lines delimit the blood glucose targets proposed for athletes with type 1 diabetes mellitus during the competition[4]: <3.9 mmol·L^{-1}, 3.9 to 10 mmol·L^{-1}, >10 to 13.9 mmol·L^{-1}, and >13.9 mmol·L^{-1}. Arrows represents bolus insulin infusion. BM, body mass; CHO, carbohydrate; LIP, fat; PT, protein.

presents the split velocities, hydration status, and sleep periods of the athlete throughout the race.

Discussion

This case report describes the dietary profile of an athlete with type 1 diabetes mellitus during a 217-km ultramarathon, with a detailed description of the food and drink ingested. Only one other known study[16] has described examples of foods consumed by athletes during an ultramarathon; however, these authors evaluated healthy runners and only reported the usual foods, not how they were eaten during the competition.

The athlete's nutritional strategies were compared to guidelines for ultraendurance sports,[16] and we can note that our athlete had insufficient energy, carbohydrate, and protein intake. The athlete in this study had nutritional intake similar to that of other triathletes who participated in a competition that lasted an average of 12 h,[17] less than a quarter of the time our athlete took to complete the ultramarathon, suggesting that there is great difficulty in consuming the recommended amounts during the race and in guaranteeing an energy intake compatible with the energy expenditure of the activity. The challenge observed is not only common for people with type 1 diabetes mellitus, but also for ultramarathon athletes in general.[16]

Although total energy expenditure (TEE) was not evaluated in this study, authors who evaluated energy demands using doubly labeled water observed that ultramarathon runners have a high energy expenditure in the event,[18] reaching more than 8 times resting metabolism.[18] A study that evaluated athletes during a 161-km ultramarathon (Western States 100, with 25.9±3.0 h [mean±SD] to complete the race), observed that the TEE was 7040±1360 kcal per 12 h, or 587 kcal·h^{-1}.[18] The literature describes that TEE may vary according to several factors, such as body composition, exercise, and environmental conditions,[16] but suggests values ranging from 48 to 72 kcal·km^{-1} during long races.[16,19] According to these data, it is possible to note that the athlete in our study had an energy consumption far below the TEE estimated in the literature (17 kcal·km^{-1} and 70 kcal·h^{-1}), reinforcing the difficulty of meeting caloric demands compatible with the high demand of the ultramarathon.

By specifically studying athletes with type 1 diabetes mellitus, authors[12] have reported carbohydrate intake rates of 0.4 to 0.6 g carbohydrate·kg^{-1} body mass·h^{-1} of exercise in an ultramarathon lasting more than 13 h, which was higher than that consumed by our volunteer.

Fluid consumption was also lower than that recommended for ultraendurance athletes[16] and for athletes with type 1 diabetes mellitus[4] and was below the water intake reported from stable isotope methodologies.[18] Despite this, the variation of −3% body mass observed during the race is compatible with the practical recommendations,[20] which suggest that body mass losses over 2% may be natural and necessary during ultraendurance events to avoid hyperhydration.

The literature[4] proposes glycemic targets for athletes with type 1 diabetes mellitus during competitions. Our results indicate that the athlete spent less time within the range of 3.9 to 10 mmol·L^{-1} (43%) and more time with his blood glucose above 13.9 mmol·L^{-1} (32%) than recommended (75 and 5%, respectively). We suspect that the athlete chose to spend more time in the highest targets of glycemia to avoid the negative consequences of hypoglycemia (<3.9 mmol·L^{-1}), which only occurred in 1 glycemia measurement during the competition (~11 h of race time, at 78 km).

The practice of reducing insulin and consuming additional carbohydrates to avoid hypoglycemia and enhance endurance performance in prolonged exercise has been observed in other athletes with type 1 diabetes mellitus.[8] In addition to managing insulin levels, athletes can manage their blood glucose by manipulating their macronutrient intake.[4] We observed that the increase in blood glucose was caused not only by carbohydrate intake, but also protein consumption. The high amount of this nutrient (around 60 g) consumed at around 18 h of race time and consumption of almost 40 g of protein at 37 h probably resulted in significant and sustained postprandial hyperglycemia. Authors[10] have reported that for individuals with type 1 diabetes mellitus, the addition of ≥28 g of protein to a mixed meal or ingestion of ≥75 g of protein alone could result in a significant and sustained increase in blood glucose in the postprandial period (2−5 h). It is worth mentioning that an insulin application could be a useful tool for adjusting the effects of protein-rich foods.

The impact of fat on blood glucose has also been discussed in the literature,[9] which described no significant effects of high-fat meals in relation to postprandial glycemia, but reported a tendency toward an increase in the long term.[9] We observed no evidence of an impact of fat on postprandial glycemia throughout the race. However, studies indicate that high-fat meals require more insulin than meals with less fat and the same carbohydrate content, supporting the need for alternative insulin dosing algorithms for high-fat meals.[9,21]

Finally, the athlete slept at 2 time points (after 116 and 195 km, totaling 7.6 h), which occurred between 0300 and 0600 of the 2 race days. Thus, he slept at night, which is the normal time for rest in humans, considering the circadian pressure for sleep, which possibly contributed to the successful performance of the athlete.[22] This pattern has also been observed in other ultramarathon runners.[23]

The present case demonstrates that although the athlete with type 1 diabetes mellitus faced the physiological challenges of the ultramarathon and implemented strategies that differed from those recommended in the literature, the insulin management strategy, food and nutrient intake, and exercise and sleep distribution adopted allowed the athlete to successfully finish the race, suggesting that past personal experiences can be considered and that nutritional recommendations for athletes with type 1 diabetes mellitus should be individualized. Continuous glucose monitoring systems may provide a more detailed glycemic response throughout the race, which could constitute a possible limitation of the present investigation. Future studies should focus on this issue.

Author Contributions: Study concept and design (LMH, TB); acquisition of the data (LMH, TB); analysis of the data (NES, NVSD, LMH, AME, TB); drafting of the manuscript (NES, NVSD, AME, TB); critical revision of the manuscript (CAG, AME, TB); approval of final manuscript (all authors).

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REVIEW ARTICLE

Prehospital Use of Ultrathin Reflective Foils

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Ultrathin reflective foils (URFs) are widely used to protect patients from heat loss, but there is no clear evidence that they are effective. We review the physics of thermal insulation by URFs and discuss their clinical applications. A conventional view is that the high reflectivity of the metallic side of the URF is responsible for thermal protection. In most circumstances, the heat radiated from a well-clothed body is minimal and the reflecting properties of a URF are relatively insignificant. The reflection of radiant heat can be impaired by condensation and freezing of the moisture on the inner surface and by a tight fit of the URF against the outermost layer of insulation. The protection by thermal insulating materials depends mostly on the ability to trap air and increases with the number of covering layers. A URF as a single layer may be useful in low wind conditions and moderate ambient temperature, but in cold and windy conditions a URF probably best serves as a waterproof outer covering. When a URF is used to protect against hypothermia in a wilderness emergency, it does not matter whether the gold or silver side is facing outward.

Keywords: survival blankets, space blankets, heat exchange, hypothermia, insulation

Introduction

Ultrathin reflective foils (URFs), often referred to as "space blankets" or "survival blankets," were first designed and brought to market in the mid-1960s. Although they were primarily used in cryogenics and space applications, their potential usefulness as a medical product was rapidly recognized. URFs are widely used in emergency medicine in an attempt to protect patients from heat loss, and in operating rooms to prevent intraoperative hypothermia.[1-3] While trade names and descriptions of these foils suggest that they possess insulating properties, there is no evidence that they are effective. The only evidence in favor of their use is theoretical calculations and a few small clinical studies.[4-7] By the 1970s, the unreasonable enthusiasm diminished, but reflective foils are still common in first aid kits, rescue kits, and emergency medical equipment.[1,8] We discuss the uses and limitations of ultrathin reflective foils in prehospital medicine.

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Structure and Properties of URFs

The main component of a URF is a polyethylene terephthalate (PET or PETE). Some URFs use Mylar, a biaxially oriented PET which is thicker and stronger than standard PET. During the process of vacuum metallization, the foil is covered by a layer of aluminum, which confers reflective properties. Although the typical thickness is only about 10 to 15 micrometers (0.01–0.015 mm), the foil has a relatively high tensile strength, flame retardancy, and resistance to penetration by water vapor or air.[9,10] Some believe that high reflectivity of the metallized side, ranging from 90 to 97%, is responsible for the thermal resistance properties (the ability of a material to resist heat flow).[4,11] The other side can be coated in any

color, typically gold or orange for mountain rescue and media uses or olive-green for military use. In the 1970s, trials of metallized foils for rescue purposes were conducted based on the mistaken assumption that the reflective layer would reflect radio waves and could be detected by radar. Both theory and practice revealed the uselessness of foils in these sorts of applications.[9] More recently, it became apparent that reflection of visible light by URFs increases detectability in search and rescue missions performed when there is good visibility, while reflection of infrared radiation increases detectability when there is poor visibility.[11,12] Metallized foils transmit only 1 to 8% of visible light and about 1% of ultraviolet B radiation (280–315 nm). URFs can provide protection from snow blindness from high solar radiation.[12]

In addition to URFs, there are also so-called "heavy reflective blankets" available for everyday use. Unlike ultrathin foils, these are thicker (about 1 mm), more rigid, and stronger with a synthetic or natural nonwoven filling or with the reflective layer applied as 1 layer of a multilayer system. Advanced multilayer systems, with corrugated aluminum foil providing air insulation between individual layers, are also available. We do not discuss heavy reflective blankets further in this review.

Principles of Heat Exchange

According to the second law of thermodynamics, heat always flows from an object at a higher temperature to an object at a lower temperature. If there is a difference between the temperature of a given object and its surroundings or a neighboring object, heat exchange will take place until the temperatures reach equilibrium.[4] Heat exchange between neighboring objects can only be slowed, not eliminated. Heat is transferred by 4 mechanisms: convection, conduction, evaporation, and radiation (Figure 1).

All objects emit thermal radiation. The kinetic energy of heat is converted into electromagnetic radiation, at a wavelength that depends on the temperature of the object (Wien's law). Human bodies emit mostly infrared radiation (heat) with a wavelength of 5 to 20 micrometers.[13] Unlike convection and conduction, in which direct contact is necessary for heat transfer, radiation does not require the presence of any medium between objects that are exchanging heat. Radiative heat exchange can occur in a vacuum. Reflection can limit absorption of radiated heat, even in space.

The power of the radiation emitted by a body as electromagnetic waves is proportional to the surface area (S) and to the fourth power of surface temperature (T_{sur}) in degrees Kelvin:[4]

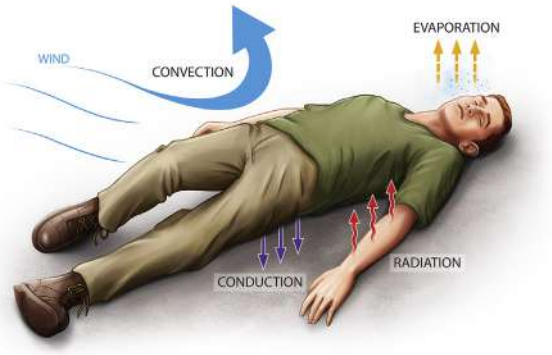

Figure 1. Mechanisms of heat loss. Authors' own graphic.

$$P_{rad} = \sigma \varepsilon S (T_{sur})^4$$

where σ is the Stefan-Boltzmann constant (5.67×10^{-8} $W/m^{-2}K^{-4}$) and ε is the emissivity of the surface, the ratio of the energy radiated from a material's surface to that radiated from a perfect emitter, known as a black body. A surface emissivity of 1 defines a perfect black body. The emissivity of human skin is very high (0.98). The emissivity of most fabrics is about 0.75.[13,14] Electromagnetic radiation can cause both heat loss and heat gain. Bodies can absorb heat from objects in the environment, such as the sun, a radiator, or a fire. Heat balance is the difference between heat that is dissipated and heat that is absorbed.

Maintenance of core temperature is critical for human physiology. The amount of heat loss depends partly on the temperature of the skin surface. The human body responds to cold by vasoconstriction of the skin, lowering surface temperature. Vasoconstriction can limit skin blood flow to less than 10% of baseline.[15] This defense mechanism attempts to keep core temperature constant at 37±0.5°C. Skin temperature can vary widely, decreasing by an average of 10°C during cooling and more in hypothermia.[16]

Because the power of radiation is directly proportional to the fourth power of surface temperature in degrees Kelvin, even a small change in body surface temperature can lead to a large change in the amount of radiated heat.[4] Radiant heat loss from uncovered skin in windless conditions can account for about a quarter of total heat loss. After longer exposure to cold, once skin temperature has decreased, radiation generally accounts for less than 5% of heat loss.[17]

In a cold environment, the temperature of the outer layer of clothing must be considered. Heat transfer takes

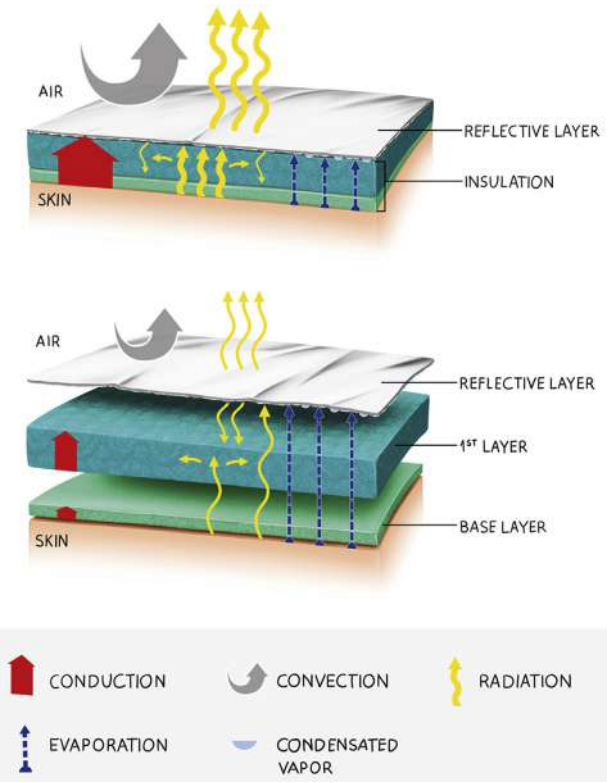

Figure 2. Simplified model of heat transfer through a fabric system. Top image: Heat loss increases when the system is compressed and there are no air spaces. Bottom image: Less heat is lost through a closely applied multilayer fabric system covered with reflective foil. Authors' own graphic.

place from the skin surface to the innermost layer of clothing and then to each subsequent overlying layer of clothing (Figure 2). A thermal gradient builds up between the temperature of the innermost surface and the temperature of the outermost surface. The higher the temperature of the outermost surface, the worse the insulation and the higher the heat loss to the environment.[5,18,19]

Practical Aspects of Protection from Heat Loss

The magnitude of heat loss from the body by various mechanisms depends on many factors, including weather conditions, clothing, body position, and individual physiology. The mechanisms and rates of heat loss can change rapidly. In a windless, cold environment, when air movement is minimal, a significant amount of heat is lost by radiation. When a person is lying on or is otherwise in contact with a cold surface, significant heat is lost by conduction. Heat loss by conduction also increases if clothing is wet.[18,19] Any movement of surrounding air, especially wind, increases heat loss by convection.[20]

Advertising for reflective foils sometimes claims that they decrease heat loss by 90%. This would be true only if there were no mechanisms of heat loss other than radiation and if the effective reflectivity of the coating was almost perfect. Aside from the impossibility of eliminating heat loss by conduction, convection, and evaporation, the surface of the foil is not perfectly reflective.[8,9] The most important factor that affects the effectiveness of a foil to limit heat loss is the method of application.

Reflective foils have been used for years to decrease heat loss during the perioperative period.[2,3] In this application, the foil is usually placed directly on the skin or on clothing, such as underwear. If the reflective side is separated from the body surface, there is a trapped air layer between them where heat exchange occurs largely by radiation. Little heat is lost because radiant heat is reflected back to skin or clothing and reabsorbed. If the foil is in direct contact with the body, it gains heat by conduction, rapidly reaching body temperature. As a "second skin," the foil loses heat by radiation and by convection.[20,21]

In emergency medical services, URFs are placed on accident victims, directly over clothing, or as the outer layer of multilayer insulation. Heat from the body flows across the air space between skin and fabric and then through the fabric system to the outer surface of the fabric system (Figure 2). During this process, heat is transferred by conduction, convection, evaporation, and radiation.[20,22-26] Although radiant heat can penetrate fabrics, it also can be absorbed or scattered.[22] At the outer surface of clothing exposed to air, heat is lost by convection and radiation. If a URF is the outermost layer, it reflects radiation emitted by the adjacent outermost insulating layer. If the insulation is effective, radiation from the outer layer to the foil is minimal.[11] In addition, emissivity of fabric is lower than that of skin, limiting radiation of heat.[13]

Conduction through clothing or other coverings is affected by the properties of the fabrics and by the construction of the textile assemblage with fabric layers and trapped air. The thermal insulating capacity of a clothing ensemble depends mostly on its ability to trap air. The amount of insulation generally increases with the number of covering garments.[11,20,23] Thermal insulation is directly proportional to the thickness of the ensemble (about 1.3–1.5 clo·cm^{-1}). Maximum heat flux is reduced by up to 80% when the air gap is increased from 0 to 1 mm. The marginal decrease of heat flux caused by further increasing an air gap tends to become smaller as the gap is increased. In thicker air layers (>10 mm), convection may occur, increasing heat loss.[23,24] Movement of trapped air can also be caused by body movement or by external factors, such as mechanical pressure or wind.

Table 1. Advantages and disadvantages of URFs

Advantages	*Disadvantages*
• Small packed volume • Lightweight • Waterproof • Windproof • Can be an element of multilayer insulation • Can increase detectability in search and rescue missions • If used over the eyes, can protect from snow blindness	• May tear in a strong wind • May tear from contact with sharp objects • Reflectivity is decreased by condensation of vapor at the inner layer of the foil • Provides little protection from conduction • Has limited effectiveness as a single layer, especially in windy conditions • No data on performance in field conditions

If the URF is wrapped too tightly, the volume of trapped air is reduced, increasing the conductivity of the clothing and decreasing the thermal gradient. In a cold environment, the result is an increase in heat loss.[5,19-21] Securing a patient to a backboard or stretcher can also cause folding or bunching of the insulation, increasing heat loss when the volume of trapped air is reduced.[21]

Placing a vapor barrier, such as a URF, outside an insulating layer can potentially increase heat loss by causing condensation of water on the inner side of the vapor barrier. Repeated cycles during which moisture in contact with the skin absorbs heat, evaporates, and then travels to the outer layers, where it condenses and returns to the skin surface, can cause heat loss. This mechanism is called the "heat pipe effect."[25,26] Moisture in textiles decreases thermal insulation when it replaces air because water has 25 times greater thermal conductivity than air. In cold conditions, condensation and freezing of moisture accumulating on the inner side of the URF significantly impair its reflectivity.[8,9] Also, folds, creases, and other defects can change reflection directionality, decreasing overall reflectivity.[21]

A URF can protect against wind and rain, but unlike a rigid covering, it can be distorted by even light air movement.[5,9] The resultant mixing of trapped air increases convective heat loss. A single layer of URF provides an additional 1 to 3 clo, depending on wind speed. In a windless environment, a URF can provide protection equivalent to that provided by a woolen blanket. Moderate wind (2–3 $m \cdot s^{-1}$) decreases protection by as much as 40%, and strong wind (>8 $m \cdot s^{-1}$) by up to 55%. In moderate or strong wind, reflection from metallized foils becomes negligible and the overall thermal protection of metallized foils is the same as nonreflective polyvinyl chloride foils of the same thickness.[5]

Most commercially available foils have a surface area of about 2.8 m^2. This is too small to cover most patients with a single foil. It is usually necessary to use 2 or 3 foils, making it impossible to create an air- or watertight covering. Leakage between foils increases convection and allows moisture to penetrate the covering, increasing heat loss.

Gold to Cold?

Theoretically, a foil should be used with the silver side toward the patient and the gold side facing the environment, but reflectivity (0.97–0.99) does not differ significantly between the 2 sides.[27,28] The emissivity of the gold side is about half that of the silver side (ε=0.02 vs 0.04). At a given foil temperature, the radiative power of the gold side is about half that of the silver side.[28,29] The clinical importance of this phenomenon is limited. Reducing emissivity is of marginal significance when there is high heat loss via conduction and convection.[8] Transmission of radiation by URFs in near, middle, and far infrared wavelengths is very low.[11,12] It does not matter whether the gold side or the silver side is facing outward when a rescue blanket is used to protect against hypothermia in a wilderness emergency.[9,12]

Clinical Studies

We found only 7 clinical studies of URFs in field conditions.[5-7,9,30-32] Most of the human studies had a small number of subjects.[9,30-32] Because the subjects were not hypothermic, skin surface temperatures were likely higher than they would have been in hypothermic subjects. Individual studies do not describe exactly how the reflective foils were placed, limiting the ability to compare results among studies. One study found no significant difference among protective materials in 2 weather conditions.[9] Heavyweight blankets and URFs provided protection from sun, wind, and rain. Blankets were durable, while URFs tore easily. Neither had any advantage over simple polyethylene bags for insulation in a cold environment. The mean decrease in skin temperatures with URFs was similar whether the reflective side faced in or out. Condensation formed on the inner

surface of the foil early in each exposure and rapidly froze, especially at lower air temperatures. In another study, a casualty bag incorporating a URF did not decrease heat loss.[30] The calculated insulation value of the complete casualty bag was close to the predicted value for textile layers without the URF. In a study of subjects with different combinations of passive insulation with and without a reflective foil, adding URF inside 1 or 2 blankets provided better thermal insulation in the wind than the blankets alone.[32] There was less decrease in thermal insulation caused by moisture inside the coverings when a waterproof reflective sheet or bubble wrap was used inside blankets. In a randomized trial of prehospital thermoregulatory interventions, all trauma patients receiving passive insulation, including reflective blankets, had a decrease in core temperature during transport.[31]

Four studies used thermal manikins.[6,7,18,32] In one study, rescue foil improved thermal protection when added to woolen blankets, but URFs used alone had no significant benefit.[6] Another manikin study compared 4 insulating blankets and 1 warming blanket. A URF was more effective than a polyester ambulance blanket, but less effective than bubble wrap, a heavyweight blanket, or the heating blanket.[7] Another study showed that thermal protection in low wind conditions was proportional to the thickness of the insulating layers, although URFs had higher insulation than expected.[5] Convective heat loss increased with increased wind velocity. In moderate and high wind conditions, thermal insulation was best preserved by waterproof, rigid systems capable of resisting compression. A URF used as a single layer achieved thermoneutrality in a low wind environment at temperatures as low as 10°C. In cold, windy conditions, URFs are probably most useful as windproof outer covers.

Are URFs Useful in Prehospital Medicine?

It is unclear whether URFs have a legitimate role in prehospital medicine. They have advantages and disadvantages (Table 1). A fundamental problem is the unfounded belief of many medical personnel that URFs have unique properties. Clinical research suggests that URFs lack meaningful advantages over other materials for the prevention and treatment of hypothermia,[6,9,30,31] or have only minor advantages.[5,7,32]

Summary

Thermal insulation is proportional to thickness and to the ability of an insulating material to maintain compartments with trapped air in and between fabrics. Ultrathin foils likely have the same level of effectiveness as other waterproof materials of similar thickness used in prehospital medicine. An ultrathin foil alone does not significantly limit heat loss. The insulation properties of an ultrathin foil can be altered by factors such as wind or by moisture condensation on the inner side. In clinical practice, it does not matter which side, silver or gold, faces outward.

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CLINICAL IMAGES

A Case of Partial Thickness Burn and Ruptured Tympanic Membrane from Rural Nepal

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A 5-y-old boy presented to the emergency department (ED) with painful blisters over the chest and both legs. Fresh blood was seen coming from his left ear. The blisters on the chest were ruptured by friction with his clothing (Figure 1). What caused these blisters? How should you manage this case?

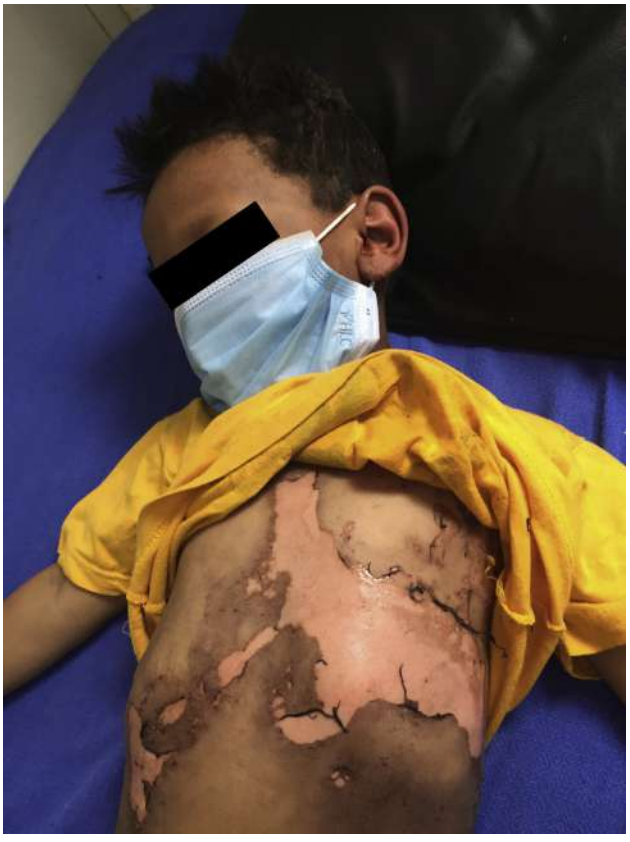

Figure 1. Ruptured superficial skin blisters over the chest and fresh blood over the left external auditory meatus.

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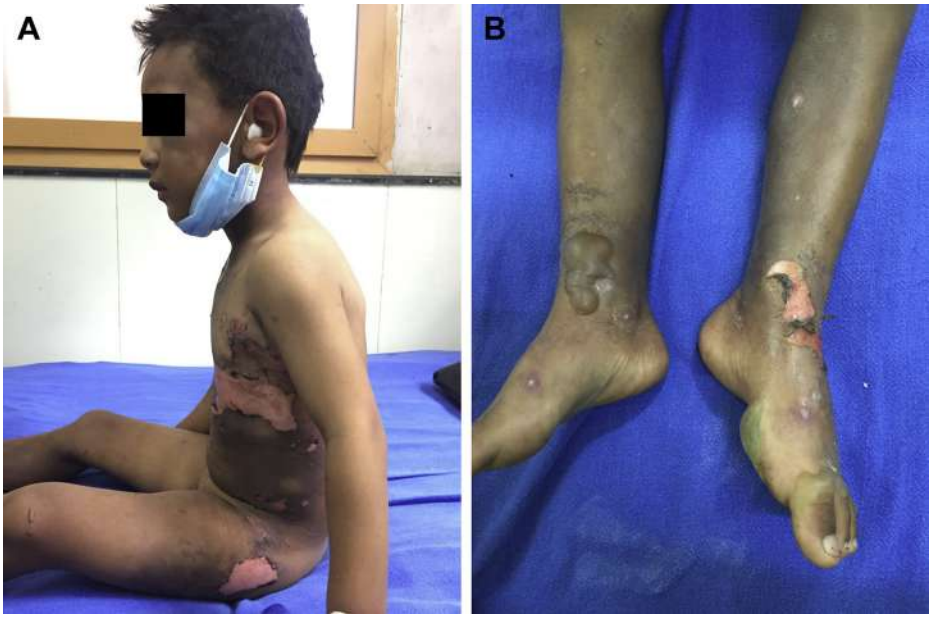

Figure 2. Superficial burns over the body (A) and lower limbs (B) of the younger brother.

Diagnosis

Partial thickness burns and ruptured tympanic membrane due to lightning injury.

Case Details

The patient was sleeping on the floor in a bed shared with his 9-y-old brother adjacent to the window in his rural house (cement floor, brick wall, and galvanized sheet roof). The elder brother reported thundershowers that night. He remembered waking up to a loud, crash-like noise, which he thought might be a thunderclap. He shouted in fear and called his parents. His parents came immediately into the room. Because there was a power outage, they lit their mobile phone flashlight. They saw a blister on the left side of the elder son's face. Then they checked their younger son, who was unconscious. There were multiple blisters over his body, and he was bleeding from the left ear. His clothing was torn. They woke their neighbors and called for help. By then, the younger son had regained consciousness. A vehicle was arranged, clothes were changed, and both children were rushed to the hospital.

In the ED, the patient was fully conscious. On examination, he had a pulse rate of 124 beats·min^{-1}, blood pressure 100/60 mm Hg, respiratory rate 20 breaths·min^{-1}, and temperature 37.7°C. General examination showed

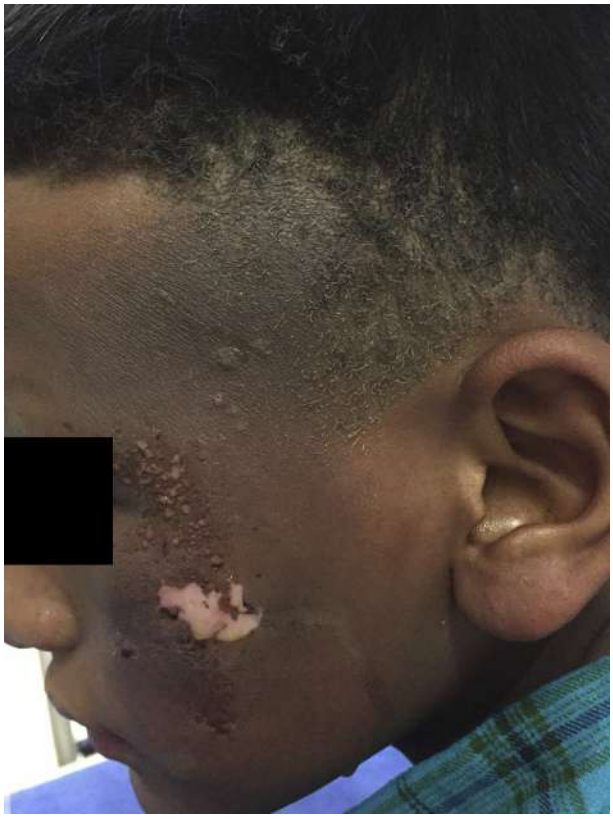

Figure 3. Supsterficial burn over the left side of the face in elder brother.

singeing of the scalp hair; superficial burns over the chest, mostly on the left side; and blisters over both legs (Figure 2). The burnt area was erythematous. Fresh blood was seen in the external auditory meatus of the left ear. When the left external auditory canal was viewed with an otoscope, minimal bleeding and perforation on the anteroinferior quadrant of the left tympanic membrane were seen. The patient reported pain at the site of superficial burns. There was no entrance or exit wound. Computed tomography of the head was normal. The patient was admitted to the burn unit for further management.

The patient's elder brother had no injury except for a 4×2 cm hemorrhagic blister over the left zygomatic prominence (Figure 3). His vital signs were stable, and there were no other complaints. The wound was cleaned, and he was discharged.

Discussion

Thunderstorms are frequent in Nepal during the rainy season. As in most less-developed countries where houses and other structures seldom have lightning rods, lightning injuries are common in Nepal. Unlike the victims of electrical injury, victims of lightning strike do not carry a residual charge and can be safely resuscitated.[1]

A stable patient with no history of loss of consciousness, a normal neurologic examination, and a normal electrocardiogram requires no intervention.[2] A patient with a history of neurologic abnormalities after a lightning strike or with an abnormal neurologic examination in the ED should undergo appropriate imaging studies and be observed.[3]

Burns associated with lightning injury are usually produced by contact with metal objects that are heated by the electric current. Full-thickness burns requiring skin grafting are rare.[1] Lightning burns usually involve side-splash and are superficial.[1] Tympanic membrane rupture caused by the blast effect is common in lightning injuries.[1]

In the present case, there was only superficial skin involvement. The exact mechanism of injury in the present case could not be determined with certainty. The most likely mechanism was side-splash, because both children sustained superficial facial burns. Lightning should be in the differential diagnosis of unexplained burns, especially during and after thunderstorms.

Written informed consent was obtained from the patient's father for the use of images and medical history for educational purposes, including publication.

Keywords: lightning, side-splash, superficial burn

Author Contributions: Literature review, manuscript draft and review, approval of final manuscript (AA, ADS, SN).

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