WILDERNESS MEDICINE

The official newsletter of the WILDERNESS MEDICAL SOCIETY

An international non-profit professional association serving the medical interests of the outdoor and wilderness community.

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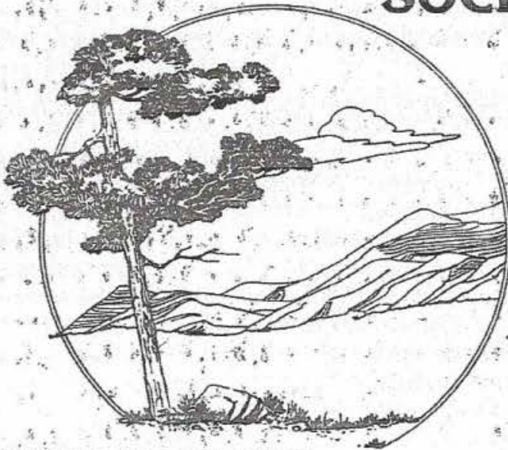
SOCIETY BUSINESS



ANNUAL MEETING

The Society will hold its 1987 annual meeting on Tuesday and Wednesday, August 25-26, at Snowmass, Colorado, just outside of Aspen. Once again the meeting will be held in conjunction with the Wilderness Medicine conference sponsored by the University of California at San Diego, which runs from August 23-28. Participants in the WMS meetings may want to register and attend the UCSD conference lectures, but the two events may be attended separately.

The annual meeting is an opportunity to meet the board of directors and other WMS members. Society business is discussed with input from the members. Those who wish to develop projects through the WMS may begin discussion and organization of their ideas. Half of the time (accredited for CME hours) is devoted to scientific papers and presentations. This year we also hope to have several outdoor rescue demonstrations. The tentative schedule is:

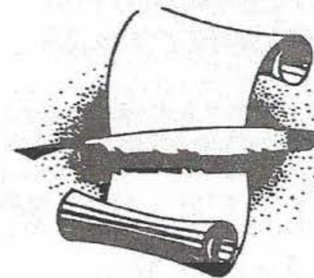
Tuesday, August 25
6:00 P.M. - Reception and business meeting.
7:30 P.M. - Dinner and installation of new officers.

Wednesday, August 26
8:30 - 9:00 AM. - Announcements, summary of business
9:00 - 12:00 noon. - Abstracts and presentations
1:30 - 2:30 - Committee or project meetings
2:30 - 4:00 - Rescue demonstrations

There will be no registration charge for WMS members to the annual meeting, but participants will have to pay for their dinner.

ABSTRACTS REQUESTED FOR ANNUAL MEETING

Members are urged to participate in the annual meeting by making a short (10-20 minute) presention of original research, projects in progress, ideas for projects, or any topic of interest related to wilderness medicine. These are intended to share interests and projects with other society members. An abstract of the presentation should be submitted to the WMS office for scheduling the meeting and for potential publication in the Newsletter.

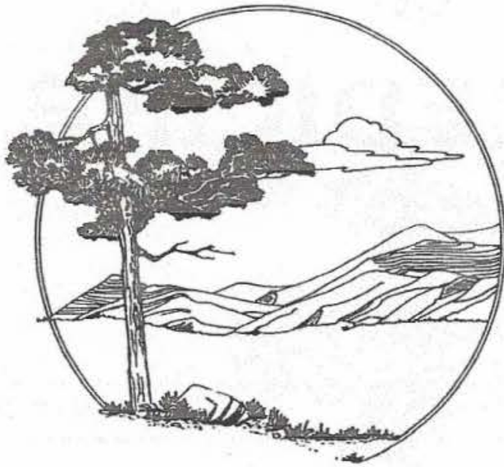


MEDICAL STUDENT RESEARCH GRANT

The WMS will award $1,000 annually, in honor of Dr. Charles Houston, to support research in wilderness medicine by a medical student. Letters are being sent to medical students throughout the country. Supporting research is seen as an important function of the WMS. The board wanted to begin this support in a tangible fashion, however small.

PRESS EXPOSURE

The Outdoor Writers Association of America is preparing an article on the WMS to be released this spring. This will expose the Society to a large number of people and outdoor groups. The San Jose (California) Mercury recently featured the WMS in an article.



BOARD OF DIRECTORS

At a recent meeting, the board made several changes in its composition. Given the current interest in participation, two members-at-large will be added to the current number, increasing the total to 7 members. Candidates for these positions are included in this newsletter. Terms will run for three years, although officers terms will remain two years. Two positions that had been vacated early were filled by Peter Hackett and Blair Erb. Dr. Hackett is the subject of Members Profile in this newsletter. Dr. Erb is a cardiologist in Jackson, Tennessee, whose interests include exercise physiology for wilderness activities and organizing the literature in wilderness medicine.

CANDIDATES FOR THE BOARD OF DIRECTORS

Four candidates have been selected from a group of interested members to fill the two positions on the Board of Directors. A ballot is enclosed in this newsletter. Please vote for a maximum of two (2) members and return your ballot by May 15.

EDWARD C. GEEHR, M.D.

Dr. Geehr is Director of Emergency Medicine at The Albany Medical College, New York, and a founding member of the Wilderness Medical Society. He has served on the Board of Directors since the Society's founding. He has a broad range of interest in wilderness medicine and is the co-author of the standard reference *Management of Wilderness and Environmental Emergencies,* as well as the author of a number of papers on poisonous plants and emergency medicine topics.

KENNETH V. ISERSON, M.D., M.B.A.

Dr. Iserson is an Associate Professor and Director of Residency Training in emergency medicine at the University of Arizona College of Medicine. He is also the medical director of the Southern Arizona Rescue Association and actively participates in their mountain, desert, river, cave and mine rescues. He is the author of numerous articles on emergency medicine and management.

JOHN P. ALLEN, M.D.

Dr. Allen is a practicing neurologist and neuroendocrinologist in Galesburg, Illinois, and an Associate Professor of Neurosciences at the University of Illinois College of Medicine. He has been involved in research in exploration medicine and in hormonal parameters in adaptation to high altitude and has participated as a medical officer on several climbing expeditions. He has published numerous articles on neuroscience topics.

PETER J. KOLTAI, M.D.

Dr. Koltai is a practicing otolaryngologist and assistant professor of surgery and pediatrics at Albany Medical College. He is an active mountaineer and member of the Adirondack Mountain Club. Special interests include head and neck injuries in mountaineering and nasophysiology at high altitude.

MEMBERSHIP QUESTIONNAIRE

About 300 questionnaires have been returned, and the data is being entered into our computer. Those who have not returned it are urged to do so. Information from about 175 members reveals the following: 85% are MD, but 40% of those were in specialties other than emergency, internal medicine, family medicine, surgery, or orthopedics; 89% commonly see environmental problems in their practice; every activity and area of special interest was represented by a minimum of 10% of members; more than half wished to become more involved in the WMS through projects, research collaboration, liason with other groups, or writing for the Newsletter. The picture emerging is that of a diverse, active, accomplished membership, with the interest and expertise to advance the knowledge of wilderness medicine. This information will be available to members of the WMS who wish to contact other members in specific geographic areas or with special interests. All requests for information will be screened by a board member.

SPEAKERS BUREAU

The WMS regularly receives requests for speakers from a broad spectrum of organizations. We would like to match these requests to appropriate, interested members. If you would like to be listed as a potential speaker, please send a letter indicating preferred topics and a CV to the WMS office. Your name would be released only to legitimate organizations whose needs match your indicated interests and requirements.

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MEMBER'S PROFILE: PETER HACKETT, M.D.

by Howard Backer, M.D.

It would be more appropriate to write a book about Peter Hackett than this brief article. Few people have more experience or expertise in wilderness medicine. Born in Chicago, Peter was introduced to the mountains through camping with his grandparents. Except for ski trips to Colorado, he remained in the midwest through college and medical school. In 1974, after completing an internship at San Francisco General Hospital, Peter was drawn to the mountains. He began work in Yosemite National Park as a fire-fighter (paid $4.47/hour), but also flew helicopter rescues and taught the first EMT course in the Park. Then came an opportunity that changed his life. An injured climber, whom Peter helped, was the director of Mountain Travel and asked him to work as a trek doctor in Nepal.

This first trip to Nepal lasted 18 months. In addition to the Mountain Travel treks, Peter spent a few months exploring the Himalayas on his own. For 3 months, he staffed a small seasonal medical clinic run by the Himalayan Rescue Association on the trail to Everest base camp at 14,000 feet. Later, he became director of the HRA, and developed it into a major seasonal medical service which treats the myriads of trekkers and climbers in Nepal, as well as the locals. For a while, this clinic had the only blood gas machine in Nepal; power was provided by a generator.

In Pheriche, Peter became interested in the large number of people with altitude sickness. He began gathering clinical data and, in 1975, published his first study on altitude sickness.[1]

Most of the next six years were spent in Nepal, trekking, climbing, and working on research, clinical medicine, and mountain rescues in the shadow of Everest. Within this period, Peter also completed a research fellowship in cardiopulmonary physiology at the University of Colorado (with 4-5 months of field work, of course).

One memorable rescue in Nepal involved a man found comatose at 16,000 feet with cerebral and pulmonary edema. This patient was carried down to the clinic (a stone hut) and ventilated by hand for two days before air evacuation to Kathmandu, where he died from cerebral edema and a large pontine hemorrhage.

The most unforgettable experiences in Pheriche were not medical cases or sunsets on Everest, but the interpersonal, cultural connections with Sherpas, who inhabit this area. Peter became a member of the family next door to the clinic. Despite the picturesque aspect, life for these people is extremely hard. In the summer of 1981, both parents of this family died of illness — refusing to travel three days to the nearest hospital since they knew Peter was coming soon. Their dying request was that Peter look after their three children. He has honored their wish.

Peter learned about this family tragedy and his new responsibility en-route to climb Mt. Everest with the American Medical Expedition. That expedition, organized by John West, was remarkably successful in gathering physiologic data at extreme altitudes as well as putting men on the summit. Peter accomplished a solo summit climb from high camp. Descending, he slipped on an 80 degree pitch, but miraculously, his fall was arrested. Peter found himself dangling upside down at 26,000 feet with his lower legs wedged behind a rock. Completely exhausted, he began attempts to extricate himself (not yet sure whether to congratulate himself on his luck). Then, he discovered a fixed rope, buried in the snow, that had been left by a previous expedition.

The year before, Peter had been co-leader of an expedition to Baruntze, and the following year he organized a group which succeeded in a highly technical first ascent of Cholatze (both are 8,000 meter peaks in Nepal). However, expedition climbing is not his favorite wilderness activity. He prefers climbing or skiing light and fast with a few close friends.

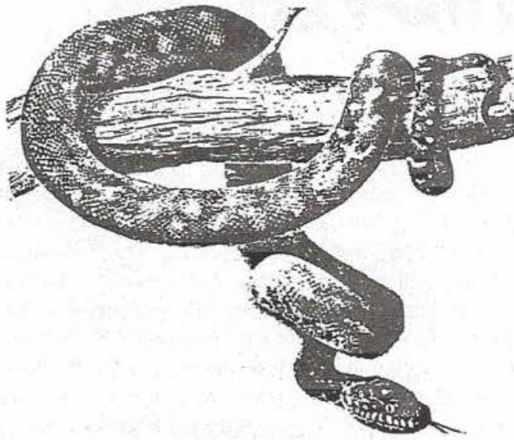
During this same period, Peter also worked as an emergency doctor in the U.S. and organized a summer rescue/treatment/research station on the side of Denali (Mt. McKinley). This camp consists of snow caves, tents, and solar powered equipment at 14,300 feet. Most of his recent research has come from this field lab, including a study analysing high altitude pulmonary edema fluid obtained from patients by bronchoscopy.[2]

The extreme environmental conditions on Denali have also created some dramatic rescue situations. Two Japanese climbers who fell were rescued in the face of an imminent storm. Comatose from cerebral edema, hypothermic, and frostbitten, they were kept alive for three days in the clinic tent before military helicopters could evacuate them. Both survived.

Currently Peter is based with the Department of High Latitude Studies, University of Alaska at Anchorage, and practicing emergency medicine. He has authored more than 30 papers, most of these original research. His research continues in the pathophysiology of altitude illness and the relationship between the brain and lung in these disorders. Having reached some of the highest points on earth, Peter has now extended his interest into space. He was a consultant for the Voyager flight, and doctor for an expedition to hang-glide off Everest. He is currently consulting for Glider 500, an attempt to set a new altitude record in a glider plane (above 50,000 feet), and for a NASA project. Peter hopes to go into space himself, and it is a safe bet that he will.

[1]Hackett PH, Rennie D. The incidence, importance, and prophylaxis of acute mountain sickness. *Lancet;* 2:1149-55, Nov 27, 1976.

[2]Schoene RB, Hackett PH, Henderson WR, et al. High-altitude pulmonary edema. Characteristics of lung lavage fluid. *JAMA;* 256:63-9, July 4, 1986.



ELECTRIC SHOCK TREATMENT OF SNAKEBITES

by Sherman A. Minton, MD
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In a communication to *Lancet,* Ronald H. Guderian and colleagues report the use of high voltage electric shock as a treatment for snakebite, based on their experience in jungle regions of Ecuador[1]. A direct current of 20-25 kV at less than 1 mA was applied to the bite area for 1-2 seconds. Usually, four or five shocks were given at 5-10 second intervals. An outboard motor was the usual source of current, and a "stun gun" with a 9 V battery was modified to serve as a portable current source.

Thirty-four patients treated within 30 minutes after being bitten had prompt relief of pain and developed no swelling, hemorrhage, bullae, or evidence of systemic envenomation. Two patients treated about two hours after they were bitten had prompt relief of pain and remission of spontaneous bleeding. Swelling did not progress but persisted three days. "Controls" were seven individuals who refused shock treatment and "experienced the classic complications;" two required amputations.

As the authors state, the biological basis of this treatment is unknown. It appears quite unlikely that electric current would inactivate selectively the polypeptid and protein toxins of snake venoms. It also seems unlikely that the cellular receptor sites for these toxins would be altered by the current. The authors suggest, "shut-down of local vessels by electrospasm may confine the venom locally long enough for it to become inactive." Contrary to the authors' opinion, venom has quite a long half-life in tissues and can be detected at the site of snakebites as long as two weeks after the injury[2]. Moreover, it appears to be spread mainly by small lymphatics that would not be subject to shut-down.

A more plausible hypothesis is the liberation by electric shock of an antagonist to some of the venom toxins. It is known that some mammals have such antagonists in their tissues. The opossum and wood rat have an anti-hemorrhagic and antinecrotizing factor for rattlesnake venom in their serum, but it is not an antibody[3,4]. Although the mongoose is highly resistant to venom of the Palestine viper and some other snakes, its serum has no protective activity[5].

The *Lancet* account leaves much to be desired. There is virtually no information about the patients. Sex and age are not given. It would be important to know if they were members of a population that has high exposure to snakebite, and thus likely to have protective antibody. There is a report of one such Ecuadorian tribe in which 78% of randomly selected adults had antibodies to snake venoms[6]. It is also possible that some of the seven patients who refused electric shock were among the more severely envenomed and did not wish to be subjected to an uncomfortable treatment.

The snakes that bit the patients are not identified. Of the six pit vipers listed by the authors as characteristic of the region, *Bothrops atrox* (the fer-de-lance to English-speaking peoples) is known as a major cause of serious snakebites. *Lachesis muta* (the bushmaster) is the largest of pit vipers and potentially very dangerous, but it is generally uncommon and accounts for few bites. *Bothrops bilineautus, B. schlegelii,* and *B. nasutus* are small pit vipers that rarely cause serious envenomation. *B. castelnaudi* is a poorly-known species, however, there is no evidence it is exceptionally dangerous. It must also be emphasized that all snakes, including the most dangerous species, may bite and inject little or no venom. It is estimated that 25-30% of *Bothrops* bites in Central America are not accompanied by significant envenomation[7]. Finally, in tropical America, there are several mildly venomous and nonvenomous snakes that are excellent pit viper mimics. Any critical evaluation of snakebite treatment must, insofar as possible, identify the snakes responsible for bites and consider the incidence of blank bites and bites by nonvenomous snakes.

The history of snakebite treatments is as old as medicine itself. This reflects the dramatic nature of the affliction and its unpredictable course. It also emphasizes the old dictum that if a disease has many remedies, no one of them is highly effective. Electric shock as a snakebite treatment needs much more thorough evaluation.

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CEREBRAL RESUSCITATION IN THE TREATMENT OF NEAR-DROWNING

by Kenneth W. Kizer, MD, MPH

Two recently published studies question the role of cerebral resuscitation and intracranial pressure (ICP) monitoring in near-drowning victims.

Bohn, et al, report their experience; with 40 near-drowned children admitted to the intensive care unit (ICU) of Toronto's Hospital for Sick Children from 1978 - 1982.[1] All the patients, whose mean age was 3.6 years, were victims of fresh water immersion, had been submerged for 5-20 minutes, and required intubation and ventilation for resuscitation. Water temperatures were not reported, but most patients were hypothermic (30°-33°C) after resuscitation. Thirteen patients died and 27 survived, seven of whom were left with permanent neurologic damage. Of the seven neurologically impaired victims, two subsequently died of delayed respiratory failure. All nonsurvivors were autopsied.

Twenty-four patients were treated with a regime of hyperventilation, hypothermia, and high-dose phenobarbitone, along with continuous ICP monitoring and other supportive measures as clinically indicated. Ten of these patients died. Three were diagnosed as having cerebral death shortly after admission from severe cerebral edema with herniation.

The other seven had marked respiratory distress, diffuse ischemic injury, and severe cerebral hypoxia without raised ICP or evidence of severe cerebral edema. Six of these seven patients developed septicemia; in all six cases, this was associated with profound neutropenia. The other 16 patients were treated with a similar protocol but without hypothermia. Three of these patients died, but only one developed septicemia.

In this series, neutropenia was associated with a very poor prognosis, regardless of body temperature, but hypothermia was clearly associated with the development of neutropenia. Hypothermia was not, however, associated with an increased number of neurologically intact survivors. Likewise, even though use of high-dose barbiturates was effective in maintaining ICP at less than 20 mm Hg, their use did not improve outcome. The authors conclude that the use of hypothermia and barbiturates to control brain swelling is of no benefit when treating near-drowning in children. Hypothermia may be harmful because it decreases circulating neutrophils, compromising neutrophil migration and reticuloendothelial clearance, and increases host susceptibility to bacterial invasion.

In a second study, Allman, et al, report their experience with 66 near-drowned children admitted to the ICU at Childrens Hospital of Los Angeles from April 1979 through September 1984.[2] All patients (mean age 2.8 years) were victims of fresh water submersion, required full cardiopulmonary resuscitation, and had an initial Glasgow Coma Score (GCS) of 3 in the referring emergency department. All cases occured in water warmer than 20°C. Thirty-three of these patients died, 17 survived in a vegetative state, and 16 appeared to recover fully.

All patients were reevaluated according to a standard protocol on arrival in the ICU and their GCS revised accordingly. None of the 37 patients with a GCS of 3 in the ICU recovered normal neurological function - 26 died and 11 survived in a persistent vegetative state. All patients with a GCS greater than 5 appeared to recover normal neurological function (although one died of delayed respiratory failure).

Forty-seven patients with a GCS of less than 6 underwent continuous ICP monitoring and aggressive pharmacologic therapy to control ICP. Hypothermia was not used. None of the patients who recovered full neurologic function had ICP elevations significantly greater than 20 mm Hg; likewise, 13 of 21 patients who died or survived in a vegetative state had no ICP elevations significantly greater than 20 mm Hg. However, as has been previously shown, sustained ICP above 20 mm HG was associated with either severe neurologic disability or death. The authors conclude that, even when ICP is maintained at less than 20 mm Hg, severe neurologic sequelae may still occur, and they question whether the utility of continuous ICP monitoring justify the expense and risks of this intervention.

Previous studies have reported a benefit to monitoring and maintaining ICP at less than 20 mm Hg, but they were small series of patients having diverse neurologic conditions and varying environmental exposures. These two relatively large and well done studies raise significant questions about the role of cerebral resuscitation in the treatment of near-drowning. They indicate that ICP monitoring cannot be recommended as part of the standard treatment protocol. Like prior studies, they emphasize the importance of aggressive initial resuscitative measures to maintain cerebral perfusion and systemic oxygenation.

[1]Bohn DJ, Biggar WD, Smith, CR, et al. Influence of hypothermia, barbiturate therapy, and intracranial pressure monitoring on morbidity and mortality after near-drowning. *Crit Care Med* 1986; 529-534.

[2]Allman FD, Nelson WB, Pacentine GA, et al. Outcome following cardiopulmonary resuscitation in severe pediatric near-drowning. *AJDC* 1986; 140: 571-575.

BRETYLIUM IN HYPOTHERMIA

by Daniel F. Danzl, MD, FACEP

Murphy and associates[1]

Murphy and associates, from the Department of Emergency Medicine at Henry Ford Hospital in Detroit, evaluated the effectiveness of bretylium tosylate (BT) in the prophylaxis and treatment of hypothermic ventricular fibrillation (VF).[1]

Twenty-two dogs were placed in a cold room and cooled to 24°C. Then the dogs received a blinded infusion of placebo or BT. Five maneuvers were performed in sequence ten minutes after infusion: oral endotracheal intubation/extubation, central line and nasogastric tube insertion, jostling, and flotation-tip catheter insertion.

Six of eleven (55%) of the dogs given placebo fibrillated with manipulation, versus only one of

eleven (9%) that were pretreated during hypothermia with 5mg/Kg BT (P = .067). Five of the six placebo dogs that fibrillated did so during Swan-Ganz catheter insertion. Endotracheal intubation did *not* induce VF. Of the placebo dogs that fibrillated, four required BT to convert to a perfusing rhythm. The other two achieved stable rhythms following countershock alone or combined with epinephrine.

Interestingly, three of the eleven dogs receiving BT fibrillated at four, six, and seven minutes into the fifteen minute infusion. The remaining BT was then given in a bolus. All three dogs were defibrillated, one with difficulty. The authors conclude that controlled clinical trials of the prophylactic use of BT in hypothermic humans are warranted.

Discussion

Following an initial tachycardia, hypothermia induces a progressive bradycardia, with the pulse decreased 50% at 28°C. The conduction system is more sensitive to cold than myocardium, as reflected on EKG lengthening of the PR, QRS, and particularly the QTc intervals. While not prognostic or diagnostic, the J (or Osborn wave) may be seen as a hump at the junction of the QRS complex and ST segment. This cardiac cycle prolongation is unresponsive to atropine.

Atrial and ventricular dysrhythmias are common below 32.2°C (90°F). All atrial arrhythmias should be considered innocent and left untreated, since ventricular response is slow. They usually convert spontaneously during rewarming.

Prevention and treatment of ventricular arrhythmias in hypothermia present several clinical problems. Preexistant chronic ventricular ectopics may be suppressed or completely eliminated by temperature depression. On rewarming, the physician noting the emergence of these ectopics is placed in a quandry unless past medical history is available.

The incidence of iatrogenic VF should be fairly low during hypothermia if patients are well oxygenated and then handled and rewarmed carefully.

The origin of the myth regarding the hazards of endotrachel intubation of hypothermic patients rests with Fell.[2] He stated, in reference to a series by Lee and Ames, that "endotracheal intubation was followed by cardiac arrest in a large proportion of the cases..." Lee and Ames[3] reported no such cases, and only cautioned that "intubation . . . *may* induce a cardiac arrest or vomiting." We reported 40 patients in a series of 135 who were nasotracheally intubated without incident.[4] Ledingham[5] also noted no problems with intubation in his prospective series of 44 patients. In a recent multicenter study, 117 hypothermic patients were intubated uneventfully.

Pharmacologic options are limited. Hypothermia induces complex physiologic changes which result in abnormal responses to drugs[6]. Metabolism and excretion are both progressively decreased, and thus some medications have exaggerated effects.

Procainamide reportedly increases the incidence of VF. However, another group 1 ventricular antiaarhythmic, quinidine, has been useful during induced profound hypothermia. Likewise, 100 mg/Kg IV magnesium sulfate spontaneously defibrillated most bypass patients at 30°C in one series of induced hypothermia. Lidocaine has not been effective for prophylaxis, and is ineffective in facilitating defibrillation.

Bretylium tosylate (BT) is a unique bromobenzyl quarternary ammonium compound with hypothermic activity. Nielson[7] found BT increased the fibrillation threshold in cats. Then Buckley[8] demonstrated its efficacy in the prophylaxis and treatment of VF in a canine model. None of the dogs given 15 mg/Kg BT fibrillated, while 42% of the controls did. In both these studies, the BT was administered *prior* to induction of hypothermia. However, in another canine study by Elenbaas,[9] BT failed to facilitate defibrillation at 22°C. Human data is lacking. We reported a single case of "chemical defibrillation" with infusion of 10 mg/Kg BT in severe accidental hypothermia at 29.5°C.[10]

This study by Murphy is the first that evaluates the effects of the administration *during* hypothermia of BT for prophylaxis of VF. In this study, BT efficacy did not achieve but approached statistical significance (P = .0671). There is probably a high degree of biological variability for VF in any species, which affects data interpretation. Five of the eleven placebo dogs never did fibrillate with any maneuver.

If VF develops, BT should be the agent of choice. Since toxicity, optimal dosage, and in particular, the ideal rate of infusion is unknown, BT prophylaxis is not yet clinically warranted. As noted in this study, three of the eleven dogs fibrillated without manipulation during the BT infusion.

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DANTROLENE SODIUM FOR TREATMENT OF HEATSTROKE

by Mark D. Bracker, M.D.

Heatstroke may result when heat gain from metabolic production plus environmental exposure exceeds heat dissipation. Clinically, heatstroke is characterized by a marked increase in body core temperature followed by central nervous system dysfunction, hemodynamic instability, and tissue damage. Muscle rigidity is also commonly observed, particularly in malignant hyperthermia and exertional heatstroke.

Dantrolene sodium has been used in the prophylaxis and treatment of anesthetic induced malignant hyperthermia for almost two decades. It is a skeletal muscle relaxant that inhibits the release of calcium ions from the sarcoplasmic reticulum in muscle cells, thus muscle contraction and heat production are decreased. Amsterdam, et al[1], raise three theoretical reasons for the use of dantrolene in human heatstroke: "(1) some cases of heatstroke may actually be 'stress induced' cases of malignang hyperthermia; (2) muscle rigidity may play an important role in the excessive heat production that characterizes exertional heatstroke; and (3) dantrolene administration will reverse the defect in central thermoregulation common to classic heatstroke, exertional heatstroke and malignant hyperthermia."

This study evaluated dantrolene's effect on the cooling rate and biochemical changes in a canine heat stroke model. Non-exertional headstroke was induced in eleven anesthetized dogs by external heating. Cooling was passive. Animals in the treatment group (N = 5) were given 5mg/kg of dantrolene sodium and mannitol when rectal temperatures reached 43.0°C. Cardiac output, core temperature, arterial blood gases, CBC, electrolytes, BUN, creatinine, calcium, phosphorus, hepatic enzymes, cardiac enzymes, coagulation parameters, and urinalysis were measured. After 12 hours, all animals were sacrificed for postmortem examinations.

Data analysis showed no significant biochemical differences between the two groups, with the exception of serum creatinine and urine output. Better renal function in the treatment group was probably due to mannitol rather than dantrolene. Similarly, no differences in degree, type, or distribution of pathological abnormalities were observed between the two groups.

This study failed to test the hypothesis that reducing muscle rigidity might decrease the morbidity and mortality from exertional heatstroke: these anesthetized dogs did not develop muscle rigidity. The data indicates that dantrolene is not useful in the treatment of non-exertional heatstroke. A common thermoregulatory disorder has yet to be found to explain the occurance of heatstroke from exertion, environmental heat, and inhalational anesthetics.

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CASE HISTORY DEATH OF A TREKKER

by Sue A. Sherrod, M.D.

In November, 1984, 14 of us begin a journey to Everest base camp. From Kathmandu, Nepal (4,500 feet elevation), we fly to Lukla, a mountainside village with a landing strip at 9,275 feet. The next day we walk to Namche Bazaar (11,300 feet). Several of us develop mild symptoms of nausea and headache, which resolve after a day of rest. Over the next few days, we climb gradually along the Dudh Kosi river valley. On the tenth day, at 15,500 feet, one member of our group, Patricia, begins to get ill. She is 34 years old with a thin, athletic build, and no known medical problems. She has been one of the strongest trekkers, always among the first to complete the day's hike. Now Patricia complains of stomach cramps and lack of appetite. After a poor night's sleep, she feels no better and develops mild diarrhea. She is afebrile, has no specific pain, and is slightly unsteady, unable to walk a straight line. My concern is cerebral edema, which I discuss with our group leader, Peter. He suggests that we take her down to the last village, a drop of 1,500 feet in altitude.

Patricia starts walking down, but after one and a half miles she requires some help from Peter. That evening, she forces down a little soup broth. During the night, she has some cramps, nausea, one episode of diarrhea, and two episodes of vomiting.

The next morning, she feels weaker, and despite encouragement, drinks minimal fluids. Our goal today is to reach Pheriche (13,950 feet) where there is a high altitude clinic. Here, we hope she can be seen by a doctor with experience in altitude illness. Since she has not improved with descent, I think her problem may be due to a gastrointestinal infection rather that cerebral edema.

Patricia tries to walk, but does not have the energy. Our Sherpa-guide obtains a yak for her to ride. Approaching the village, we pass the doctor, who is on his way to locate the body of a woman who was lost crossing a high pass. After hearing my history, he suggests that Patricia's problem is more likely dehydration than mountain sickness, and advises rehydration.

We leave Patricia at Pheriche with a nurse from our group, while the rest of us go on to Everest base camp.

Three days later, we return to find Patricia better, but not well. She has been taking three liters of electrolyte solution daily, but is still quite weak. All signs of ataxia had resolved and sleep patterns had normalized.

She emphatically declines to fly back to Kathmandu. She wants to reach Thyangboche, a well known Buddhist monastery, which seems to hold some mystical meaning for her.

She seems almost a different person, somewhat uncooperative, but there is no consensus in the group as to whether she has changed. We are not sure if this is physical illness or a manifestation of home sickness and depression. By this time, cerebral edema is low on our list of differential diagnoses. In any case, Thyangboche (12,680 feet) is a descent from our current elevation; so we decide to go.

Patricia walks slowly. People help her, carry things for her, encourage her. She will not eat, but she drinks a little. She is getting weaker, not better. I reexamine her, but there are no abnormal findings.

The next morning everyone but Patricia eats breakfast. She does not even try to pack her bedroll. On the trail, she is at the rear of the group. We arrive mid-afternoon at Thyangboche. Patricia relaxes in the hotel dormitory and appears to be okay. At dinner, she even eats a little.

People in the hotel had all sorts of minor illnesses, and while I was 'making rounds,' someone calls me to Patricia. She is vomiting and although oriented, is acting inappropriately, throwing off her clothes and sleeping bag. We are alarmed, but not sure what to do; I have nothing in my limited medical kit that will help the situation.

We reassure her and settle her down. In the middle of the night, she again begins to vomit and throw off all covering and clothes. She becomes disoriented, talking as if she is at work in her laboratory. Within twenty minutes, she is unable to do anything but babble repeating sounds. We find a doctor experienced in altitude medicine who is confident this is cerebral edema. A bottle of oxygen is found and brought to her. We cannot move her until dawn. The night is long.

In the first light of morning we load Patricia on the back of a Sherpa, who carries her to a village 2000 feet down the mountain. We also send word to Namche Bazaar to radio for a helicopter from Kathmandu. The next morning, when the rest of us arrive in the village below, we find out that Patricia is dead. Due to cultural beliefs, the hotel owners had not allowed our Sherpa to bring Patricia into their houses. Finally, he found a man who let them in his house, where by nightfall, she died.

Stunned, we try to take care of Patricia's body. The helicopter never comes. It makes little difference now; no airplane in Nepal will carry a body. There is, of course, no way to obtain an autopsy. We finally face the fact that we must dispose of her body ourselves. Burial is not allowed here; the only way is cremation.

The next morning a pitiful procession winds its way around the mountian, led by a Buddhist monk dressed in a red robe, beating a drum, and chanting. Behind him comes a makeshift stretcher carrying Patricia's body in her sleeping bag. The rest of us follow, in mourning. The cremation takes about three hours, during which we all sit in silent meditation, wishing we could turn back the clock, wishing we had done something differently.

COMMENTARY

by Peter Hackett, M.D.

Only an autopsy could establish the cause of death in this unfortunate case. However, there are a number of features that merit discussion. The rate of ascent, the altitude, and time course of onset of the illness are all consistent with acute mountain sickness. The symptoms, however, are atypical. There is no mention of headache, and she appeared to have a gastrointestinal disorder on the first day of illness. The next day she was ataxic, more weak than expected, and a descent of 1500 ft. was somewhat helpful. No other treatment was attempted, and she improved while staying at Pheriche, but never completely recovered. She then developed personality changes, continued to be anorectic and weak despite further descent, acutely deteriorated neurologically, and died, despite oxygen and descent to 10,600 ft.

Clearly, this was not a typical course for AMS and/or cerebral edema. She may have had AMS in the beginning, with her initial improvement due to resolution of this component of her illness. If it were only AMS, the extra time acclimatizing and the further descent should have resulted in continued improvement. There were a number of features compatible with cerebral edema, especially the terminal events. However, cerebral edema of altitude steadily progresses or clears, and always responds to descent. A trial of acetazolamide and/or dexamethasone would have been worthwhile.

The ataxia, extreme weakness and lassitude, perhaps the anorexia and vomiting, and the terminal events point to a primary neurological process. The absence of pulmonary symptoms and rales rules out HAPE. There may have been a concurrent GI illness as well, such as traveler's diarrhea or giardiasis, but this did not directly contribute to her death. We are left with a terminal cerebral event, preceded by a week of fluctuating symptoms, which occurred despite acclimatization time and descent. One explanation is cerebral venous thrombosis, probably as a complication of AMS and mild cerebral edema.

CEREBRAL VENOUS THROMBOSIS AT ALTITUDE

by Peter Hackett, M.D.

Thrombosis of the venous cerebral circulation is gaining recognition as an unusual complication of high altitude. Clinically, it has been associated and confused with cerebral edema. The diagnosis can only be made by diagnostic imaging or autopsy.

The first documented case was a physician I treated in 1978 at Pheriche, Nepal. He had developed symptoms of AMS on his way into Pumori basecamp, but instead of resolving with three weeks acclimatization at the same altitude (17,000 ft.), his symptoms waxed and waned. His teammates described persistent ataxia and insomnia. He declined to descend, and on day 20, he suddenly became worse. He began to descend on foot to Pheriche, but was too weak, so he returned to basecamp. He lapsed into a coma that night. When I saw him the next day, he was deeply unconscious with papilledema and decorticate posturing. Pulmonary edema was also present. He died, despite aggressive therapy and evacuation to Kathmandu (4200 ft.). Autopsy showed that the pulmonary edema had cleared, but there was gross cerebral edema, and thrombosis of the posterior and transverse dural sinuses and of the veins in the subarachnoid space. There were extensive areas of hemorrhagic infarction throughout the brain. Pathologists speculated that the venous thromboses were a primary cause of death, contributing to the hemorrhages and edema, but this cannot be established with certainty. The case was published in reviews by Dickinson[1] and Song et al.[2]

Subsequently, three other documented cases have been published. The most carefully documented case, studied by Fujimaki et al[3], is that of a 27 year old climber. A lesion showed on CT scan and cerebral angiography, which proved upon craniotomy to be a cortical venous thrombosis with secondary hemorrhagic infarction. This patient made a complete recovery. A 38 year old trekker in Nepal died of apparent altitude cerebral edema after evacuation to Kathmandu.[1] Autopsy revealed no edema of the brain, but there was extensive antemortem thrombus of the subarachnoid veins, and hemorrhages and petechiae in the cerebral hemispheres and grey areas of the brain. Aoki et al[4] reported a cerebral infarction in a 31 year old climber. CT scan showed a low density lesion of the parietal lobes with high density in the left, almost identical to the case of Fujimaki. Although pathologic material was not obtained, this was attributed to venous thrombosis. Ten other autopsies of altitude illness showed evidence of cerebral edema and cerebral hemmorrhages but no venous thromboses.[2] The occurrence of venous thrombosis with altitude cerebral edema is thus inconsistent and apparently not due to cerebral edema.

The clinical presentations of these cases were somewhat varied, but three were marked by a prolonged course of fluctuating neurological signs and symptoms, similar to the case presentation in this newsletter. The Japanese climber reported by both Song and Fujimaki was well-conditioned and healthy. Descending from the summit of Lhotse (which he had climbed with oxygen), he developed a headache and anorexia. The headache cleared with further descent, and his exam was normal. The next day, the headache returned, and he became drowsy and ataxic. He was treated with steroids and oxygen, with some improvement, but headaches and drowsiness fluctuated over the next ten days. There were never any pulmonary symptoms or signs. After his return to Japan, CT and craniotomy established the diagnosis.

The pathophysiology of cerebral venous thrombosis at altitude is unclear. The Japanese authors attribute the cause to polycythemia, plus a disturbance of the cerebral circulation from the increased intracranial pressure of AMS. In these cases, the finding of polycythemia was inconsistent; two were not at altitude long enough to become polycythemic. Another contributing factor might include dehydration. Thromboses of the pulmonary and cerebral circulations, as well as hemorrhages, are consistent pathological findings in high-altitude illness deaths. Studies of coagulation and fibrinolysis at altitude have reported conflicting results; the issue is not settled (see Heath and Williams[5] for a thorough discussion).

These cases should remind us that not all neurological symptoms at high altitude are due to cerebral hypoxia, AMS, or cerebral edema. The diagnosis of cerebral venous thrombosis, as well as CVA, arterial-venous malformation, aneurysm and other cerebral circulatory disturbances should be considered in persons with persistent or fluctuating neurological symptoms that do not respond to descent and dexamthasone. Appropriate management is evacuation to a hospital for complete evaluation.

References:

[1]Dickinson, J., D. Heath, J. Gosney, and D. Williams. Altitude-related deaths in seven trekkers in the Himalayas. *Thorax.* 1983; 38:646-656.

[2]Song, S.Y., T. Asaji, Y. Tanizaki, T. Fujimaki, M. Matsutani, R. Okeda. Cerebral thrombosis at altitude: Its pathogenesis and the problems of prevention and treatment. *Aviat. Space Environ. Med.* 1986; 57:71-76.

[3]Fujimaki, T., M. Matsutani, A. Asai, T. Kohno, M. Koike. Cerebral venous thrombosis due to high-altitude polycythemia. Case Report. *J. Neurosurgery.* 1986; 65:148-150.

[4]Aoki, T., T. Tsuda, T. Onizuka, et al. A case of high altitude illness with pulmonary and cerebral infarction. *Jap. J. Thor. Dis.* 1983; 21:770-4.

[5]Heath D., Williams Dr., *Man at High Altitude.* Churchill Livingston, Edinburgh, 2nd Ed., 1981.

HEPATITIS B VACCINE AND THE TRAVELER

by Karl Neumann, MD

Travelers not immune to Hepatitis B virus should consider vaccination prior to travel. Immunity, generally determined by the presence of HbsAg (surface antigen), can be determined by a blood test available from most laboratories.

The prevalence of Hepatitis B carriers varies from one region of the world to another:

Geographic area	*% of population carriers*
Northern Europe, North America, Australia, New Zealand	1%
Central and Eastern Europe	5%
Southern Europe, Central and South America	5 - 10%
Africa, Asia, and the Pacific	20% or more

Southeast Asia and sub Saharan Africa are areas with especially high risk for transmission of Heptatitis B.

The vaccine is particularly important for the following travelers:

- medical personnel providing health care.
- trekkers and adventurers living with local populations.
- travelers who eat in local restaurants and street stalls.
- long-term travelers (more than three weeks) to endemic area.

Some authorities suggest that all non-immune travelers should be vaccinated, even short-term visitors who stay and eat in luxury hotels. The reason is: each year, some travelers who claim to follow all the preventive health rules have converted from negative HbsAg to positive after visiting areas of high incidence.

Certain "high risk"situations for contacting Hepatitis B have been identified:

1) Accupuncture in Hong Kong. Needles are re-used and may be inadequately sterilized in alcohol or other cleansing solutions; autoclaving is not generally used. Tatooing, barber shaving, ear piercing, and dental treatments may carry similar risks.

2) Blood transfusions. Blood tests for HbsAg in donor blood are now available throughout the world, but may not be used in a specific area. Limit transfusions to life and death situations. If feasible, postpone surgery and consider evacuation.

3) Injections. Medications are more often given by injection in developing countries than in America. Needles may be reused after inadequate sterilization, so check for disposable needles. Before departure, obtain any immunizations required for entry by all countries on the itinerary. Otherwise, injections at the airport may be demanded as a condition of entry.

4) Sexual activities. Sexual contact with local persons increase the risk of contacting Hepatitis B in areas with high incidence of infection. Additionally, these areas (Southeast Asia and sub Saharan Africa) have a high incidence of venereal disease, with organisms resistant to the usual antibiotics used in the United States. Also, in sub Saharan Africa, there is a high incidence of AIDS (Acquired Immune Deficiency Disease); and in this area, AIDS is a heterosexual disease.

In the tropics, do biting insects play a role in the spread of Hepatitis B? Results of investigations are conflicting, according to Arie Zuckerman, Director of Medical Microbiology, London School of Hygiene and Tropical Medicine, and of the WHO Collaborating Centre for Reference and Research on Viral Heptatitis. Hepatitis B surface antigen has been detected in several species of mosquito and in bed bugs that have either been trapped in the wild or fed experimentally on infected blood. But no convincing evidence of multiplication of the virus in insects has been obtained. However, mechanical transmission of the infection via an insect's biting parts is a possibility.

Hepatitis B vaccine is extremely safe. It does not contain "whole" or live virus; they are inactivated during multiple preparation steps. There is no evidence that AIDS can be transmitted by the vaccine. But the vaccine is not suggested for people who are immunosupressed and who are pregnant. The HBV vaccine is approved by the FDA and endorsed by the World Health Organization. A genetically engineered vaccine is now available.

Several studies show that protective titers of anti-HBsAg antibodies, as defined by a fall in the incidence of HBV illness, occur in over 80% of recipients. Response after vaccination depends on age and prior health status. The exact duration of protection is presently unclear.

RESOURCES FOR INTERNATIONAL HEALTH

National Council for International Health

The National Council for International Health (NCIH) is a non-profit professional organization which works to strengthen United States participation in international health. Their role is to promote support, coordinate information and activities, provide training and technical assistance, and develop networking among private and governmental agencies involved in providing services outside the U.S. They publish a monthly newsletter which reports on programs, publications, legislation, conferences, and progress in the field of international health. Infomation on membership benefits can be obtained by writing to NCIH, 1101 Connecticut Ave, N.W., Suite 605, Washington, D.C. 20036.

NCIH also publishes a directory of U.S. based agencies involved in international health assistance. Agencies are listed alphabetically with descriptions of activities and regions of involvement. In addition, agencies are listed by geographic region and by categories of assistance.

AMERICAN SOCIETY OF TROPICAL MEDICINE

The American Society of Tropical Medicine and Hygiene is devoted to research, clinical practice and public health aspects of tropical medicine and parasitology. The clinical committee of that society has prepared a directory of its members who offer consultative service in medical parasitology and travelers' health. This directory is a resource for those seeking local experts in exotic diseases and also indicates centers where unusual immunizations such as yellow fever or Japanese encephalitis can be obtained. Information about the society or about the directory can be obtained from Leonard C. Marcus, VMD, MD, Tufts University, 200 Westboro Road, North Grafton, MA 01536.

Dr. Marcus is one of the consultants, and is a member of the WMS.

MOUNTAINEERING & ROCK CLIMBING ACCIDENTS IN THE GRAND TETON NATIONAL PARK

by L.C. Schussman, M.D.

We studied the climbing and mountaineering accidents in Grand Teton National Park (GTNP) over the ten years from 1971 through 1980.[1] When this study was first published the medical, epidemiological, and climbing literature included only one study of the epidemiology of climbing accidents. In that paper, Wilson described accident data from Denali for 1976.[2]

In order to evaluate climbing data in a meaningful way, it is necessary to know the number of people at risk for having an accident, which is the total number of climbers. This data is often difficult to obtain. The GTNP offered a unique study opportunity because of its enforced regulation that all climbers must register.

METHODS

GTNP registration data, GTNP accident records, death inquisition reports, and the American Alpine Club summaries were computerized to perform multiple cross-tabulations. For each accident, the following information was recorded: date, time, day of week, people involved, age, sex, place of origin, previous climbing experience, route, weather, pre-accident conditions, precipitating factors leading to the accident, details of the accident itself, details of the search and rescue efforts, rescue costs, injuries, and the severity of injuries. We adopted the injury severity score, developed by Baker et al [3] and found to be closely related to clinical outcomes.

RESULTS

71,655 climbers registered during the ten year study period. Of these, 53,274 (74%) completed their climbs. 144 accidents required rescue by park rangers. 158 people were involved in these accidents, and 247 separate injuries were diagnosed. There were 30 deaths.

The annual accident incidence rates (number of accidents per time per number of people at risk) remained stable over the study period at two accidents/1,000 climbers/year. The case fatality rate (number of fatalities per accident per year) also remained fairly stable over the study period at 0.10 to 0.30. Rescue costs increased steadily. The total cost was $144,205, an average of $1,000 per accident and approximately $2 for each registered climber.

Not surprisingly, lead climbers and steeper routes accounted for more accidents than following climbers and those on less steep pitches. 37% of the accidents involved falls while climbing rock pitches. 26% occurred while traveling on snow. 20% of all accidents occured because climbers were unable to self arrest on snow fields and slid into obstacles.

Climber error was involved in 39% of the accidents. Errors were attributed only after careful investigation by climbing rangers or their boards of inquiry. Failure to use adequate equipment (usually no ice axe) and failure to place adequate protection where it was clearly indicated each accounted for 25% of the error-related accidents. An additional 18% involved climbers who were off route, and 14% involved failure of an anchor. Ignoring developing storms, rushing, and failure to heed ranger warnings (especially of storms or avalanche danger) each accounted for about 5% of error-related accidents.

Climber experience information was available for only 69 of the 144 accidents. Of these, 58% had no experience or had climbed less than one year.

Among the 247 injuries, there were 83 fractures, 47 lacerations, 59 significant contusions and abrasions, six cases of hypothermia, four lightning burns, and three cases of frostbite.

Most injury severity scores were in the moderate category, characterized by severe lacerations or distal extremity fractures.

More than half of the 30 fatalities involved snow or ice travel, despite the fact that climbers spent much less time on snow than on rock. Avalanches accounted for ten deaths, and faulty self-arrest or out of control glissading for the others. Clearly, time spent on snow was associated with higher risk of injury relative to time spent on rock.

We are currently in the process of updating this information with data from 1981 to 1986, and are also making plans for prospective studies.

I would like to establish a special interest group within the WMS to exchange research data and to collaborate on a study of mountaineering and rock climbing accidents. Our interest centers on the epidemiology of these accidents — their causes, persons involved, outcomes, rescue efforts, and costs.

Anyone sharing research interest in this area please contact:

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Assistant Professor, University of Utah
Dept. of Family & Preventive Medicine
1750 Lake Street
Ogden, Utah 84401

[1]Schussman, LC, Lutz, LJ. Mountaineering and Rock Climbing Accidents. *Physician and Sportsmedicine;* 10:53-61, June 1982.

[2]Wilson, R., et al. Death on Denali. *West J Med;* 128:471-6, June, 1978.

[3]Baker, SP, et al. The Injury Severity Score. *J Trauma;* 14:187-196, March, 1974.

SEARCH AND RESCUE

DATA-BASED FIRST AID

by Warren Bowman, MD

This article was first published in RESPONSE! magazine and is reprinted with permission of JEMS Publishing Company.

A few months ago, it occurred to me that there is a lack of hard data on wilderness accidents and illness. However, there are many published lists for Search and Rescue (SAR) personnel of potential problems, medical procedures, and equipment. If good data were obtained and analyzed, the requirements for SAR training and equipment could be determined accurately and subjected to the criterian of minimum weight and cost, maximum use and durability, multiplicity of use, and adaptability for improvization.

I found the best data from studies of mountaineering accidents. Results from the following references were analyzed, and listed in the table below.

1. *Accidents in North American Mountaineering,* published by the American Alpine Club and the Alpine Club of Canada, for the six year period 1980-85. There were 353 accidents, not including 32 miscellaneous fractures. There were 183 deaths, categorized as victims either found dead or who died shortly after being reached, for a total of 536 incidents altogether.
2. Mclennan, J.G., MD and Ungersma, J. MD, "Mountaineering accidents in the Sierra Nevada," *Am. J. Sports Medicine 11:3,* p. 160-163, 1983. 215 accidents and 17 deaths, all on Class V climbs. The authors estimated that more than 60% of injuries were associated with acute mountain sickness and over 10% with hypothermia.
3. Schussman, L.C. MD, Lutz, L.J. MD. "Mountaineering and Rock-Climbing Accidents," *Physician and Sports Medicine, 10:6,* p. 53-61, 1982. There were 144 accidents and 30 deaths in Grand Teton National Park from 1970-1980. (Discussion in current issue of *Wilderness Medicine.)*

Table 1 lists the relative frequency of specific problems. Table 2 lists equipment required to manage these. Tables 3 and 4 separate the equipment into special categories for consideration. Comments are solicited.

TABLE 1

Description of Accident/Incident	Percentage of Total Accidents/Incidents (%)		
Death	34.0	8.0	17.0
Fracture of leg or ankle	11.4	9.0	7.4
Multiple injuries (usually serious)	10.0	?	3.1
Head injury	7.0	2.0	4.7
Frostbite	6.7	5.0	0.7
Upper extremity fracture (arm, wrist, shoulder, clavicle)	3.4	18.0	3.5
Hypothermia	3.2	8.0	2.3
Pulmonary edema	3.0	7.0	?
Vertebral fracture	3.0	4.0	1.6
Laceration(s)	2.4	?	12.5
Hand fracture	2.0	1.0	0.3
Knee sprain, dislocation	2.0	11.0	0.7
Femur fracture	1.5	2.0	3.1
Foot fracture or crush injury	1.3	3.0	1.5
Contusion	1.0	?	7.4
Ankle sprain	1.0	14.0	2.3
Cerebral edema	0.7	1.0	?
Shoulder dislocation	0.7	2.0	2.7
Hip or pelvis dislocation, fracture	0.5	4.0	?
Heat injury	0.5	0.0	1.5
Lightning injury	0.5	0.0	0.0
Facial injuries	0.5	?	5.8
Bites, stings (including anaphylactic reaction)	0.4	0.0	0.0
Pneumonia	0.1	?	0.0
Chest or rib fracture, bruise	0.05	3.0	1.9

TABLE 2
Analysis of injuries by first aid equipment required

	Category	% of Total	First Aid Equipment Required
1.	Death on arrival or shortly thereafter	34.0	None
2.	Leg or ankle fracture	11.4	Splint (long or short leg), pain meds. If open, see "Laceration"
3.	Multiple injuries (various combinations of head, chest, abdominal and extremity injuries	10.0	Dressings, bandages, airways, suction, bag-mask, splints, cravats, I.V.'s or MAST, oxygen, KED, pain meds, cricothyrotomy set, chest tube, Foley catheter, urinal, vasoline gauze
4.	Head Injury	7.0	Dressings and bandages, extrication collar, airways, suction, KED, oxygen, Foley catheter, urinal, I.V.'s
5.	Frostbite	6.7	Stove, pot, thermometer, dressings
6.	Arm, wrist fracture	3.4	Short arm splint, sling, pain meds. If open, see "Laceration"
7.	Hypothermia	3.2	Rewarming device, stove, shelter-building equipment or tent, sleeping bag, I.V.'s
8.	Pulmonary edema	3.0	Descent, oxygen, Lasix
9.	Vertebral fracture	3.0	Extrication collar, KED, pain meds
10.	Laceration	2.4	Dressings, bandages, sterile saline for irrigation, antiseptic soap
11.	Hand fracture	1.3	Splint, wad of gauze, sling, pain meds. If open, see "Laceration"
12.	Knee sprain, dislocation	2.0	Cravat, Ace bandage, long leg splint, Baggie for snow
13.	Femur fracture	1.5	Traction splint, pain meds, MAST or I.V.'s. If open, see "Laceration"
14.	Foot fracture or crush injury	1.3	Splint (long or short leg), pain meds. If open, see "Laceration"
15.	Contusion	1.0	Plastic bag for snow, splint (if extremity)
16.	Ankle sprain	1.0	Adhesive tape or Ace bandage, plastic bag for snow
17.	Cerebral edema	0.7	Oxygen, descent, Decardron, airways, suction
18.	Shoulder dislocation	0.7	Cravats for sling, pain meds
19.	Hip, pelvis fracture	0.5	Backboard (Thompson or Stokes litter), pain meds, Foley catheter, urinal
20.	Heat injury	0.5	Poncho for shade, cold water
21.	Lightning injury	0.5	Dressings and bandages, antiseptic soap, pocket mask, airways, oxygen, monitor-defibrillator, I.V.'s
22.	Facial injuries	0.5	Airways, oxygen, dressings, bandages
23.	Bites, stings (including anaphylaxis)	0.4	Adrenalin, Benadryl (see "Laceration")
24.	Pneumonia	0.1	Antibiotics, oxygen
25.	Chest, ribs bruise	0.05	Adhesive tape, Ace bandage

TABLE 3
Projected SAR First Aid Equipment Based on Tables 1 & 2

Most Useful	Useful But Heavy, Expensive or Needing Special Expertise	Less Useful
Heat generating device (heat pads, Heatpak®, Applinc® Heat Treat®)	Oxygen	Monitor-Defibrillator
Long leg air splint	IV solutions, sets, needles, TK	Instant glucose
Dressings, bandages, Kling® tape, cravats, Ace®	MAST	Short leg air splint
Sterile irrigation solution (IV bag of 0.9% saline)	Suction device (mechanical)	
Germicidal soap	Bag-mask	
Pain medications	Foley catheter, gloves, lubricant, clamp and plug	
Arm/hand splint (wire, SAM®)	For high altitude: Diamox®, Decadron®, Lasix®	
Airways	For allergic reactions/anaphylaxis: Bee Sting Kit (Epipen®, Benadryl, epinephrine)	
Pocket mask	For pneumonia and other infections: Antibiotics	
Extrication collar (can use SAM®)	Cricothyroidotomy set	
Portable, short backboard (KED, Oregon Spine Splint®)	Chest tube set (Heimlich valve, McSwain dart)	
Flashlight		
Notebook and pencil, tags		
Urinal or pee bottle		
Thermometer (low-reading for cold weather/high altitude; regular for hot weather)		
Litter		
Plastic bags (for snow, sprain and contusion treatment)		
Light traction splint (Sager® or Kendrick Traction Device®)		
Suction device (bulb or syringe)		
Scissors		
Lubricant for thermometer		
Alcohol sponges		
Seam ripper		
Safety pins		
20 cc syringe for irrigating		
Vasoline gauze		

TABLE 4
Rescuer's Personal Gear of Use

- Stove, gas, cooking pot
- Food and hot drinks
- Tent, Bivouac Sac
- Extra clothing
- Poncho
- Ice axe, ice hammer, ski poles, ensolite, Thermarest® (use for splint improvisation)

WELCOME NEW MEMBERS

Abbuhl, Stephanie, M.D.
Agness, Mark S.
Allen, Robert G. Jr., M.D.
Argenyi, Zsolt B., M.D.
Arnone, Alan J.
Ashcraft, Walter C., M.D.
Bard, Gary W.
Belding, William A.
Belknap, Harold Jr., M.D.
Benedict, William L.
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Blitz, Marilyn
Bottorff, Melvin K., M.D.
Breault, Joe M.D.
Brizgys, Romuladas V., M.D.
Bryan, Henry G., M.D.
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Doherty, Elizabeth, M.D.
Downing, Peggy, M.D.
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Ellsworth, Allan, Pharm.D.
Etges, Thomas J.
Fisher, Bob B., M.D.
Freedman, Jonah S., M.D.
Freedman, S. Robert, M.D.
French, Steve, M.D.
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Gieser, Dick, M.D.
Goldman, Jay M., M.D.
Gordon, Kilbourn, III, M.D.
Grishaw, William D., M.D.
Halbert, R.J., M.D.
Hendricks, Everett D., M.D.
Hobson, Bartholomew, M.D.
Horsmann, Robert, RN
Houle, Richard, M.D.
Hull, Daniel J., M.D.
Hunt, Dexter W.
Huss, Chuck, M.D.
Jacobsen, Sheldon, M.D.
Jenkins, James H., M.D.
Jui, Jonathan, M.D.
Kauffman, Lawrence, M.D.
Kelly, Joseph E., M.D.
Kittleson, Kevin, M.D.
Kleinsteuber, Ken, M.D.
Kowalski, Peter C., M.D.
Kramer, Samual N., M.D.
Kraus, G. Thomas, M.D.
Kumaki, David James, M.D.
Laubscher, Frederick A., M.D.
Lippman, Michael, M.D.
Lobis, Robert A., M.D.
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LIMITATIONS OF SWIFT WATER RESCUE

A Case Report of a Drowning on the Upper Kings River

by Eric A. Weiss, M.D.

An alarming increase in the number of deaths from kayaking occurred in the United States during 1986. Increasing popularity of the sport, combined with a cavalier attitude toward white water contribute substantially to the safety record. Entrapment, when a kayaker is pinned under water, is the cause of most fatalities. Seasoned paddlers rely on skill and experience to avoid such situations. But, they use fewer safety precautions while pushing the limits of navigable rivers. Ironically, it was not the novice, but the expert boaters who died on the rivers last year.

On Sunday, August 10, Bob Porter died while kayaking the Garlic Falls section of the Upper Kings River. Porter, a thoughtful graduate of Harvard University, was respected for his skills at reading water and for his exceptional judgement on rivers. He had pioneered many new runs in California and was affectionately known in the paddling community as "Big Water Bob." The circumstances of his death emphasize the inherent limitations and frustrations of swift water rescue.

The Upper Kings River flows through a remote wilderness canyon and is rated a solid class V (class I - V) because of its steep gradient, technical rapids, and inaccessibility. The accident site was a class V rapid 4½ miles from the put-in at Kings Canyon National Park. Porter was attempting to negotiate a drop which required paddling to the right of a large boulder in the middle of the passage, then dropping off a five foot ledge into a short pool above a second, more difficult rapid. The move itself was not particularly difficult, but on the approach, a submerged rock, above the boulder, caught Porter's boat, deflecting him to the left. The current washed him sideways against the boulder with the bow of his boat pointed to the left. At this point, he had only two options. He could wash backwards down the right side, where he wanted to be, or attempt to go forward and run the left side.

Porter went to the left, and as he plunged off the drop, the front of his kayak stuck between some rocks on the bottom of the river. He was pinned in a near vertical position with the river pouring off the left side of the boulder onto his back. A rope was thrown from the left shore which he managed to grab. Holding onto the rope, he was pulled from his kayak into the water. Once in the water, he attempted to swing his feet downstream to assume the correct position for swimming through a rapid. In so doing, his right leg became trapped beneath an undercut rock. He went under the water between two boulders and did not resurface. Multiple attempts were made to reach the spot where he was pinned, but the combination of slick rocks, vertical canyon walls, and a formidable rapid made it impossible. Not until many weeks later, after the runoff from the Sierra snowpack had receded, could his body be extricated.

Technical rope-based systems are an important adjunct for rescues in the mountains or on rock faces. Their application to swift water rescue is severely limited by short submersion survival times. Consequently white water rescue options should be simple, requiring minimal preparation. The most important component may not be "high-tech" equipment, but past experience and understanding water dynamics. Porter was accompanied by three other world class kayakers; despite their cumulative years of experience, they could find no means of rescuing their friend. Perhaps the only lesson from this frustrating death is the inherent risk of whitewater sports and a reminder of the impersonal forces in the wilderness. After an accident has occurred, sometimes no one can bring back what the River Gods have taken.

NEW MEMBERS (Cont'd)

Mackie, Laura L., M.D.
MacLeod, Caroline L.L., M.D.
Marcus, Leonard C., V.M.D., M.D.
Martinez, Ricardo
Mayer, Nancy
McDonald, Alison J., M.D.
Meredith, Thomas S.
Miller, Harry, M.D.
Minyard, Frank, M.D.
Moundalexis, Athena M., M.D.
Muelleman, Robert L., M.D.
Murdock, Carol
Myers, C. Arthur, D.O.
Myers, Gary J., M.D.
Neft, Martin, M.D.
Newberger, David S.
Ogilvie, W.E. III, M.D.
Oshinsky, Arnold, M.D.
Peloso, Ole A., M.D.
Poliner, Jay R., M.D.
Raney, Gus A., M.D.
Reul, Charles G., M.D.
Rischitelli, D. Gary
Rodriguez, Luz E., M.D.
Roe, Chester T., M.D.
Roloff, James S., M.D.
Rozin, Joda A.
Russman, Burton A., M.D.
Sathre, Howard P.
Schaffner, Nancee, D.O.
Schiller, Andrew, M.D.
Schlatz, Ivan, M.D.
Schmid, Heinrich, O.E., M.D.
Schuetz, John N., M.D.
Segars, Scott, M.D.
Selliger, Edward H., Jr., M.D.
Singer, Robert P., M.D.
Sloan, Robert
Southworth, Michael, M.D.
Speer, Gregory C., M.D.
Spitze, Randall L.
Stein, Bob, D.O.
Steinman, Mark, M.D.
Stepaniak, Philip C., M.D.
Stewart, Jerry, M.D.
Stonecypher, David D.
Strait, W. Frank III, M.D.
Taylor, Randy, M.D.
Tek, Deniz Server, M.D.
Thomas, Frank
Ullrich, Reinhold, M.D.
Vassallo, Susi, M.D.
Walker, Angela T., M.D.
Weinstein, Harold, M.D.
Wenner, Karl C., M.D.
Wiggins, Michael
Wilson, Mary E., M.D.
Wilt, Timothy, M.D.
Wingo, L. Earl, M.D.
Winterwood, Charles, M.D.
Woodbury, Derrik, M.D.