Wilderness Medicine Letter

The official newsletter of the Wilderness Medical Society

An international nonprofit professional association serving the medical interests of the outdoor and wilderness community.

Vol. 7, No. 3 Edited by Howard Backer, M.D. July 1990

SOCIETY BUSINESS

New Board Members

Joseph Serra, M.D., an orthopedist from Stockton, California, and Susan Snider, M.D., a family practitioner from Spruce Pine, North Carolina, were elected to serve on the Board of Directors. They will begin their three-year terms at the annual meeting in Snowbird, Utah.

IN THIS ISSUE....

Journal of Wilderness Medicine

Society members should have received the first two issues of the *Journal of Wilderness Medicine* by this time. Please notify the WMS office in writing if you have not received your copies.

Paul Auerbach, M.D., co-editor of the *Journal of Wilderness Medicine,* requested funds to expand the journal by sixteen pages. The response to the *Journal* has been very good and its editors are in the enviable position of having an unacceptable publication delay due to the number of manuscripts accepted. Considering the importance of the *Journal* and the reasonable rate offered by the publisher for the extra pages, the Board approved this expense (pending review by the Finance Committee).

Research Grants

The board reviewed seven submissions for medical student and resident research awards and selected only one, leaving four grants uncommitted. Since selection criteria for these awards is not excessively rigid, either the monetary award is not large enough (which is probably not a factor) or the announcements are not reaching prospective applicants.

The Awards Committee will improve marketing. Members of the Society should encourage students and residents with whom they work to formulate research projects in the area of wilderness medicine and apply for these grants. Details can be obtained from the Society office and regular announcements will appear in the Newsletter.

WMS Research Award Recipient

Craig Lambrecht, M.D. a resident in the Department of Emergency Medicine at the Medical College of Wisconsin, was awarded $2,500 for a project investigating "Hunting related illnesses and injuries." His hypothesis is that firearm injuries contribute only a small percentage of medical problems from hunting. Although environmental factors probably play an important role in hunting injuries, he predicts that there is no difference in injury rates between resident and out-of-state hunters. In addition, he will seek to correlate alcohol consumption with medical problems among the hunters (probably no surprises there). Data will be gathered from emergency departments and the Department of Fish, Wildlife and Parks in Montana.

Newsletter Contributors

Contributions by WMS members to the newsletter are solicited. The more participation, the more interesting and informative the newsletter will become. A broad spectrum of subjects, ideas and styles can be accommodated. Those interested in writing should contact the editor. Members are encouraged to report pertinent projects or research in which they are involved. Publications and lectures can be edited for the newsletter.

Contributing editors are also needed for the newsletter. Basic writing skills, time and interest are the main prerequisites. Anyone interested should contact the editor to discuss details.

Eric Weiss, M.D., will work as an associate editor.

Thanks to Larry Johnson, M.D. (Cincinnati, OH) for assistance in proof-reading this newsletter.

WILDERNESS MEDICINE LETTER
is published quarterly by the
Wilderness Medical Society
Send inquiries or comments to:
P.O. Box 397
Point Reyes Station, CA 94956
(415) 663-9107
Membership information/application is located on the last page.
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Items available from the Society

The following can be ordered from the WMS office. *(Price includes postage)*

Three Ring Newsletter Binders (with printed cover and spine)	$7.00
Reprints of WMS Position Papers	3.00
Past Issues of the Newsletter	1.50-2.50
WMS Patch (with logo)	4.00
Cap (emblem on front); blue, beige	10.00

Winter Meeting

The WMS will sponsor its first Winter Wilderness Medicine meeting, February 13-16, 1991, in Crested Butte, Colorado. This resort was chosen for its spectacular mountain beauty with excellent skiing and winter sports, its remote yet accessible location, comfortable facilities and the relaxed ambiance.

Warren Bowman, M.D. is the program chairman. Warren is medical advisor for the National Ski Patrol and author of *Outdoor Emergency Care,* the textbook for their Winter Emergency Care course; chairman of the medical committee for the National Association for Search and Rescue (NASAR); and ventures regularly into the winter backcountry. He will be assisted by a program committee consisting of Drs. Chris Moore, Sherilyne King and Doug Gentile.

This conference will be completely different from our summer Annual Meeting. It will focus on cold and winter wilderness activites. There will be an even greater emphasis on participatory workshops in the field.

Please join us at this conference, which will offer the best of wilderness medicine, collegiality and winter sports.

Wilderness EMT Advisor

Mel Otten, M.D., a member of the WMS Board of Directors and chairman of the Prehospital Care Committee was appointed to sit on a program advisory board for the NASAR Wilderness Medicine program. This advisory board will consist of representatives from thirteen national organizations that have a direct interest in the wilderness EMT training program that is being refined and sponsored by NASAR. Their function will be to review the program annually, to comment on any proposed curriculum changes, and to advise NASAR Medical and Education Committees as requested. The Prehospital Care Committee chaired by Dr. Otten is currently writing standards to be applied to wilderness emergency care. It is anticipated that these standards will be highly regarded and widely adopted, given the medical expertise and nonproprietary interest of the Wilderness Medical Society.

PRESIDENT'S COLUMN

Blair D. Erb, M.D., F.A.C.P.

DOES THE WILDERNESS MEDICAL SOCIETY HAVE A RESPONSIBILITY IN WILDERNESS PUBLIC HEALTH EDUCATION?

The wilderness currently is enjoying an enormous wave of popularity. As a result, there has been rapid growth in the number of people seeking wilderness experience. Unfortunately, skill and knowledge of participants in many instances has not kept pace with their enthusiasm.

Not all emergencies, however, involve only neophytes. An experienced wilderness venturer may become overextended or, because of fatigue or other physical factors, may make a poor decision that leads to problems. One way of preventing such problems is by guiding individuals away from activities beyond their capacity. This is an important role for the physician and for the group leader. Articles in this issue of the *Wilderness Medicine Letter* discuss factors that might limit an individual's performance or tolerance for wilderness activities.

Even under the best circumstances, medical emergencies may occur and, when they do, the outcome may depend on the medical and first aid skill of individuals closest to the problem. Optimally, all participants should have some knowledge of medical concerns associated with harsh environments so that they can prevent emergencies from arising in the first place or manage the emergencies when they occur.

This approach is a form of public health education. But is it possible to educate the public adequately to prevent environmental emergencies?

Issues to consider in wilderness public health education can be divided into four categories:

1. characteristics of the population at risk;
2. nature of the risk;
3. mechanisms for educating those who are vulnerable;
4. resources for such an ambitious undertaking.

Characteristics of participants at risk are the easiest to define; these have been addressed by the classification outline. It is more difficult to define the nature of the risks. What risks are inherent in specific ventures? Reliable, accurate sources of risk identification must be developed.

How should educational interventions be developed that influence the health and safety of participants in wilderness ventures? Some have suggested regular newspaper or magazine columns sponsored by the Wilderness Medical Society. Others feel that formal education of nonmedical wilderness leaders or even formal lay education programs may help. Monographs or videos on "Wilderness Health" are easy to prepare, but experience has shown that single-page, printed "flyers" often serve only to litter the landscape.

From a purely practical perspective, we can provide only what our resources allow. Are there extra resources that can be tapped, such as grants, corporate gifts or contributions?

Issues relating to the Wilderness Medical Society's responsibility for public health in wilderness activities are complex. We ask ourselves, "Does the Wilderness Medical Society have a responsibility in wilderness public health education?" If so, "How do we go about it?" Please let us hear from you.

MEMBER'S PROFILE:

KENNETH W. KIZER, M.D.

by Howard Backer, M.D.

Throughout his career, Dr. Kenneth W. Kizer has pursued a keen interest in environmental medicine. He has been instrumental in establishing wilderness medicine as a valid field of study. In addition to his work as Director of the California Department of Health Services, he manages to publish prolifically in the area of environmental medicine. His curriculum vitae would fill more than one newsletter; somehow, he accomplishes the work of five people.

While born in Indiana, Ken spent much of his youth in a poor rural area on the central Oregon coast. Most of his free time was spent hiking in the mountains and hunting or fishing to put food on the table. At the age of 12, when his widowed mother died, he lived in a succession of foster homes in Oregon and California, and finally went to Reno, Nevada. There, among other outdoor activities, he tried the cowboy life, horseback riding and roping deer from the wild herds that wintered near town. In high school Ken applied himself and excelled in academics and extracurricular activities.

Because of his interest in zoology and animal behavior, Ken had considered studying veterinary medicine. However, he entered Stanford University initially intending to study law. But his interest in biology kept him vacillating. He spent one year doing research with monkeys and, at one point, arranged to study chimpanzee behavior in Africa with Jane Goodall. Also while at Stanford, Ken learned how to scuba dive, which rekindled his childhood fascination with marine biology. Finally, these interests led him to study medicine.

For medical school, Dr. Kizer chose the University of California, Los Angeles, where he entered a joint MD/MPH (Master's in Public Health) program that allowed him to pursue an interest in international health. He also found time to take classes outside the medical school on topics such as shark behavior and exercise physiology.

With the military draft threatening (because of a single digit draft lottery number), Ken joined the Navy Reserves during medical school, hoping to pursue diving medicine. After a rotating internship at the Naval Regional Medical Center in Portsmouth, Virginia, he completed the U.S. Navy Undersea Medical Officer course and graduated from the U.S. Navy School of Diving and Salvage. From 1977-1980, Dr. Kizer served as a Navy Submarine and Diving Medical Officer in Hawaii with multiple assignments, including special operations, harbor clearance, submarines, and recompression chamber operations, along with more traditional emergency medicine at the Navy Regional Medical Center at Pearl Harbor.

He learned to improvise care in various settings. He treated cases in remote areas of the Pacific, like a Japanese fisherman partially eviscerated by a swordfish and a merchant marine captain who nearly exsanguinated from a bleeding peptic ulcer. The recompression chamber in Oahu is the busiest in the world. On average, Dr. Kizer treated 1-2 cases per week of decompression sickness or air embolism (mainly among civilians). One memorable patient had trigeminy, probably from a bubble in a coronary artery; however, the chamber was not equipped to handle a cardiac monitor, so Ken had to monitor the arrhythmia and adjust a lidocaine drip by feeling the patient's pulse during recompression treatment. He has published the results of his extensive experience with diving accidents in numerous reports.[1-5]

After his military service, Dr. Kizer began a radiology residency at the University of California, San Francisco. But, after a year and a half, followed by several months in private practice, he switched to a program in occupational medicine because this was the only medical specialty that formally included environmental medicine. He also continued his practice in emergency medicine. Currently, Dr. Kizer is board certified in emergency medicine, medical toxicology, and preventive medicine (with certification in both occupational medicine and public health, and general preventive medicine).

In 1981, Ken met Dr. Paul Auerbach, who also had an interest in marine medicine. They began to organize dive trips with continuing medical education courses. These seminars have provided the opportunity to travel to remote and exotic places in search of the best diving in the world — in Ken's opinion, this includes the Coral Sea, the Philippines, the Red Sea, and the Galapagos Islands.

The association with Paul Auerbach also led to the formation of the Wilderness Medical Society. Along with Dr. Edward Geehr, an associate of Paul who shares an interest in environmental medicine and medical education, the three decided there should be a non-profit organization to promote knowledge and education in the area of wilderness medicine. They formed the Wilderness Medical Society and divided up the duties. Ed drew the short straw and was "elected" the first president. Ken was our second president and subsequently served as secretary-treasurer. Ken has also been active in the Undersea and Hyperbaric Medical Society, the National Association of Underwater Instructors, and several other similar organizations.

In all his years of diving, Dr. Kizer has only once suffered from decompression sickness. While in the Navy, he rescued a civilian diver at 120 feet, having to bring him straight up to the surface, which placed him right on the edge of the no decompression dive table. He then flew to Oahu in an unpressurized small plane. Right after that, he went running and developed severe shoulder pain about five miles into the jog. One of his most frightening experiences underwater occurred last fall while diving alone in the Caicos Islands. It was dusk and Ken had lost track of his boat. While swimming to the surface to take a sighting, an eight foot shark suddenly sped toward him from out of the darkness. Only when it was a few feet away could he clearly identify it as a non-aggressive Nurse shark.

In 1983, Dr. Kizer was appointed Director of the California Emergency Medical Services Authority. After one year, he became Chief of Public Health Programs at the Department of Health Services. A few months later, the Governor asked him to take over as Director of the California Department of Health Services. At the time of his appointment, Dr. Kizer was the youngest ever director (age 33), and he now has the longest tenure in office. In his current capacity, Dr. Kizer manages a budget of approximately $10.5 billion and 6,000 employees. He is responsible for California's public health programs, the $8 billion Medicaid program, all licensing and certification of health facilities (about 5,500 of them), and toxic substances control. He is currently a member of more than 30 state or national committees and task forces (and still maintains involvement in several medical organizations).

Most of his publications are now in the areas of health policy and public health, especially toxins and AIDS. But Ken encounters and reports many aspects of environmental toxicology and infectious diseases in his work[6-9] and still manages to pursue his own interests in wilderness medicine.[10-16] Overall, Dr. Kizer has authored or co-authored over 170 scientific publications. He is an Associate Clinical Professor of Medicine and Community Health at the University of California, Davis.

At least two or three times a month, Dr. Kizer still works in emergency medicine, commuting from his home near Sacramento to work in the San Francisco Bay Area at Marin General Hospital. Between his shifts, he usually camps in a nearby redwood forest park — an example of how Ken finds time for wilderness in his busy life. He also finds time to spend with his wife, Suzanne, and his two daughters. Their family enjoys skiing and hiking together, and the two girls recently took their first dive trip.

Ken feels that environmental medicine is a strong interest but not a career for him. He does not fantasize on running a dive shop on a tropical island, but he would like to pursue his interest in outdoor and underwater photography. . . . In the meantime, he will continue to advance the goals of the Wilderness Medical Society by promoting a scientific approach to the field of wilderness medicine.

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THE BLOOD GAS IN HYPOTHERMIA: CORRECT OR NOT?

Susi Vassallo, M.D.
(Emergency Department Bellevue Hospital/New York University Medical Center; New York City Poison Control Center.)

Should arterial blood gases in the hypothermic patient be corrected or remain uncorrected for temperature? Although most clinicians have an answer, few can justify their choice. The *uncorrected* value is read directly from the blood gas machine that warms the blood to 37°C and reports what the blood gas values would be in a normothermic patient. The *corrected* value is obtained mathematically from equations that describe the effects of cooling on the pH and gas pressures of blood.

The controversy regarding corrected versus uncorrected blood gas values began in the 1950's, when surgeons first discovered the protective effects of hypothermia during cardiac procedures. The original question was: what degree of ventilation would result in the lowest incidence of central nervous system and cardiac complications in the hypothermic surgical patient? The two solutions to this question represent the two sides of the controversy. One approach is to correct the arterial blood gas values to the temperature of the patient and to keep the corrected pH at 7.40 and the pCO_2 at 40 mmHg. This can occur only by inducing a respiratory acidosis to increase the CO_2 content of blood, since arterial pCO_2 decreases with temperature. The second solution is to use the value from the blood gas machine without changing it for temperature and to maintain the pH and pCO_2 at 7.40 and 40 mmHg respectively, which maintains the same CO_2 content as a normothermic patient with normal ventilation. It results in an apparent respiratory alkalosis if the values are corrected for the temperature of a hypothermic patient.

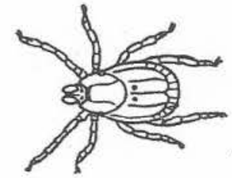
When blood is cooled the pH rises. The pH of water, a neutral reference point, also rises with cooling. This is due to the effects of cold on the dissociation of hydrogen ions. A blood gas reading of pH 7.40, pCO_2 40 at 98.6°F (37°C) would become pH 7.72, pCO_2 14 when corrected to 61°F (16°C). (Remember that the blood gas machine is designed to warm the blood to 98.6°F, indicating what the patient's acid-base status would be at normothermia.) To correct these values a set of in vitro observations of temperature effects on pH and gas pressures are applied to the *in vivo* pH and gas pressures. Since the reference point (the pH of neutral water) has also changed, the clinical meaning of the corrected values is unfamiliar. The uncorrected values are normalized to a reference point that is clinically relevant: the pH of neutral water at normal body temperature. The physician who uses the corrected values in the example above and maintains the pH and pCO_2 near normothermic values (pH of 7.47 and pCO_2 of 40) must induce a significant respiratory acidosis (pH of 7.18, pCO_2 of 111, uncorrected). Thus, the physician attempting to maintain the corrected pH near 7.40 must hypoventilate the patient and risk impairment of oxygenation.

Recent cardiovascular surgical literature suggests that maintenance of adequate ventilation decreases the incidence of myocardial injury and ventricular fibrillation. Respiratory acidosis depresses myocardial contractility and myocardial muscle tension in response to an action potential. Studies in hypothermic animals demonstrated improved ventricular function curves, decreased mortality, and decreased time to resuscitation when the corrected pCO_2 was allowed to fall during cooling. Other studies showed that animals maintained at an alkalemic pH of 7.80 (uncorrected) had a significant increase in cardiac output after rewarming compared with animals maintained at an uncorrected pH of 7.40. These and other studies suggest that allowing corrected pH to rise and pCO_2 to fall as the temperature decreases, or maintaining the uncorrected pH at 7.40, leads to improved ventricular function.

No attempt should be made to correct the blood gas readings to compensate for changes in body temperature. The gases should be read uncorrected after the blood sample is warmed to 98.6°F and interpreted similarly to gases from a normothermic patient. This approach is clinically useful and physiologically sound.

(A more detailed discussion of this topic can be found in: Delaney KA, Howland MA, Vassallo S, Goldfrank LR: Assessment of acid-base disturbances in hypothermia and their physiological consequences. *Ann Emerg Med* 1989;18:72-82.)

TOXINOLOGY



ASPECTS OF LYME DISEASE

Willis A. Wingert, M.D.
(Professor, Joint Appointment Pediatrics, Community Medicine, Public Health and Emergency Medicine, University of Southern California; Director, Venom Laboratory, USC.)

Lyme disease, caused by the spirochete *Borrelia burgdorferi* and transmitted by the deer tick *(Ixodes* sp), has received increasing attention in recent years. As more cases are reported, the epidemiologic and clinical aspects become more complex. Selected topics are discussed in this article.

Lyme disease may be acquired even in your backyard, especially if your residence is in a wooded area in the northeastern or western states. The disease is transmitted by the tiny nymphal stage of the tick that may not be seen or felt as it feeds on the human host. Therefore, a negative history of tick bite or wilderness travel should not exclude the diagnosis of Lyme disease. Apparently, ticks must be attached for 24 hours before the spirochetes are transferred to the human victim. So if people remove ticks at the end of each day, in all likelihood they will not contract Lyme disease.

Several other preventive measures to avoid tick bite are recommended: wear socks over pants when hiking in the woods; apply permanone spray to clothes; and apply insect repellant with N,N,diethyltoluamide (DEET) to the skin. Since seizures have been reported following topical application of DEET, clinicians evaluating patients with unexplained seizures, especially those appearing in rural hospital emergency departments, should consider exposure to DEET.

Bell's palsy can no longer be considered "idiopathic" without inquiring about recent tick exposure. The onset may occur two or three months after the appearance of skin lesions. Bilateral Bell's palsy is pathognomonic of Lyme disease.

A young man presented to an emergency department with a sudden syncopal episode. Two months earlier he had a flu-like illness with fever and joint pains but no skin rash. The patient was an avid camper in the forests of Wisconsin. Cases of heart block have been reported in previously healthy men with periods of ventricular asystole lasting over 10 seconds. EKG demonstrated A-V block, sometimes accompanied by T wave flattening and ST segment depression. The conduction defect is secondary to Lyme carditis; it may occur late in the disease in 10% of patients. Antibiotics (penicillin, tetracycline, ceftriaxone, or erythromycin) may resolve the problem, often within tnree days.

The incidence of Lyme disease currently surpasses that of Rocky Mountain spotted fever, plague, tularemia and other vector-born diseases. However, all that appears to be Lyme disease might not be. The rickettsial organism *Erhlichia sennetsu,* which also has a tick-vector, may cause a severe flu-like illness with arthralgias, myalgias, fever and a non-circular skin rash not resembling erythema migrans. With a history of tick exposure, this syndrome might be diagnosed as atypical Lyme disease. Cases have been described in midwestern and southern states and are regularly confused with Rocky Mountain spotted fever in Oklahoma. The tip-off to the diagnosis is leukopenia and a low-platelet count during the acute illness. Diagnosis of *Erhlichia* can be made by serological testing at state health departments or at the CDC in Atlanta. Fortunately, the antibiotic tetracycline will cure all rickettsial infections as well as borreliosis.

Laboratory diagnosis for borreliosis is still unreliable. Culturing the organism is difficult and may require several weeks. Direct antigen detection in tissue lacks sensitivity. Serological testing (ELISA or IFA) assays have not yet been standardized, the cut-off value for a positive test is uncertain, and many false-positives and false-negatives occur. Treponemal antibodies cross-react, and the lesions of secondary syphilis resemble those of Lyme disease. Fortunately, high doses of penicillin will eradicate both spirochetes, even if the diagnosis is missed.

Can Lyme disease be eliminated? Elimination of the rodent reservoir is impossible and reduction of deer herd size is not popular. Large scale use of acaricides is of questionable effectiveness and often meets with public resistance. (Note the uproar over malathion spraying for "Med flies" in Los Angeles). Vaccination of high-risk individuals and susceptible domestic animals is the only practical measure.

Johnson and workers at the University of Minnesota have successfully developed a whole cell, inactivated vaccine against *B. burgdorferi* that was highly immunogenic in hamsters. Protection of 86% to 100% of vaccinated animals was achieved after 14 days. Unfortunately, the duration of immunity was short-lived, declining to 25% protection in 90 days. Much further investigation will be required regarding the optimal immunizing dose, use of adjuvants, booster doses, its use in larger animals, and finally human subjects. Commercial production of this vaccine is many years away.

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MEDICAL KITS FOR WILDERNESS TRAVEL

This new section in the *Wilderness Medicine Letter* will present a series of medical kits for wilderness activities. It is my opinion that most articles on medical kits are too general. In an attempt to include all eventualities for all types of activities and environments, the list becomes exhaustive and lacks specificity. The medical kit for a given trip depends on multiple factors, including environmental factors, planned activities, length of travel, the number of people in the party and their health status, the medical experience in the group and the availability of outside health care.

The kits in this series are designed for specific trips. The authors are not merely good clinicians with some interest in the outdoors; they are frequent wilderness travelers describing what they take on the trips described. They have designed these kits and *have actually carried them,* then refined them. The minimal contents of these kits (which was surprising to me) indicates that empiric experience, not an academic exercise, determined their design. It is informative to see which items are common to all trips, which are specific to particular endeavors, and which are never taken by these experienced authors.

Readers are welcome to comment.

Howard Backer, M.D., Editor

MEDICAL KIT FOR ROCK CLIMBERS

Thomas K. Hunt, M.D.
(Climber; Anesthesia Resident, Modesto, CA)

Rock climbers don't get hurt, or so they believe. Consequently, few are likely to accommodate a bulging first aid kit. More often than I like to admit, we leave the kit in the car, citing weight, space, or our safety record as the excuse.

In our study of 220 injured climbers in Yosemite[1], minor lacerations and lower extremity orthopedic injuries accounted for 80% of all injuries, and in most cases the party could effect self-rescue. Forty-four percent of Yosemite climbing injuries were to the lower extremity, particularly ankle sprain/strains and fractures, and 50% were lacerations or abrasions. Prevention is the best preparation for more severe injuries—usually head trauma and hypothermia; 65% of head trauma is fatal among climbers. Thus, my kit emphasizes local wound care and sprain or fracture management.

Setting: Rock climbing (Grade IV and above, or remote Grade II-III)
Duration: one long day to three days
Team: two to three persons
Geography: temperate climate; moderate altitude
Container: These items fit in a $3 watertight Tupperware container (8"x4"x2.5"). Wrap the SAM® splint[2] around it. The finale weight is 450 grams (about 1 pound).

Item (Quantity)
Uses & Comments

Bandage/first aid supplies

Bandaids (6-10)
Gauze sponges: 4x4 (2), 2x2 (4) and 2" gauze wrap (1); for covering lacerations and abrasions; can use for eyepatches.
Ace wrap (1): 4 inch for sprains;
to help immobilize a dislocated shoulder or fractured clavicle. Four inch cloth tape may provide superior support for ankles.
Splint (1):
the SAM® splint is the best I've seen. Some climbers carry chicken wire, 6 inches by 3 feet—easily rolled up, lightweight, but with good strength when doubled over. There are innumerable ways to improvise: a plastic water bottle cut apart and stuffed with ensolite then attached by duct tape; also try using pitons, wall hammers, ice axes.

Instruments/minor surgery

Needle driver (1):
I use the clamp from a suture set.
Small scissors (1):
general purpose, including debridement.
Tweezers (1):
splinters, ticks, etc.
Suture material (2 packets):
4-0 Ethilon for superficial wounds, and 0-Vicryl for the less likely event of a gaping wound.
Steri-strips, 1/2" (1 pack):
lightweight, fast.
Syringe, 10-20 cc (1):
for wound irrigation; I pour drinking water over the wound for irrigation, but a syringe provides more forceful cleansing.

Topical Medications

Bacitracin® or Neosporin® (3 packets):
for wound care; wrap these in Saranwrap® to avoid a mess.
Antiseptic pads, alcohol and/or povidone-iodine (6):
local wound care.
Benzoin, single-use capsule/applicator (1):
for use with steri-strips.

Oral Medications

Antihistamine: diphenhydramine (6):
bee sting, allergic reaction, "sleeper" on ledges.
Decongestant (6): Actifed® or Sudafed® or equivalent:
reduces nasal congestion on vigorous approaches and climbs; helps maximize breathing and comfort.
Non-steroidal anti-inflammatories (12): naproxen or ibuprofen:
for sprain/strains; critical for the weekend warrior anticipating Monday morning muscles.
Analgesics (12): acetaminophen with codeine or oxycodone:
pain.
Antibiotics:
I don't carry antibiotics on short climbs.

Parenteral Medications

Morphine sulfate in tubex
Epinephrine 1:1000 (1 ampule):
wrap ampules in toilet paper for protection.
Lidocaine 1% (1 ampule)
Syringe, 3cc with 24 gauge needle (1)

Miscellaneous

Oral airway:
among our study population, Yosemite Park medics used these several times with clear benefit.

Butane lighter (1):
to start heat or signal fires.

Pocket mirror (1):
rescue signal.

Prevention (1 ounce):
equals a pound of first aid kit.

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PREDICTORS OF SUCCESS IN WILDERNESS VENTURES

The following articles were adapted from a panel discussion held at the 1989 Annual Meeting of the Wilderness Medical Society, moderated by Dr. Blair Erb.

PHYSICAL ACTIVITY, THE ENVIRONMENT, AND FATIGUE

Blair D. Erb, M.D., F.A.C.P.
(Clinical Professor, Department of Preventive Medicine, University of Tennessee College of Medicine; Cardiologist; Director, Work Physiology Laboratory.)

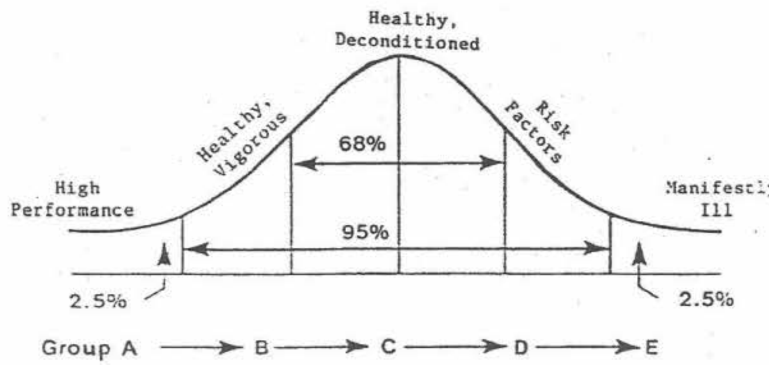
The popularity of wilderness ventures has attracted individuals with varied goals, skills, and physical abilities to participate in potentially hazardous activities. It is the burden of organizers to match qualified participants with particular ventures. Individuals who might have difficulty with the venture or who may place themselves or others in jeopardy must be recognized. Adventure planners, therefore, seek to identify factors that make a group compatible, cohesive, successful and, above all, safe. Physicians are frequently consulted in the selection of individuals for participation in wilderness ventures.

It is helpful to define the nature of the venture and the characteristics of the individual. A classification scheme of wilderness ventures according to physical demands includes: 1) extreme performance (e.g., peak above 6,000 meters, 2) high performance (e.g., remote hunting activities, jungle and high altitude trekking), 3) recreational (e.g., alpine hiking, trail walking), and 4) therapeutic (activities for those with illness or disability).

A useful classification of prospective participants in wilderness ventures according to their functional capacity and previous experience includes: A) demonstrated high performance individuals; B) healthy, vigorous individuals; C) healthy, "deconditioned" individuals; D) those with risk factors; E) those with demonstrated illness or physical limitations.

These may be schematically represented in a curve.

DISTRIBUTION CURVE FOR WILDERNESS VENTURERS



Participants in extreme performance activities have traditionally been selected by empiric techniques—elite cadre of experienced leaders and participants come together through a mutual selection process. Those who aspire to join an expedition to Mount Everest should have participated in similar activities and have proven their will and capacity to function at that level.

Of particular concern are individuals, many of whom are business or professional people, who are not used to high level activities and have become "deconditioned" yet still have the desire to participate in an exciting activity, often as a reaffirmation of their youth or vigor. Outdoor activities also have been used as a form of physical therapy, including cardiovascular rehabilitation. Subjects in this category are unique and require careful medical evaluation and a high degree of supervision.

Physicians often are asked to assess the capacity of the prospective wilderness venturer. To assist in developing an assessment protocol, I surveyed selected physicians and wilderness organizations and asked them to rank the relative importance of the following information: 1) personal, 2) historical, 3) medical, 4) physiological, and 5) psychological.

Although medical and physiologic factors are recognized as important for those who participate in wilderness outings, a history of successful or failed participation in previous ventures is a more important predictor. From the perspective of leaders and previous participants, the ability to predict failure is often more accurate than the ability to predict success. A history of psychosocial problems is also a major factor in anticipating failure. Consequently, there is a consensus that interviews for personal evaluation of prospective participants are critical in putting together a team.

Ultimately, multiple variables outside the realm of the selection process play a major role in outcome. It tests the skill of the leader to mold a group into a functional unit with common interests, and to maintain the "team effect" essential to the success of wilderness ventures.

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THE EFFECTS OF FATIGUE AND HEAT STRESS ON PERFORMANCE

Peter G. Snell, Ph.D.

(Exercise physiologist, University of Texas Health Sciences Center.)

Attempts to define fatigue have proven difficult. For this reason I will only discuss physical fatigue induced by strenuous or prolonged exercise. As every athlete knows, fatigue is a constant training companion but must be controlled carefully to produce improvement in performance.

Because fatigue ultimately limits all endurance performance, it is useful to consider fatigue states that impair *expected* performance levels. Naturally, if performance expectations are unrealistically high, failure due to early onset of fatigue occurs.

The acute fatigue experienced during brief bouts of heavy physical activity is usually caused by temporary accumulation of metabolic products, such as lactic acid, that inhibit muscle contraction. In wilderness ventures, however, the fatigue arises from sustained physical activity and is related to the depletion of energy stores, water loss and heat stress.

If muscle fibers become glycogen depleted, they are no longer able to participate in muscle contraction and their work must be taken over by non-depleted fibers. Carbohydrate intake at frequent intervals that matches the energy expenditure will spare muscle glycogen. If muscle fibers have not been recruited on a regular basis, they will not be adapted to sustained activity and will fatigue easily. Training reduces muscle glycogen usage by improving the ability to metabolize fatty acids. Muscle glycogen consumption is also reduced by decreasing the intensity of exercise. In summary, muscle glycogen, an important source of fuel, may be protected by: maintaining intake of complex carbohydrates during the exercise; improving fitness levels; and slowing down and/or taking brief rest breaks.

Water loss and heat stress are closely related because dehydration often precedes and disposes to heat stress. The body's ability to prevent heat stress depends largely on evaporative cooling from the skin; humid conditions that reduce evaporation are particularly dangerous. Adaptive defenses against heat stress include:

1. reduced body fat (decreased insulation);
2. improved peripheral circulation;
3. increased sweat rate;
4. increased blood volume;
5. improved efficiency of movement decreased energy expenditure).

The first four adaptations accompany endurance training, particularly training under hot conditions.

Preventive measures to minimize heat stress include the following:

1. wear loose light-colored clothing;
2. protect the head and neck from radiant heat by wearing a hat;
3. maintain hydration with regular fluid intake (the thirst mechanism allows significant dehydration);
4. avoid caffeine, alcohol or other diuretic substances; and
5. evaluate the environmental condtions. tions.

Adequate fluid intake especially before the need is felt, cannot be overemphasized.

Medical Director

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FACTORS INFLUENCING HUMAN PERFORMANCE IN A COLD ENVIRONMENT

Warren Bowman, M.D.
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Humans have evolved as "tropical" animals with only limited ability to adapt physiologically to cold from either short or long exposure. Humans may have been able to bypass natural selection by using their brains to design protective clothing and shelter. Mechanisms of cold adaptation observable in other animals can be demonstrated to only a small extent in humans.

Study of specific populations that suffer repeated, limited exposures to severe cold, such as Eskimos and Gaspe fishermen who work with their hands in cold water for long periods, has demonstrated some degree of increased extremity cold tolerance through enhancement of peripheral circulation. However, these groups do not seem to be more resistant to hypothermia than other populations. In peoples chronically exposed to moderate cold, such as Australian aborigines and Tierra del Fuegan Indians, some adaptation has taken place allowing them to tolerate lower body temperatures comfortably without shivering; these individuals are able to sleep naked on the ground at temperatures close to freezing.

Body temperature, which in homeotherms must be narrowly regulated for survival and even more narrowly regulated for optimal function, is the net result of voluntary and involuntary mechanisms. The most important organ for survival and optimal function in the cold is the human brain, since voluntary actions such as putting on clothing and seeking shelter are more important than involuntary mechanisms such as shivering and decreasing peripheral circulation. Accidental cold injury is therefore due to failure to use clothing and shelter properly rather than failure of the normal body's limited mechanisms of heat production or thermal regulation.

Based on personal experience and cold weather physiology, the most important elements in predicting cold weather performance are the following:

1. **Familiarity with a cold environment.** Persons who have grown up in a cold climate usually develop protective habit patterns by experience, observation, and specific training. The degree to which these patterns develop depends on innate common sense.
2. **Good aerobic physical conditioning and good nutrition.** These assure that an individual has the strength and endurance to travel to safety, construct shelter, and maintain heat generating muscular activity such as shivering and physical activity.
3. **Favorable psychological characteristics.**
   a. The ability to work as part of a team.
   b. A minimum of impulsive behavior and the tendency to avoid unnecessary risks.
   c. Slow to panic.
   d. Mature and uncomplaining.
   e. Self-reliant, thorough, not a "moocher."
   f. Clean and orderly in personal habits.
   g. Imaginative, particularly in predicting the need for essential equipment and remembering to include it in the pack.
   h. Non-addictive, non-self-indulgent personality.
4. **Absence of significant illnesses or injuries** that increase risk in cold weather through interference with heat production, heat transportation or temperature regulation.
   a. Peripheral vascular disease.
   b. Uncontrolled hypertension.
   c. Generalized vascular disease or heart disease, including arteriosclerotic heart disease, congestive heart failure, angina, previous myocardial infarction, previous coronary artery bypass surgery.
   d. Raynaud's phenomenon, cold agglutinin disease, cryoglobulinemia.
   e. Cold or exercise induced asthma.
   f. Poorly controlled diabetes.
   g. Previous history of frostbite.
   h. Heavy smoker.
   i. Regularly required medications that interfere with temperature regulation or heat production, such as major tranquilizers, anticholinergics, beta-blockers.

In summary, acquired experience in the cold is the most important criterion. Equally important is the ability to function in a group under stress. Because of the physical and psychological challenges of a cold environment, there is little margin for error. Nevertheless, organizations such as Outward Bound and the National Outdoor Leadership School have shown that with good leadership and equipment, neophytes can live with relative safety in the winter wilderness.

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WHO WILL YOU TAKE ON A HIGH MOUNTAIN EXPEDITION?

Charles S. Houston, M.D.
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My predictors of success on an expedition are based on three major Himalayan climbs, 12 years of medical research on a mountain at high altitude, and a 45-day research project in a chamber at simulated altitude. Information used to choose participants in these ventures include: personal characteristics, historical "track" record, physiological testing, medical history and physical exam. A similar approach can be used for other kinds of wilderness expeditions.

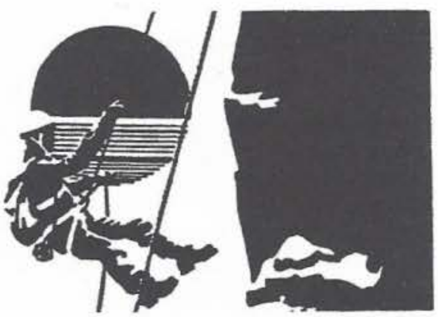
The personal aspects of the candidate are important. Is he or she someone with whom you would like to spend days or weeks stormbound? Does his or her personality mesh with yours and with others already chosen? Will he or she be a good conversationalist and know when to talk and when to keep silent? Motivation is hard to ascertain but it may be the most important consideration in choosing the right companions and, thereby, improving the chances of success.

In 1936, four of us put together the Nanda Devi expedition; we knew each other well, had climbed together, and we were all fit and healthy. We invited four Brits, whom we did not know, on the basis of their track records. It was a supremely happy and successful trip. In 1938, two of us put together the first American K-2 trip. We looked at track records, personalities, references from friends, and relied heavily on a personal interview, choosing people we liked over some with longer and harder climbs to their credit. In 1953, two of us followed the same procedure: we chose people who had experience, who were strong (not necessarily the best) climbers, but above all, people we liked and could enjoy even in tough situations. Both of these trips were unforgettably happy expeditions; our survival in 1953 depended on the cohesiveness of the team.

For the Mt. Logan physiology studies that ran every summer from 1967 to 1979, I selected the subjects. People heard about the study by word of mouth and volunteered. I asked for eight letters of reference (and settled for six), as well as completion of a form that asked about career plans, aspirations, climbing record, and other interests. From several dozen applicants, I interviewed one-third. During the interview, I tried to understand the person's motivation in life and for the project. I looked for humor—in retrospect, the few persons who did not do well did not have a sense of humor. I probed for what the candidate considered his or her strong points and weaknesses, and usually got revealing and helpful answers. The letters of reference (which I acknowledged) were often very helpful not only to exclude someone, but to rank his or her desirability. The form did ask about health, medications, injuries, previous mountain experience, etc, and a recent medical examination and doctor's statement were required, but seldom helpful. In the end, the selection was made by my assessment and intuition, primarily from the interview, of what each person would be like in the stressful environment of research at 17,500 feet over a six-week period.

A list of previous climbs, with some estimation of difficulty and the course of events, is helpful but limited. It will tell what the individual has done, but it may not reveal how well or poorly it was done. But in reviewing this record, some insight can be gained into the personality and motivation of the candidate. A long list of climbs done with the same companions is a plus; numerous extreme solo ventures or climbs with widely different teams needs further investigation.

Physiological testing for altitude tolerance is still imperfect. No test or battery of tests will predict how one will adjust to high altitude, or to the extreme cold and privation to be anticipated on a great mountain. Decompression chamber and hypoxic ventilatory response tests are affected by too many variables to yield accurate predictions.



Given a healthy, physically fit person, the cardiogram, pulmonary function tests, treadmill stress studies, blood chemistry and others are also of little help. One reason is that we do not have a precise definition or measure of how well an individual is acclimatized at any point in time, only a perception of the ability to perform without symptoms, or at least with minimal symptoms, since everyone has problems at very high altitude. In fact, training does not improve altitude tolerance, only the ability to exercise. In short, physiology will not help us much in predicting success or in choosing a team member for a mountain expedition.

We look at the medical history and examination. What illnesses or injuries has the person experienced? How has he or she tolerated altitude and privation in the past? Someone who has had HAPE or HACE or even severe AMS once or several times in the past is not a good candidate, because we know that those who have had altitude illness are more likely to have it again. Does the person have some underlying problems with any part of the oxygen transport system—lungs, heart, circulation, blood or tissues? Some lung problems (e.g., asthma) may or may not impair performance. Some heart problems (congenital absence of a pulmonary artery) are absolute contraindications. Some heart murmurs, like mitral valve prolapse, are innocent; others are significant. Anemia is often a deterrent, as is poor peripheral circulation, including Raynaud's disease and a history of severe frostbite. High blood pressure, kidney disease, diabetes, epilepsy and many others need to be examined very carefully. But few of these are absolute criteria: history is full of remarkable achievements by severely handicapped persons. In summary, the medical history and examination will help to identify persons with the potential for extreme problems, but those individuals would be unlikely to apply. In most cases, the history and physical will not be helpful in predicting success or failure.

In conclusion, the priorities I would give the various factors in selecting members for a mountaineering expedition are:

1. personality;
2. intuition of whether this person will fit with the others;
3. climbing experience;
4. prior altitude illness (only serious, repeated episodes are significant);
5. physiologic and medical tests.

The following articles from the Journal of the American Medical Association *are reprinted with permission of the publisher and the authors.*

TREKKING IN NEPAL: SAFETY AFTER CORONARY ARTERY BYPASS

(*JAMA*, June 3, 1988; 259:3184)

Question: A 51-year-old physician recently had a coronary artery bypass procedure performed, with excellent results. He has not had a myocardial infarct and has excellent myocardial function, including good results from a postoperative stress electrocardiogram and from a thallium scan. He has always enjoyed good health and has been physically active, including long-distance jogging. Risk factors include a low level of high-density lipoprotein, a cholesterol level of 6.47 mmol/L (250 mg/dL), and probably an "A" personality type.

He plans a trekking vacation to 5.76 km in Nepal. Is there any reason not to proceed with this trip? He is back to jogging and has lowered his cholesterol level to just under 5.17 mmol/L (200 mg/dL) with diet alone. His high-density lipoprotein level is still low, and he will consider taking low doses of lovastatin.

Answer: Our first concern would be about the results of the exercise electrocardiogram and thallium scan. It would be good to know specifically what they showed. In addition to the exercise electrocardiogram results, it would be helpful to know what work level in metabolic equivalents of oxygen consumption the patient was able to reach during the exercise test as well as the maximal heart rate, double product, and systolic blood pressure response. All of these have functional and prognostic implications. Of importance also would be whether he has returned to physical activity, and, if so, his running speed and distance.

From a secondary prevention point of view, it is advisable to try to raise his high-density lipoprotein level and lower his cholesterol level by diet and exercise. In addition to these measures, it would be good to prescribe aspirin and/or dipyridamole (Persantine) daily to increase the probability of keeping the grafts open.

Now specifically in regard to the question. At the altitude of 5.76 km the barometric pressure is about 380 torr and inspired oxygen pressure (PO_2) only 70 torr (vs 150 torr at sea level), and arterial PO_2 at rest will be in the 30s to low 40s, depending on how well acclimatized one is. This contrasts with the normal PO_2 at sea level of about 90 torr in a man of this age. During exercise at this altitude, arterial PO_2 will probably be even lower. Thus, a trek to this altitude will cause severe arterial hypoxemia and substantial reduction in tissue PO_2 (including the myocardium).

Although the bypass procedure sounds successful, it is unlikely that coronary circulation is normal. We believe that going to such a high altitude would entail some risk, and so the answer to the question "Is there any reason not to proceed with the trip?" is yes. Both of us believe it would be better to err on the side of being safe rather than sorry.

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THE SAFETY OF TREKKING AT HIGH ALTITUDE AFTER CORONARY BYPASS SURGERY

(*JAMA*, Oct. 21, 1988; 260:2218-2219)

To the Editor

In a recent QUESTIONS AND ANSWERS section[1], it was asked if a 51-year-old patient with an excellent result from coronary bypass surgery should go on a trek to Nepal up to altitudes of 5760 m (19 008 ft). An exercise treadmill test and a thallium scan had shown "good results" and the patient had been doing long-distance jogging. Two consultants stated that "some risk" would be entailed owing to the low arterial oxygen partial pressure at that altitude and the patient was advised not to go, since "it would be better to err on the side of being safe rather than sorry."

I have reservations regarding this advice. If this policy is applied to the many thousands of active individuals who have had coronary bypass surgery, it will result in needless fear and concern about the effect of altitude on the heart and continue to promote the erroneous concept that altitude is bad for the heart.

In the post-bypass coronary patient, an individual approach is indicated. In general, if grafts are patent, left ventricular function is normal, and treadmill exercise can be performed to normal levels without symptoms, ischemia, or significant arrhythmia, I see no reason why a physically fit active individual should not participate in trekking at high altitude providing proper acclimatization is observed.

It is true that rapid ascent to high altitude is occasionally associated with a modest increase in heart rate, blood pressure, and cardiac output, owing to increased activity of the sympathetic nervous system. This increase in cardiac work can result in a transient increase in symptoms of angina or heart failure. Restricted activity and appropriate medication may be necessary. After a few days, sympathetic activity subsides as acclimatization proceeds and symptoms abate. In the acclimatized individual, there are two aspects of the effect of altitude on the heart that must be considered: (1) Does high altitude increase cardiac work over sea level values? (2) Does the hypoxemia of high altitude depress cardiac function?

Cardiac work during heavy exercise is less at high altitude than when doing the same exercise at sea level. At high altitude, physical activity is limited by dyspnea and pulmonary gas exchange and not by the circulatory pump or cardiac output.

Even maximal exercise at altitude is accompanied by a lower heart rate than at sea level. At sea level, young men can attain a heart rate of 178 beats per minute at a work load of 2400 kg/m/min. At 6300 m (20 790 ft.), maximal exercise is accompanied by a heart rate of only 146 beats per minute at a work load of 1200 kg/m/min, indicating a significant reduction in cardiac work.[2] The patient with coronary disease is therefore more likely to die while running at sea level than trekking at high altitude. Trekking does not involve maximal work, but a comfortable pace that can be continued for many hours.

Does the decreased arterial oxygen saturation at high altitude adversely affect cardiac function? During maximal exercise at extreme altitudes, arterial oxygen saturations as low as 50% to 59% have been recorded. Under these conditions, no abnormalities of cardiac function have been observed.

Left ventricular filling pressures remain normal.[3] Echocardiograms indicate normal left ventricular function.[4] No electrocardiographic evidence of ischemia or arrhythmias is present. Animal studies have shown no depression of left ventricular function at arterial oxygen saturations of 51%, even when the heart is denervated.[5]

For these reasons, I see no reason to interdict high-altitude trekking in patients who have had a good result from coronary bypass surgery, providing appropriate tests are performed and acclimatization is observed. It is of interest that the patient in question went on the trek with no cardiac symptoms or any difficulties.

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In Reply

Dr. Hultgren raises some interesting points. However, I stand by our original contention that a patient who has had coronary bypass surgery would undergo some risk by trekking to an altitude of 5760 m (19 008 ft.).

There is overwhelming evidence of severe arterial hypoxemia at this great altitude irrespective of whether the trekker is well-acclimatized. For example, when a group of physiologists lived at a similar altitude of 5800 m (19 140 ft.) for several months during the Himalayan Scientific and Mountaineering Expedition of 1960 to 1961, the mean arterial oxygen saturation was only 67% at rest and fell further during exercise.[1] The calculated resting arterial oxygen partial pressure (PO_2) was only 36 mm Hg. Such a low arterial PO_2 must cause severe tissue hypoxia.

Although the 51-year-old patient had a coronary artery bypass with apparently excellent results, it is unreasonable to assume that his coronary circulation is normal. There are bound to be atheromatous areas that reduce blood flow. This together with the greatly reduced arterial PO_2 and oxygen content must cause some risk.

Another factor to consider is that the patient will be extremely remote from good medical treatment if he treks at this great altitude in Nepal. It should be pointed out that 5760 m (19 008 ft.) is exceptionally high for trekking. Very few passes are as high as this and they are all remote. In addition, every westerner at these altitudes develops marked periodic breathing during the night, which will cause further severe depression of arterial PO_2 as a result of the apneic periods.[2]

Dr. Hultgren cites evidence that the normal heart tolerates extreme altitude well. This is true, but hardly relevant in the present context, where the coronary circulation is not normal.

Several statements need specific comments. Dr. Hultgren writes, "Cardiac work during heavy exercise is less at high altitude than when doing the same exercise at sea level." There is no evidence to support this. In acclimatized subjects, cardiac output at a given work level is the same at high altitude as at sea level[3,4] and systemic blood pressure is essentially unchanged.[5] There is no reason to think that cardiac work is less, and certainly it is carried out at a very much lower myocardial tissue PO_2.

The statement that "even maximal exercise at altitude is accompanied by a lower heart rate than at sea level" needs clarification. This is true only because maximal work capacity is reduced at high altitude (because of tissue hypoxia). However, at a given work level the heart rate is higher at altitude than at sea level.

The contention that "the patient with coronary disease is therefore more likely to die while running at sea level than trekking at high altitude" is not only questionable but irrelevant. No one is advocating either course. Both forms of activity entail some risk.

In summary, while the normal heart apparently tolerates extreme altitude very well, there is every reason to believe that a patient with residual coronary artery disease is at increased risk at the very high altitude of 5760 m (19 008 ft.). Of course, the patient can choose whether to accept this risk, but it is there.

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WILL MOUNTAIN TREKKERS HAVE HEART ATTACKS?

(*JAMA*, Feb. 17, 1989; 261:1045-1046)

A consequence of the fitness boom has been that it is now commonplace for symptomless people to go to their physicians to be checked out before they take up jogging, racquetball, or aerobic dancing. What most people are concerned about is the presence of coronary arterial disease and the possibility that the exercise will cause sudden death or myocardial infarction. The public, persuaded that such checkups are as crudely mechanistic as those for automobiles, is happy to pay physicians to carry them out, although there is little evidence that such routine checks effectively reduce morbidity or mortality from subsequent exercise.

Increasing affluence and a greater interest in the outdoors have caused ever larger numbers of people to go to the mountains to exercise—either to climb, walk (trek), or ski cross-country. (Downhill skiing, where the work is done by the lift, is not an impressive aerobic challenge.) Many of these people will ask their physicians whether it is safe for them to ascend to high altitudes. Increasingly, trek operators are insisting, for legal reasons that do not necessarily have anything to do with clinical reality, that candidates are accepted only if accompanied by a certificate signed by a physician.

Physicians, although unaware of the real risks, will be forced back on what they know about altitude physiology to be able to give the patient some sort of theoretical explanation about possible risks. A sophisticated physician will explain that with increasing altitude the atmospheric pressure falls and with it, the partial pressure of oxygen, so that at around 5500 m (18 045 ft) the atmospheric pressure is half that at sea level, and at the summit of Mount Everest (8848 m, 29 028 ft), it is one third (253 mm Hg).

They will go on to point out that the oxygen tension in the arterial blood mirrors this, so that average readings in reasonably well-acclimatized people who have ascended to these altitudes might be about 45 and 28 mm Hg, respectively—figures that would catch the attention of any clinician. Although very few trekkers will go as high as 5500 m, the physician might naturally predict that the heart, which has high demands for oxygen, would, with the exertion of ascent, suffer cardiac damage. Indeed, when Ravenhill,[1] in 1913, described three young men with what we now call "high-altitude pulmonary edema," he thought it was due to cardiac failure, although he was careful to note that the men were all perfectly healthy beforehand and that "a great many people with weak or diseased hearts would fail to show cardiac symptoms" on ascent to the high Andes.

In the October 21 issue of *The Journal,* the question of whether it would be safe for a 51-year-old man, who had made an excellent recovery after coronary bypass surgery, to walk up to 5760 m (19 008 ft) in Nepal was argued by two men who had made extraordinary contributions to the field.[2, 3] The physiological argument was made by West,[3] who pointed out that as there was severe hypoxemia at this altitude, as maximum work capacity was reduced, and as the man's coronary circulation could not be normal, it was better to be safe rather than sorry and advised him to stay home. Hultgren[2] disagreed, saying that there was no abnormality of left ventricular function at high altitudes and that the patient should be permitted to go. (He did and was fine.)

We can summarize the vast mass of physiological data by saying that there is no evidence that the *normal* heart, up to the height of Mount Everest, limits human endeavor or that altitudes threaten the *normal* heart by making it fail or suffer an infarction. The question for the physician is therefore whether the heart of the symptomless "patient," who comes to see the physician solely to get a clean bill of health so that he can go to high altitudes, is normal. The physician then has to consider whether we have an infallible method for predicting, in symptomless people, morbidity and mortality from coronary arterial disease. And if we do, whether it will predict cardiac troubles brought on by exercise and, finally, whether it will predict that these problems will develop on exposure to high altitude, with or without exercise.

For a symptomless man aged 50 years, the predictive power of a positive exercise electrocardiogram (ECG) (where the prevalence is 5.4%; the sensitivity, 50%; and the specificity, 90%) is around 22% when

the data are drawn from research teams that use strict criteria.[4] Thus, a positive result will be wrong four times out of five. We have no reason to believe that a positive exercise ECG will be any more useful when done on symptomless people in the average physician's office. We have no idea how predictive this test would be of cardiac problems that develop during heavy exercise (though if the subject is too unfit to complete the treadmill test, he is unlikely to enjoy exercising in the mountains), and none at all about the ability of a positive test to predict cardiac problems on ascent to high altitudes.

We can increase the test's predictive power by combining the exercise ECG with independent tests such as the thallium myocardial reperfusion scan and radionuclide cineangiography with technetium. However, I do not believe that it is reasonable to do any of these expensive tests in fit, symptomless individuals who are contemplating ascent, unless the treadmill test is strongly positive, because even if adding more and more tests increased the positive predictive power, we still would not know what to do with the results. We have no reason to think that any medical or surgical interventions would decrease future morbidity or mortality, and we certainly do not know whether a positive result after all these tests would be a contraindication to ascent.

If the patient has symptoms, has had a prior infarction, has clinically evident coronary arterial disease, or has had a coronary bypass operation, the situation is very different. Neill and Hallenhauer,[5] Ziolkowski and Wojcik-Ziolkowska,[6] Brammell and colleagues[7] have shown that on ascent, patients with coronary disease have decreased exercise tolerance and earlier appearance of angina and ST-segment changes. Hultgren[8] has pointed out that angina develops at the same level of work at whatever altitude; sympathetic activity is temporarily increased immediately after ascent and with it, heart rate and blood pressure. Is there, then, evidence that illness and death from coronary disease occur commonly at high altitudes? This is not a trivial question in that Moore[9] has estimated that 35 million people visit altitudes above 2439 m (8000 ft) in the United States yearly, of whom 2.9 million have hypertension and 456 000 (2%) have coronary arterial disease.

Deaths from jogging at low altitudes are estimated to occur at the rate of 1 per 7620 joggers.[10] Singh et al[11] found lower rates of almost all conditions, including hypertension and ischemic heart disease, in 20 000 men stationed between 3692 and 5538 m compared with the rates in 130 700 soldiers stationed below 760 m, but it is quite unclear how much selection took place to determine who was sent to high altitude. Malhuber et al[12] summarized their results in 1273 "cardiac patients" who ascended to altitudes between 1500 and 3000 m. They found negligible morbidity in 593 hypertensives and 434 patients with coronary disease, 141 of whom had sustained prior myocardial infarctions, although one of these did have a new infarction. These authors mention that among 151 000 vacationers of both sexes who went to high altitudes in the Alps, including 69 460 persons who were more than 40 years of age, there were six deaths. Of these, one person had a myocardial infarction in the valley, and two had myocardial infarctions in the mountains. Two persons died of unknown causes.

In 1983, Dickinson et al[13] reported autopsies after seven altitude-related deaths in Nepal among men aged 27 to 62. All had pulmonary or cerebral edema or both. None died of myocardial infarction. One, aged 38 years, who had just completed a rapid ascent of Kilimanjaro (5900 m) had severe occlusive disease of the anterior descending and circumflex vessels, but this had nothing to do with his death. Three of the seven were physicians; perhaps being a doctor is a risk factor.

The study by Shlim and Houston,[14] in this issue of *The Journal*, provides us with more information. Of 148 000 people who obtained trekking permits in Nepal during 3-1/2 years, eight died of illness. There were no proven cardiac deaths, although the cause in two persons was unknown. Six people were evacuated by helicopter for cardiac reasons—two were men in their late 50s with severe cardiac disease; one 27-year-old man with persistent ectopic beats; and three men aged 39, 41, and 55 years with chest pain that was not believed to be angina. No data are provided about the age and sex of the trekkers. Dr Peter Hackett and I found that in 1976 the mean age of people who reached 4243 m (13 921) on the way up to see Mount Everest was 33 ± 13 years (± 1 SD) and 71% were men.[15] We may assume that they, and possibly their physicians, thought they were fit enough to trek in the high mountains. Shlim and Houston do not provide data regarding length of time spent trekking, altitudes reached, speed of ascent, or drugs taken by the trekkers. We do not know the incidence of symptoms or the incidence of evacuations, whether by porter, horse, yak, or airplane.

This series and the series of vacationers reported by Malhuber et al[12] include 299 000 people. If we assume that a maximum of six persons died with myocardial infarctions and that none of them had symptoms beforehand, and if, as before, we assume a sensitivity of 50% and a specificity of 90%, then the predictive value of an exercise ECG in a symptomless person who wanted to ascend to a high altitude would be 0.0001%. We can conclude that there is little point in doing such a test for screening to prevent people with completely silent ischemia from going to high altitudes, since a positive test will be far more likely to be false than true, and the subject will be put at risk of all sorts of iatrogenic, psychological, and clinical hazards.

My own practice is to take a careful history from people who ask me whether they can go trekking at high altitudes. I explain that if they are not able to carry out strenuous, long, continued exercise comfortably at sea level, they can hardly expect to be able to do so at high altitudes. I suggest that, if possible, they should give themselves a trial at moderate altitudes, say 2500 m (about 8000 ft). If they have any symptoms, for example, angina, they should ascend even more slowly than usual so that they can acclimatize. Above all, this will prevent pulmonary edema and the accompanying severe arterial desaturation. I would remind individuals that they are going on an adventure—getting away from it all. One of the things they are getting away from is good, prompt emergency medical care. This means that they must take full responsibility for themselves because although the remoteness will be their greatest pleasure, if they become ill, it could also be their greatest problem.

I do not believe that "better safe than sorry" is sufficient ground to prevent anyone from having an adventure. The patient mentioned earlier is not alone in having trekked comfortably to very high altitudes after bypass surgery. If we are to respond to our patients appropriately, we need more complete studies to be able to make accurate predictions about the risks that someone with or without symptoms of heart disease faces on ascent to high altitudes. For the moment, however, I believe that if we are to forbid an activity that may have great meaning to an individual, we should do so only on the basis of studies that convincingly controvert the data we now possess. These data, though admittedly incomplete and heterogeneous, are reassuring. What we may conclude from them is that going up into the mountains to trek or ski cross-country is a safe undertaking for those who believe they are able to do this. Physicians have no good, objective evidence either to embark on elaborate testing (such as exercise ECGs) or to stop their patients from doing what they want.

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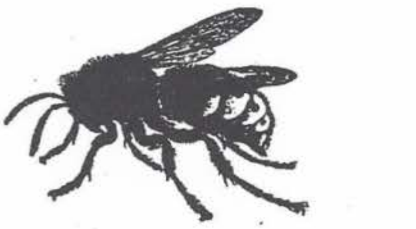

EPIDEMIOLOGY OF INSECT VENOM SENSITIVITY

Golden DB, et al. *JAMA* 1989; 262:240-244.

Reviewed by ***Edward C. Geehr, M.D.***
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The prevalence of insect sting reaction and venom sensitization in adults is unknown. Immediate hypersensitivity reactions to insect stings can be effectively prevented by Hymenoptera venom immunotherapy. Thus, in order to develop a rational approach to adult immunotherapy, it is essential to understand the population at risk. In a recent article in *JAMA,* a research team from Johns Hopkins determined the extent to which adults have systemic allergic sensitivity to insect stings.

In a stratified random sample of 269 subjects, the prevalence of systemic allergic sting reactions was 3.3%. Another 26.5% had IgE antibodies to venom demonstrated by skin test or radio allergosorbent test (RAST). Asymptomatic sensitization, that is those with no history of allergic sting reaction, was observed in 15% of subjects. The highest frequency of positive tests (35%) was observed in those known to have been stung within the past three years. The rate declined to 20% in subjects tested more than three years after the sting.

The clinical significance of asymptomatic sensitization is unknown in those with no history of allergic reaction. The authors are currently undertaking a long-term study to determine the prognosis for regression of sensitivity and the risk of anaphylaxis in such sensitized subjects.

BRIEF REVIEWS

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Cerebral Perfusion Deficits in Dysbaric Illness.

Adkisson GH, Macleod MA, Hodgson M, Sykes JJW, et al. *Lancet,* 15 July 1989;2:119-22.

Dysbarism is a term for all pathological changes secondary to altered environmental pressure. Clinical problems are seen most frequently in divers, compressed air workers and aviators. The two main forms of dysbarism are barotrauma and decompression sickness (DCS). Barotrauma results from volume/pressure changes of gas in a closed space. Pulmonary barotrauma results from gas expansion in lung tissue during ascent that may lead to alveolar rupture with the potential of a fatal cerebral arterial gas embolism (CAGE).

DCS results from gas bubbles formed during or after decompression. DCS is usually categorized as type I (mild: peripheral pain, non-neurological) or type II (serious: neurological). Type II is predominantly a spinal cord disease with infrequent cerebral involvement; however, complaints of lethargy, mental cloudiness and confusion are common.

Cerebral perfusion studies (by injecting ^{99m}Tc-hexamethylpropylene-amineoxime) and single photon emission tomography were obtained in 28 divers with DCS and CAGE. Cerebral perfusion deficits were present in all cases of type II DCS and CAGE. In this study no deficits were present in the single case of type I DCS. The results indicate that type II DCS is a diffuse, multifocal, central nervous system disease.

Type II Altitude Decompression Sickness (DCS): U.S. Air Force Experience With 133 Cases.

Wirjosemito SA, Touhey JE, Workman WT. *Aviat Space Environ Med* 1989;60:256-62.

In 1960, Golding classified decompression sickness (DCS) into type I (limb and joint pain) and type II (multiple systemic symptoms). Type II altitude-related DCS is often difficult to diagnose. To better understand this condition, 133 cases of type II altitude DCS (filed at the U.S. Air Force Hyperbaric Medicine Division, School of Aerospace Medicine, Brooks AFB, TX) were reviewed retrospectively. Ninety-five per cent of the cases followed altitude chamber training.

Clinical manifestations:	**%**
Headache	43.6
Visual disturbances	30.1
Extremity paresthesias	27.8
Mental confusion	24.8
Extremity weakness	16.5
Extreme fatigue	10.5
Cerebellar signs	9.0
Choking sensation	6.0
Extremity numbness	4.5
Truncal pain	3.8
Unconsciousness	1.5
Hypotonia of anal sphincter	1.5
Flaccid paralysis (extremity)	0.8
Skin mottling	0.8

Hyperbaric oxygen treatment produced successful results in 97.7% of the cases, while 2.3% had residual defects. There were no deaths.

Predisposing factors mentioned in the literature include obesity, exercise, old injury, hypoxia, dehydration, alcohol, cold, age, time of day (lower in the afternoon), sex (females higher), post-immunization, repeated exposure, and individual susceptibility.

Differential diagnosis includes: angina pectoris, spontaneous pneumothorax, non-DCS limb pain, alternobaric vertigo, abdominal distention, hypoxia, motion sickness, delusions with spatial disorientation, acceleration atelectasis, and psychological stress.

A unique classification method was developed on the hypothetical ability to perform a complicated task (land an aircraft):

- Class 1: Headache or minor neurological signs only, which should not have interfered with flying abilities.
- Class 2: Conscious and would have been able to land an aircraft safely.
- Class 3: Conscious but would not have been able to land an aircraft safely.
- Class 4: Unconscious or unable to respond and not able to land an aircraft.

It was concluded that individuals with symptoms limited to Class 1 and 2 type II DCS can safely remain on flying status.

Childhood Near-Drowning: Is Cardiopulmonary Resuscitation Always Indicated?

Nichter MA, Everett PB. *Crit Care Med* 1989;17:993-5.

Because of the difficulty in predicting the outcome of pediatric near-drowning, 93 consecutive warm water near-drowning victims with significant physiologic impairment on emergency department (ED) arrival were retrospectively reviewed for reliable absolute predictors of eventual outcome. Variables generally available to ED physicians were unreliable outcome predictors — including age, sex, length of submersion, core temperature, arterial pH, absence of spontaneous respirations, lack of response to pain, and pupillary nonreactivity. Sixty-eight percent of those who required CPR alone went on to intact survival. However, all victims who required cardiotonic medicines (epinephrine, atropine) to establish a perfusing cardiac rhythm during the initial resuscitation had severe neurologic damage or death. Based on this series, the need for cardiotonic medicines in pediatric warm water near-drowning is strongly associated with a poor prognosis.

The Hemodynamic and Cardiovascular Effects of Near-Drowning in Hypotonic, Isotonic, or Hypertonic Solutions.

Orlowski JP, Abulleil MM, and Phillips JM. *Ann Emerg Med* 1989;18:1044-9.

Early literature on drowning has reported hypervolemia-hemodilution with fresh water and hypovolemia-increased blood viscosity with seawater. The hemodynamic effects of instillation of 20ml/kg of solutions of various tonicities (sterile water, 0.225% sodium chloride [NaCl], 0.45% NaCl, 0.9% NaCl [normal saline], 2% NaCl, and 3% NaCl) into the lungs of anesthetized mongrel dogs were evaluated and compared with five minutes of anoxia in control animals. All animals had an immediate decrease in cardiac output (CO) and lung compliance (LC) with an increase in the pulmonary capillary wedge pressure (PCWP), central venous pressure (CVP), and pulmonary vascular resistance (PVR). The PCWP and CVP peaked at ten minutes and then declined over the four hours of the study. The CO and LC remained depressed throughout the study, while the PVR gradually increased. These results support a hypothesis that the cardiovascular changes following near-drowning are the direct result of anoxia and are independent of the tonicity of the aspirated fluid.

BOOK REVIEWS

Environmental Emergencies

by Charles E. Stewart. Baltimore: Williams and Wilkins, 1990, 411 pages, $39.50.

Reviewed by: ***Edward J. Otten, M.D.***
(Faculty, Department of Emergency Medicine, University of Cincinnati; Military Survival Instructor; Board of Directors, WMS.)

Lately there has been a plethora of publications in "environmental" medicine. However, I have yet to find a clear definition of this term. Most authors invoke the principle, "When I use a word it means exactly what I choose it to mean—neither more nor less." From my review of the literature I have found common characteristics in previous discussions of environmental medicine. First, most authors emphasize emergencies, probably because the majority are trained in emergency medicine. Second, the environment usually involves the "wilderness." However, I would guess that more cases of heat stroke occur in inner-city apartments and more cases of scromboid occur in restaurants than any wilderness areas. Third, prevention is frequently stressed. I find preventive medicine to be the most interesting and important aspect of "environmental" medicine.

This book fits the above profile. The author is a board certified emergency physician, most of his chapters include references to problems encountered in the wilderness, and he mentions preventive measures as well as treatment. The text is divided into fourteen chapters and an appendix. The author includes the usual topics: heat injuries, hypothermia, frost bite, altitude sickness, sunburn, near-drowning and diving injuries. A large section is devoted to toxinology: poisonous plants and animals, venomous bites and stings. He includes two chapters of a strictly preventive nature: water disinfection and prevention of arthropod envenomation. In addition, chapters on burns, electrical injuries and radiation are included. While treatment of these latter topics was well done, I do not see their relevance to a textbook of environmental emergencies, in preference to topics like skiing injuries, avalanche injuries, or fish hood removal. With some creative publishing the author could have two books: 1) environmental emergencies and 2) burn injuries: thermal, chemical, electrical and radiation.

One disadvantage of a single author text is the difficulty of handling many topics in depth. But Dr. Stewart has done an excellent job researching and compiling material. He has not avoided controversial subjects; the book contains excellent discussions of fasciotomy in snakebite, antibiotics for animal bites, CPR in hypothermia and treatment of mushroom poisoning. The chapter "Environmental Emergencies in the Wilderness Context" with Dr. Keith Conover (the lone contributor) also confronts controversies and offers reasonable solutions.

For the most part, the text is organized well; however, I became slightly confused reading the "Bites and Stings" chapter. It would be easier to use if venomous and nonvenomous bites were in separate chapters with another chapter for poisonous animals. The text is easy on the eyes and the illustrations were very well done. I especially enjoyed many of the tables and insert summaries of important items. The references are up to date with few exceptions. The index, the most important part of a textbook, was complete. There were some minor typographic errors, but not enough to distract the reader. In summary, I commend Dr. Stewart for writing an excellent work. I am sure it was a monumental task for a single author and it reflects an exceptional physician. I recommend this book to those who need an introduction to environmental emergencies, but cannot afford the Auerbach and Geehr "bible" *(Management of Wilderness and Environmental Emergencies)*. If Dr. Stewart removes the burn material and cuts the size down a bit in the next edition, it would fit nicely in my backpack.

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