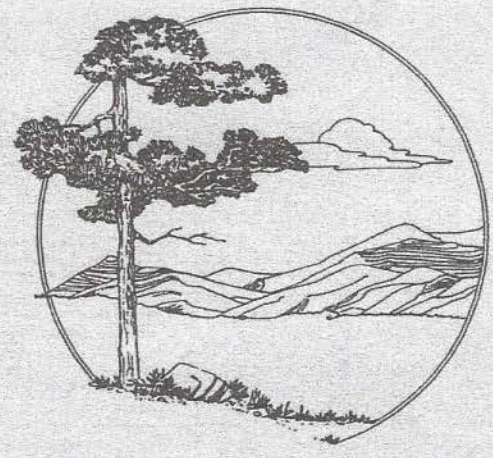
WILDERNESS MEDICINE

The official newsletter of the WILDERNESS MEDICAL SOCIETY

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

Vol. 5. No. 1 | Edited by Howard Backer, M.D. | January, 1988

SOCIETY NEWS



Binders for the Newsletter

Three-ring binders to hold issues of *Wilderness Medicine* are available now from the WMS office. The binders have the Wilderness Medical Society name and logo printed on the cover and spine. This is the ideal way to keep copies of the newsletter together, accessible, and identifiable on your bookshelf. The cost for each binder is $7.00, which is the Society's cost plus postage.

Membership and Treasury

Membership currently numbers 1,135. We are gaining approximately 40 new members per month. Our best source of new members has been by word-of-mouth and by discussion of the Society to interested groups, such as wilderness medicine continuing education meetings. Five membership brochures are included with this issue of the newsletter for members to distribute to friends and colleagues. Please help in the growth of the Society.

The treasury currently contains $16,592. The Board is bonding the treasury to ensure that no person can run off and squander this fortune.

Annual Meeting and Wilderness Medicine Conference

The WMS will sponsor a continuing education conference focusing on mountain medicine, along with our annual business and scientific presentations, at Jackson Lake Lodge in Grand Teton National Park, September 14 through 18, 1988. Dr. Howard Donner is assembling an excellent program, which will include discussions of high altitude, determinants of exhaustion, and topics such as hypothermia and near-drowning. The entire program will be printed in an upcoming issue of the newsletter. Plan to attend; if possible, spend a few extra days to enjoy the beauty of the Tetons and nearby Yellowstone Park.

Newsletter

Contributions by WMS members to the newsletter are regularly solicited. The more participation, the more interesting and informative the newsletter will become. A broad spectrum of subjects, ideas, and styles can be accommodated. Members are encouraged to report pertinent projects and research in which they are involved. Reviews of articles and summaries of published research are important to keep current in this broad field. Those interested in writing should contact the editor to discuss subject material.

Medical Student Scholarships

Research proposals were received from five medical students for the grant sponsored by the WMS in honor of Dr. Charles Houston. The Board has awarded $1,000 to William L. Krause, a third year medical student at Dartmouth Medical School, to study "The Effect of Acute Exposure to Hypoxia on Physiological Tremor in Man." The results will be presented at a WMS annual meeting. The board wanted to encourage a second research proposal by David A. Petersen, a second year medical student at the University of Arizona in Tuscon, for a study entitled "Local Tissue Response to Electric Shock Treatment for Snakebite." When this research is completed, the Society will pay expenses for David to present his findings at our annual meeting.

Environmental Research Symposium Proceedings

Papers from the 1987 Research Symposium on Environmental Emergencies, sponsored by the University Association for Emergency Medicine, have been published in the *Annals of Emergency Medicine* (Vol 16, No 9, Sept 1987). Dr. Paul Auerbach edited this issue which is devoted entirely to environmental medicine. Many of the papers will be reviewed or discussed in future issues of the WMS newsletter, but members who do not subscribe to the *Annals of Emergency Medicine* may wish to purchase a copy of this issue ($5) from the publisher: 1125 Executive Circle, Irving Texas 75038-2522.

ASTM Committee on SAR

Not only will the American Society for Testing and Materials (ASTM) be involved in developing standards for Emergency Medical Services including Wilderness EMT (see President's Column), but it is forming a committee to develop consensus standards for search and rescue activities. Areas of search and rescue activity specified as needing standards include: equipment and uses, organization and management, personnel training and education, search operations and techniques, rescue operations and techniques, and terminology. All interested parties should join ASTM and participate in the development of these standards; the organizational meeting will be held March 4-5, 1988. Contact Wendy Dyer, ASTM, 1916 Race Street, Philadelphia, PA 19103, (215) 299-5526 for more information and meeting locations.

Advertising in the Newsletter

The Board has decided to allow appropriate advertising in the WMS newsletter to help cover expenses of publication. Rates and information can be obtained from Dian Simpkins, the society secretary.

Environmental Medicine Research Director

The Department of Emergency Medicine at Bellevue Hospital is seeking an experienced candidate for the position of Director of Research to focus on Environmental Medicine. Qualifications include either training and certification in Emergency Medicine with research experience (M.D.) or pure research (Ph.D) in Emergency Medicine/Environmental Medicine. Excellent salary and benefits are offered. Interested persons should forward a CV to Lewis Goldfrank, M.D. Director, Emergency Medical Services, Bellevue Hospital Center, 27th Street and First Ave, New York, New York 10016. Telephone: (212) 561-3346.

Expedition Members Wanted

1988 Noah's Ark expedition seeks more participants for their archaeologic search for the biblical ark on Mt. Ararat, a 17,000 foot peak situated on the Turkish/Soviet/Iranian territorial triangle. Two prior expeditions have located a promising site. Under special research permit, advanced ice sonar, drills and saws will be used on this trip, which will establish a base camp at 16,800 feet on rugged terrain. Five weeks will be spent in this activity during July/August 1988. Interested persons should contact Dr. Charles D. Willis, Box 3118, Pinedale, CA 93650.

Current Board of Directors of the Wilderness Medical Society; *Standing, left to right: Blair Erb, Howard Backer, Edward Geehr, Peter Hackett, Kenneth Kizer; Kneeling, left to right: Paul Auerbach, Kenneth Iserson*

WILDERNESS MEDICINE
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.
Appropriate advertisements accepted.
Copies of most past issues are available.

THE PRESIDENT'S COLUMN

Paul S. Auerbach, M.D.

I would like to take this opportunity to inform the WMS members about the American Society for Testing and Materials (ASTM) involvement with Wilderness EMT (EMT-W) standards, announce a bit of good news regarding a new Journal of Wilderness Medicine, give advance notice of a membership benefit that you will soon be offered, remind you to set aside time for next year's Annual Meeting, and ask for help recruiting new members.

ASTM is an organization that was initially created to set consensus standards for materials, products, systems, and services, but which has subsequently become involved with health care matters such as ambulance standards and standards for prehospital care personnel. ASTM defines a standard as "a rule for an orderly approach to a specific activity, formulated and applied for the benefit and with the cooperation of all concerned." The Department of Transportation has indicated that they will pay close attention to the consensus recommendations that evolve from these committees, and thus it appears to be important that the most qualified individuals possible make an effort to become involved. There is no benefit to organizational membership, in that it costs seven times as much without any increase in voting power. Therefore, I hope that any WMS member who is knowledgeable in prehospital care and wilderness medical issues will take the time to join Committee F-30 on Emergency Medical Services (Subcommittee .02.05 on Wilderness Medicine), in order to generate as much WMS input into these new standards as possible. The cost is $50 per year; further information and application information can be obtained from ASTM, 1916 Race Street, Philadelphia, PA 19103; telephone (215) 299-5400.

Chapman and Hall, Ltd. publishers in Great Britain have tentatively agreed to publish *The Journal of Wilderness Medicine* beginning in 1989. This will be a quarterly peer-reviewed medical journal with an international editorial board and worldwide distribution. I am honored to have been asked to share the primary editorial duties with Dr. Oswald Oelz of Switzerland. The journal will be the official journal of the WMS, and will be included with membership in the organization. The publishers have pledged to make it available to WMS members at the lowest cost possible, so that the impact upon dues will be kept to a minimum. The WMS Board of Directors is incredibly excited about this project, and we will keep you posted on our progress.

The WMS Board of Directors has agreed to allow the Maryland Bank of North America to offer a special VISA card to members of the WMS. A sign-up and card renewal award, as well as a per use bonus credited to each transaction, will be donated back to the WMS treasury, which will be used to support Society activities. Each member will receive an invitation from MBNA, and I urge you to take advantage of this opportunity, which will directly benefit the WMS at no extra cost to the Society or to members who participate.

Next year's Annual Meeting will be held September 14-18, 1988 in Grand Teton National Park. Mountain medicine issues will be stressed, and the invited speakers program will be finalized in the next few months. A full day will be devoted to Society business and presentations by members, and we can look forward to fascinating evening programs, our annual banquet, and more superb wilderness medicine and rescue demonstrations. Please plan to support your Society by attending this meeting.

Finally, I would like to stress how important it is to the Society's activities that our membership growth be smooth and continuous. We have surpassed the 1,000 member mark over the past few months, and it would give us a position of better fiscal strength to double or triple membership over the next few years. Our strongest advocates have been the members, and we have therefore avoided advertising or media-based recruitment. With this issue of the newsletter, you have received 5 membership brochures. Please distribute these to your professional associates or friends if you think that they might benefit from the information and activities derived through WMS membership. We welcome them as members and thank you in advance for your assistance.

MEMBER'S PROFILE:

SHERMAN A. MINTON, M.D.

Howard Backer, M.D.

Dr. Sherman Minton, a founding member of the WMS, remembers a time when he was afraid of snakes, because he thought that they were all poisonous. However, during his school years in New Albany, Indiana, he was already capturing and keeping captive snakes. During high school, Sherman suffered his first venomous bite from a copperhead that he had secretly caged. Afterwards, his safety-minded parents allowed him to build a good cage and bring his hobby out of the woods. But he was warned by his father – who was afraid that Sherman would run off and join the circus – that he would never make a living as a herpetologist.

Sherman did go to medical school, but he never gave up herpetology. As a medical officer during WWII and an intern at San Diego Naval Hospital, he developed an additional interest in parasitology. He performed graduate studies in microbiology and herpetology at the University of Michigan. In 1947, Dr. Minton joined the new microbiology department at the Indiana University School of Medicine, where he is now professor emeritus.

Although his early medical research concerned viruses, the majority of his writing has always been about snakes and other reptiles. By 1957, his medical research merged with his reptile work, focusing on venom toxicity and immunology. Recent research examines the use of serologic methods in taxonomy of snakes, immunological relationships of snake venoms, and the mechanism of resistance of certain animals to snake venoms.[1] Dr. Minton has authored or coauthored more than 90 technical papers and monographs and nearly as many textbook chapters, journal reviews, editorials, and book reviews.

Sherman humbly states that he has little wilderness experience; he always enjoyed camping since childhood Boy Scout outings, but now he usually sleeps in town while doing field work. However, he has taken every opportunity to study venomous creatures in

their natural, remote habitats: 1955, a sabbatical to Big Bend, Texas; 1957, a tropical medicine fellowship in Costa Rica; and 1958-62, visiting professor at the Medical Institute in Karachi, Pakistan. Later field work took him to Australia, back to Pakistan and Iran (as a research associate with the American Museum of Natural History), and on three research expeditions with Scripps Institute of Oceanography to Australia and the Philippines. Family vacations with his wife and three daughters were often 7-10 days in the desert collecting specimens.

While his daughters have not developed their father's passion for venomous animals, Sherman's wife, Madge, has coauthored several books with him.[2,3,4] In Dr. Minton's opinion, these and a few other works[5,6] are among his most significant contributions to science. His work has included a broad spectrum of reptiles and amphibians, including turtles, sea snakes, scorpions, and crocodiles, in addition to snakes.

During the course of his work, Dr. Minton has experienced five venomous snake bites and one scorpion sting – fortunately, none serious. But the last copperhead bite, in 1983, was "very unpleasant." Only one bite was in the field. In fact, in Sherman's experience, poisonous snakes are hard to find in the field and one must know where to look for them. In 1984, Dr. Minton retired from Indiana University, but there has been no decrease in either his writing or his lifelong investigations of reptiles, amphibians, and toxicology.

References

1. Minton SA. Present tests for detection of snake venom: clinical applications. *Ann Emerg Med* 1987; 16:932-937.
2. Minton SA and Minton MR. *Venomous Reptiles* 1969; Charles Scribner's Sons, New York, xii + 273 pp. pls.
3. Minton SA and Minton MR. *Giant Reptiles* 1973; Charles Scribner's Sons, New York, xiii + 345 pp. illust.
4. Minton SA and Minton MR. *Venomous Reptiles,* revised edition, 1980; Charles Scribner's Sons, New York.
5. Minton SA. *Venom Diseases* 1974; Charles C. Thomas, Springfield, Ill, xv + 235 pp., illust.
6. Minton SA. *Amphibians and Reptiles of Indiana* 1972; The Indiana Academy of Science, Indianapolis, v + 346 pp., illust.

ABSTRACTS FROM THE 1987 ANNUAL MEETING

A LIAISON BETWEEN THE WILDERNESS MEDICAL SOCIETY AND THE BOY SCOUTS OF AMERICA

John N. Schuetz, M.D., F.A.C.S.

(Practice of Obstetrics and Gynecology, Park Forest, Illinois; Member, National Safety Council; Representative to National Council, Member National Exploring and Safety Committees, Boy Scouts of America.)

The Boy Scouts of America (BSA) is the largest outdoor-oriented youth organization in the United States. Membership is close to four million, which includes youth from ages six to eighteen and older leaders. The BSA has been concerned with outdoor safety since its inception. In cooperation with the American Red Cross, water safety standards were established in the 1920's and updated continuously. The Merit Badge program stresses safety, with ten badges in the field of aquatics alone.

Thousands of camps are operated by local BSA councils all over the country. The National Council owns or uses several "High Adventure Bases" in different parts of the USA. The largest is Philmont Scout Ranch in New Mexico – more than 100 square miles of rugged country in the Sangre de Christo mountain chain – where several thousand young people (male and female) go on the wilderness treks every summer.

Every four years the BSA stages a National Scout Jamboree. The last two were located in Camp Hill, Virginia. In addition to 35,000 Scouts and leaders staying in the camp, there were tens of thousands of visitors. The U.S. Army not only provided the site, but also gave logistical assistance, including a M.U.S.T. Hospital in camp and an evacuation system to civilian hospitals. The U.S. Public Health Service was also involved in the operation.

A wide variety of outdoor activities sponsored by the BSA could result in injury: e.g., hiking, climbing, lumberjacking, archery, and use of pack animals. Fatalities and disabilities are, of course, of great concern to all Scout leaders. The cost of jury awards (or out-of-court settlements) resulting from liability suits are rapidly increasing. In the effort to prevent mishaps, the BSA established a National Health and Safety Committee, with membership consisting of physicians and liaisons from various organizations, including the American Red Cross, National Safety Council, AMA, Presidential Council on Physical Fitness and Sports, National Rifle Association, representatives of insurance groups, and experts in aquatics.

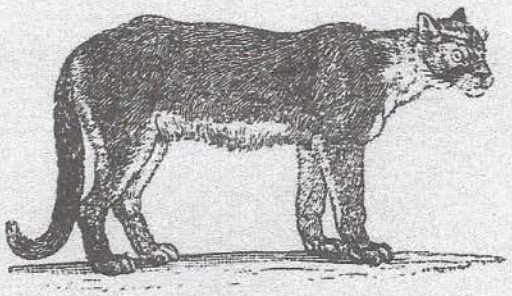
A few of the responsibilities of this group are: (1) to establish safety standards for camping operations and travel, (2) to supervise and scrutinize reports of fatal and serious accidents or illnesses, (3) to train camp staff and various aquatic personnel, (4) to develop programs related to fitness, safety and various sports.

The BSA is in need of additional experts to assist in their effort to offer exciting and meaningful outdoor programs to young persons with maximum safety. Therefore, I would like to establish a liaison between the WMS and the BSA. By sharing data, ideas, and solutions to wilderness medical problems, we could improve preparedness and response to problems within the BSA. Boy Scouts who are taught preventative practices will practice these during a lifetime of wilderness activities.

PASTEURELLA MULTOCIDA INFECTION COMPLICATING A COUGAR BITE

Kenneth W. Kizer, M.D., M.P.H.

(Director, California Department of Health Services; Founding Member and Past President of the WMS.)



Attacks on humans by cougars are very rare, despite these animals being the most widely distributed large animals on the American continent. In fact, just one report of such could be found in the medical literature,[1] and only infrequent brief mention of such attacks is made in other relevant literature.[2,3,4] The author recently treated two people who were attacked by a pet cougar, one of whom developed infection with *Pasteurella multocida* in a bite wound on the neck.

Case 1. A 28-year-old woman who recently began working as a caretaker for a pet cougar entered the animal's cage and was attacked. The animal bit her left arm and her neck. A detailed description of the attack was not given initially. The animal had been declawed.

On presentation to the emergency department, the patient had scattered deep abrasions and small avulsions on her left forearm and hand; a 2 cm horizontal subdermal laceration on the right side of her neck overlying the mid-sternocleidomastoid muscle, with mild swelling over the mandible and temperomandibular joint; and superficial (non-penetrating) bite wounds of the lower anterior neck. There was no crepitus about the wounds. On inspection of the wound, the sternocleidomastoid muscle appeared to have been penetrated, but there did not appear to be any deep injury. The victim was fluent, without stridor or hoarseness. The remainder of her examination was normal. After deliberation and consultation, the neck wound was closed with interrupted simple sutures. She was prescribed dicloxacillin and given detailed follow-up instructions.

Two hours after being discharged from the ER, the patient telephoned to report the development of hoarseness and production of blood-streaked sputum. On repeat examination shortly thereafter, she was found to have increased swelling and some crepitus about the wound. Radiographs of the neck demonstrated subcutaneous emphysema involving the prevertebral and retropharyngeal spaces, as well as subdermally on the right side of the neck. Chest radiograph showed no acute abnormalities. On specific questioning, she reported that the animal "shook" her when it bit. ENT consultation was obtained, and subsequent esophagoscopy, laryngoscopy and tracheoscopy, as well as a barium esophagram, did not detect a perforation, although slight ecchymosis of the vocal cords and some bloody mucus in the immediate subglottic area were found.

Because of increased swelling, redness and edema about the neck wound on the day after the attack, it was opened after the patient underwent panendoscopy. A substantial amount of sanguinopurulent fluid was drained. The wound was surgically extended, and the neck explored. A small perforation on the posterior side of the SCM muscle was found, as well as a tract that headed in the direction of the thyrohyoid membrane. Subsequent cultures of the wound drainage were positive for *Pasteurella multocida.* She was continued on dicloxacillin and recovered completely except for paresis of the right vocal cord, which is slowly improving.

Case 2. A 34-year-old man who accompanied the first patient into the cougar's cage was also attacked as he tried to disengage the animal from the woman. He sustained numerous bite wounds (deep abrasions) of both upper extremities and the left knee. These wounds were cleaned, and a small laceration on his right index finger was steristripped. All his wounds healed uneventfully.

Pasteurella multocida is a saprophyte of the respiratory tract of many animals and is a well-known pathogen causing infection of domestic cat bites.[5,6] It also has been identified as a cause of serious infections from lion, tiger and panther bites;[7] however, *P. multocida* infection has not been previously reported as a complication of cougar bites.

P. multocida is a gram negative coccobacillus that is unusual because of its sensitivity to penicillin; this drug or one of its derivatives is the treatment drug of choice. In cases of penicillin allergy, tetracycline, chloramphenicol, cephalosporins and possibly, trimethoprim-sulfamethoxazole are the preferred alternative drugs. Erythromycin is generally effective, although some treatment failures have been reported with this agent. Since plasmid-mediated antibiotic resistance has been demonstrated with this pathogen, sensitivity testing is necessary.

This case underscores two important points. First, *P. multocida* should be considered as a contaminant and potential pathogen in any cat bite wound, and second, any wound resulting from a large-cat bite should be assumed to penetrate deeply. Unfortunately, because of the paucity of clinical experience with such wounds, it is impossible to make recommendations about primary wound closure of cougar bites; however, closure would seem to be reasonable, since these injuries are much more like dog bites than domestic cat bites.

REFERENCES

1. Grant DF. Massive trauma to the head and face, attack by a cougar: report of case. *Journal of Dentistry for Children,* May-June, 1979. p. 58-61.

2. Clarke J. *Man is the Prey.* Stein and Day Publishers. New York. 1969. p. 157-159.

3. Fitzhugh EL and Gorenzel WP. Biological status of mountain lions in California. In Salmon TP (ed), *Proceedings of the Twelfth Vertebrate Pest Conference,* University of California, Davis. 1986. p. 336-346.

(references continued next page)

4. Nowak RM. *The Cougar in the United States and Canada.* U.S. Fish and Wildlife Service, Washington, D.C., and New York Zoological Society, New York, New York. 1976.
5. Kizer KW. Epidemiologic and clinical aspects of animal bite injuries. *JACEP* 1979; 8: 134-141.
6. Weber DJ, Wolfson JS, Swartz MN, et al. *Pasteurella multocida* infections: report of 34 cases and review of the literature. *Medicine* 1984; 63: 133-154.
7. Burdge DR, Scheifele D and Speert DP. Serious *Pasteurella multocida* infections from lion and tiger bites. *JAMA* 1985; 253: 3296-3297.

MEDICAL REVIEWS & NEWS

THE ROLE OF BAROMETRIC PRESSURE IN HIGH ALTITUDE ILLNESS

Benjamin D. Levine, M.D.

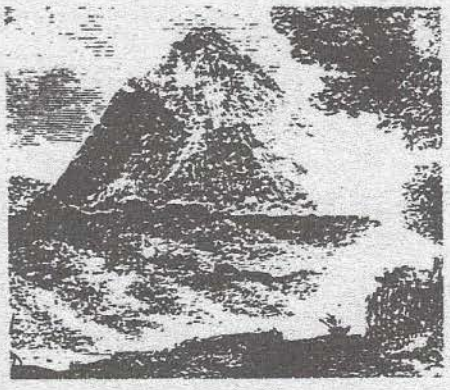
(Cardiology Fellow and Research Associate, Human Exercise and Space Medicine Laboratory, University of Texas Health Science Center, Dallas)

While most physiologists would currently agree that hypoxia is the dominant environmental stress at altitude, a number of recent studies have begun to suggest that it may not be the whole story. Clinical observations by Hackett[1] and Houston and Dickinson[2] have shown that descent (i.e., increasing barometric pressure *and* inspired oxygen concentration) more rapidly improves the signs and symptoms of patients with acute mountain sickness than does simple administration of supplemental oxygen. Additionally, animal studies by Bland et al.[3] have failed to produce a model for high altitude pulmonary edema, even with extreme degrees of alveolar hypoxia at sea level.

The role of barometric pressure in the physiologic response to altitude has been studied by Grover, Tucker, and Reeves at the University of Colorado. In the late 1960's and 1970's, they investigated ventilatory acclimatization to altitude, as well as the effect of altitude on the hypoxic ventilatory drive.[4,5] They demonstrated that upon ascent, minute ventilation increases due to hypoxia. However, because the mass of air is decreased at altitude, the number of molecules of oxygen ventilated per minute is actually less than at sea level, resulting in a relative hypoventilation. Over a period of days, the volume of ventilation continues to increase so that once acclimatization is complete, the mass of air moved is virtually the same as at sea level.

While this ventilatory adjustment is intimately tied to changes in acid-base balance, their findings suggest that, in addition to the role of reduced inspired oxygen tension and the secondary reduction of reduced alveolar carbon dioxide, barometric pressure may play a specific role in the ventilatory response to altitude. These investigators have also demonstrated an acute, albeit transient, effect of hypobaria per se on respiratory chemosensitivity, describing a depression in the ventilatory response to hypoxia after normoxic decompression.

Recently, Gray[6] has likened mountain climbing to ascent from a "saturation dive" in scuba diving;



decompression causes intravascular bubble formation and institutes a host of hematologic changes leading to pulmonary edema. This theory is based in part on earlier work[7] demonstrating reduced platelets in animals and human volunteers exposed rapidly to altitude. Microbubbles may serve as a nidus for platelet aggregation or leukocyte activation, generating release of vasoactive substances and chemical mediators such as prostaglandins and bradykinin, which could produce lung injury. Pulmonary edema has been produced experimentally in this fashion by injection of air bubbles into sheep.[8]

Arguing against this theory is the fact that the formation of bubbles is critically dependent on both the rate and absolute magnitude of decompression. Bubble formation is known to occur with rapid ascent in diving and aviation; but in those circumstances, ascent is orders of magnitude faster than that routinely seen in mountaineering. Pulmonary edema can occur with rapid ascent ("the chokes"), but decompression sickness manifests much more frequently as local joint pains ("the bends") or spinal cord involvement. One would expect that if bubble formation were to occur frequently enough to be an important cause of acute mountain sickness, then at least some cases of the bends would have been reported in the mountains. However, this is not the case, despite the fact that altitude illness is a common phenomena. Acute mountain sickness has been reported in up to 50% of trekkers in Nepal;[9] subclinical rales have been observed in approximately 20% of climbers above 4,000m.[10]

Recently, Levine and colleagues[11] at Shinshu University in Japan attempted to determine the relative contribution of hypoxia and barometric pressure to pulmonary fluid balance at altitude. Using the Staub model of chronically instrumented sheep with catheters in the efferent pulmonary lymph duct and relevant vascular compartments, they exposed each animal to 3 separate conditions: hypobaric hypoxia, normobaric hypoxia, and normoxic hypobaria. The sheep were studied in 3 successive levels of decreasing barometric pressure and/or inspired oxygen concentration, corresponding to 2600m, 4600m, and 6600m of altitude. Transition between stages was accomplished slowly, to prevent or minimize bubble formation. They found that only under combined conditions of hypobaric hypoxia did lymph flow increase, reflecting increased extravascular lung water. Neither hypoxia nor hypobaria alone produced any change in lung fluid balance. They hypothesized that reduced barometric pressure, possibly mediated through decreased partial pressure of nitrogen or reduced hydrostatic pressure, might modify the response of the pulmonary vasculature to

hypoxia. Alternatively, decreased alveolar pressure might increase the driving force for fluid filtration across the pulmonary capillary, particularly when combined with increased pulmonary pressures generated by hypoxic vasoconstriction.

Finally, Schoene et al.[12] have recently shown on Mt. McKinley that directly increasing inspired air pressure using expiratory positive airway pressure can improve oxygenation at rest and during exercise, both in normals and patients with high altitude pulmonary edema.

Thus, evidence continues to accumulate that while hypoxia is certainly the most important stress at altitude, barometric pressure per se may well be a critical factor in the pathophysiology of high altitude exposure. Parenthetically, at the Himalayan Rescue Association clinic in Pheriche, Nepal (4243m), a small pressure chamber carried up by the Japanese in the late 1970s has been used occasionally in patients with severe high altitude illness, with anecdotal reports of dramatic success. These data provide further support for the clinical maxim that descent is the treatment of choice for acute mountain sickness.

References

1. Hackett, PH. Acute mountain sickness—the clinical approach. *Adv. Cardiol.* 1980; 276-10.
2. Houston, CS; Dickinson, J. Cerebral form of high altitude illness. *Lancet* 1975; 2:758-761.
3. Bland, RD; Demling, RH; Selinger, SL; and Staub, NC. Effects of alveolar hypoxia on lung fluid and protein transport in unanesthetized sheep. *J. Appl. Physiol.* 1977; 40(3):268-274.
4. Grover, RF. Effects of hypoxia on ventilation and cardiac output. *Ann. N.Y. Acad. Sci.* 1965; 121:662-673.
5. Grover, RF; Tucker A.; and Reeves, JT. Hypobaria: An etiologic factor in acute mountain sickness? in *Oxygen Transport to Human Tissues.* Loeppky, JA; and Riedesl, ML; eds., N.Y., Elsevier North Holland Inc. 1982.
6. Gray, GW. High altitude pulmonary edema. *Semin. Resp. Med.* 1983; 5(2):141-150.
7. Gray, GW; Bryan, AC; Freedman, MH; Houston, CS; Lewis, WF; McFadden, DM; and Newell G. Effect of altitude exposure on platelets. *J. Appl. Physiol.* 1975; 39:648-651.
8. Staub, NC; Schultz, EL; and Albertine, KH. Number and distribution of neutrophils (PMN's) in sheep lungs after air embolization. *Am. Rev. Resp. Dis. 123.* 1981; (4, part 2):1235.
9. Hackett, PH and Rennie, D. The incidence, importance and prophylaxis of acute mountain sickness. *Lancet.* 1976; II:1149.
10. Hultgren, HN. High altitude illness. In *Management of Wilderness and Environmental Emergencies.* Auerbach, PS and Geehr, EC, eds., N.Y., Macmillan Publishing Co., 1983.
11. Levine, BD; Kubo, K; Kobayashi, T; Fukushima, M; Shibamoto, T; and Ueda, G. The role of barometric pressure in pulmonary fluid balance and oxygen transport. *J. Appl. Physiol.* January 1988, in press.
12. Schoene, RB; Roach, RC; Hackett, PH; Harrison, G; and Mills, WJ. High altitude pulmonary edema and exercise at 4,400 meters on Mount McKinley, effect of expiratory positive airway pressure. *Chest.* 1985; 87(3):330-333.

INITIAL REPORT: HIGH ALTITUDE RESEARCH EXPEDITION

William N. Bernhard, M.D.

(Associate Professor of Anesthesiology, N.Y.U. Medical Center)

The 1987 American Andes Biomedical Research Expedition conducted a series of observations and tests on short-term adaptation to high altitude between June 3 and June 19 in the Cordillera Blanca of Peru. The expedition reached its high camp at 18,000 feet in the col between Nevado Pisco and Huandoy Este via the Lake Llanganuco route in Huascaran National Park on the eighth day of the climb. All members of the expedition climbed to the summit of Nevado Pisco (19,000 feet).

During ascent half the team (4 from sea level and 1 from Boulder, Colorado) received one 500 mg Diamox Sequel daily for 8 days and dexamethasone 2 mg QID for 7 days, followed by 3 days of tapering doses. The other 5 team members, who live between 5,000 and 7,000 feet, chose not to take Diamox and dexamethasone to prevent acute mountain sickness (AMS).

The ascent began with a drive from Lima to Huaras (10,000 feet) (day 1). After two nights in Huaras, we drove to 14,000 feet (day 3) and stayed two nights. From there, we climbed to 15,500 feet and slept 2 nights (days 5 and 6). Research gear and packs were carried to each higher camp, but climbers returned to sleep at the lower camp. The expedition reached the final base camp at 18,000 feet on the seventh day and climbed to the highest point of 19,000 feet on days 8 and 9.

Measurements obtained on all members at sea level, 10,000, 14,000, and 18,000 feet included symptom scores, performance tests, pulmonary function tests (PFT), psychometric tests, tear flow, hemoglobin, and visual evoked potentials (VEP).

VEPs are electroencephalographic recordings done with a needle inserted in the scalp at the vertex. LED goggles are used to stimulate both eyes with strobe flashes. Signals are averaged and entered into a microprocessor for analysis. Latency of response is an indicator of early cerebral edema.[1,2]

Major findings from this pilot study can be summarized as follows:

(1) No climbers taking Diamox and dexamethasone for prevention developed AMS. Two of the 5 highlanders who did not receive prophylactic medication developed significant AMS symptoms.

(2) The 2 obvious cases of AMS showed latency changes in VEPs in comparison with baseline tracings.

Their VEP normalized with resolution of AMS symptoms (1 after 4 mg of dexamethasone q 6 h and 1 with spontaneous improvement).

(3) Performance on the symbol digit modality test did change significantly with increasing altitude. Short-term memory did not decrease.

(4) Calculator performance decreased with increasing altitude. Climbers with better physical conditioning had less decrease and less variation in performance. We were not satisfied with the Gibney calculator test for task performance and memory; it is not difficult enough to test altitude effects on performance.

(5) Tear flow measurements demonstrated that tear output is the same at high altitude and at sea level. This suggests that contact lenses can be safely worn when climbing and trekking at high altitude.

(6) Serial hemoglobin readings demonstrated a 3-4% increase (0.5 gm) in red blood cell mass during the two-week period at altitude (which included 5-7 days at 18,000 feet). It is unlikely that this small increase in hemoglobin or red cell mass can account for short-term acclimatization (less than 2 weeks).

(7) Minute ventilation increased with altitude and Diamox. Data from this pilot study suggest that sustained-release Diamox given with dexamethasone can prevent AMS. Additional studies are needed to compare the effects of using Diamox Sequels alone to the combination of Diamox and dexamethasone. The correlation of clinical AMS symptoms with changes in VER suggests that AMS is related to hypoxia-induced cerebral edema.

We hope to perform further studies on the prevention of AMS on a climb up Huascaran, Peruvian Andes, in 1988. Climbers interested in a cooperative research climb in June 1988 should contact William N. Bernhard, MD, Department of Anesthesiology, University Hospital, NYU Medical Center, 560 First Avenue, New York, NY, 10016, Telephone (212) 340-5024.

References

1. York D, Legan M, Benner S, Watts C. Further studies with a noninvasive method of intracranial pressure estimation. *Neurosurg* 1984, 14(4):456-61.

2. Wohns R, Colpitts M, Colpitts Y, Clement T, Blackett B. Effect of high altitude on the visual evoked responses in humans on Mt Everest. *Neurosurg* 1987, 21:352-356.

Editor's Comments:

These observations suggest areas of productive research for future studies. Particularly interesting are the measurement of tear flow and the use of visual evoked potential (VEP) as a marker of subclinical cerebral edema. It appears that VEP is sensitive to hypobaric hypoxia; Dr Bernhard just received unpublished data of a chamber experiment which supports his data from the mountain.

Recent work studying dexamethasone suggests that this drug can prevent symptoms of altitude illness, but may be better for treatment than for prophylaxis.[1] *Acetazolamide is still preferred for prophylaxis of altitude illness because the physiologic effects actually promote altitude acclimatization.*

1. Hackett PH, Roach RC. Medical therapy of altitude illness. *Ann Emerg Med* Sept 1987; 16:980-986.

MULTICENTER HYPOTHERMIA SURVEY

Daniel F. Danzl, M.D., F.A.C.E.P.
(University of Louisville)
Robert S. Pozos, Ph.D.
(University of Minnesota - Duluth)

No single facility treats a large enough number of hypothermic patients with similar predisposing or precipitating factors, so there are no prospective controlled studies evaluating the residual effects of treatment after stratifying adverse outcome risks.

Therefore, a multicenter hypothermia survey was performed to evaluate the presentation, treatment and outcome of civilian hypothermia. A questionnaire which was developed by the study group was completed on hypothermic patients presenting during the study period. Pertinent historical, physical, diagnostic and therapeutic findings were recorded. The database on these 428 cases was obtained from thirteen sites (Table). There were 296 males and 132 females. Although most cases presented during "winter" months, 69 occurred in Florida. No significant differences were found by decade of age in presenting temperature or rewarming strategy. The results were recently published.[1]

TABLE

Alaska (N = 27) - William Mills, Jr.
British Columbia (N = 5) - Sheldon Glazer
California—San Francisco (N = 45) - Paul Auerbach
Colorado—Denver (N = 26) - John Marx
Florida—Tampa (N = 69) - Jon Miller
Kentucky—Louisville (N = 52) - Sal Vicario
Maryland (N = 3)
Michigan—Detroit (N = 82) - Richard Shields, Richard Nowak
Minnesota (N = 20) - Patrick Lilja, Eric Johnson
New York City (N = 62) - William Goetz
Oregon—Portland (N = 34) - Jon Jui
Washington—Bellingham (N = 3) - Marvin Wayne

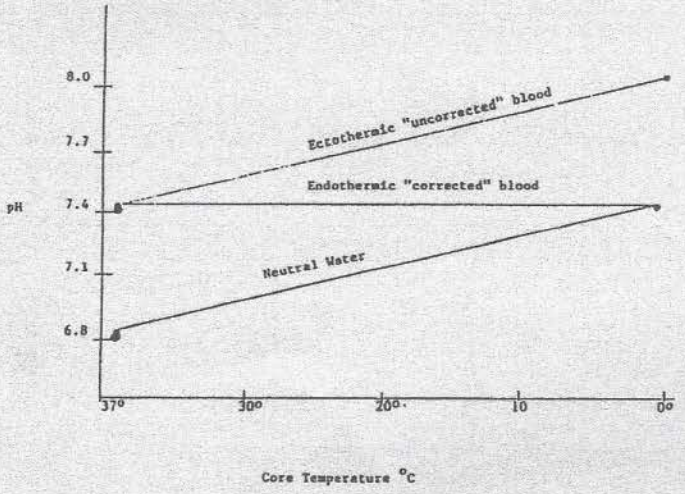
Core temperatures in this series ranged from 35 degrees C to 15.6 degrees C. The majority of the cases (N = 272) and 62 of the 73 fatalities had core temperatures less than or equal to 32.2 degrees C. Ninety-eight of the 169 (58%) patients located indoors were less than or equal to 32.2 degrees C, as were 25/37 (67.6%) immersions, 54/82 (65.9%) trauma cases, 116/174 ethanol ingestors and 128/188 (68.1%) with serious illnesses.

Mortality was not increased despite the fact that the first (0.75 degree C/hr), second (1.17 degrees C/hr), and third (1.26 degrees C/hr) hour rewarming rates for the population over 60 years of age all exceeded the widely recommended 0.55 degree C/hr maximum rewarming rate for elderly patients. There were also no clinically significant differences in treatment selection or mortality profiles by sex.

The pH ranged from 6.4 to 7.7 in this survey, with 77.6% of the cases less than or equal to 7.4. There

were no significant differences in pH by temperature, age, sex, ethanol ingestion or outcomes. However, the CPR group was significantly more acidemic (pH 7.12 vs. 7.27).

The optimal strategy to maintain acid-base homeostasis during treatment of accidental hypothermia is being challenged. Rather than using a "corrected" pH of 7.42 as the ideal at all temperatures, a better intracellular pH reference may be electrochemical neutrality of water. Since the neutral point of water at 37 degrees C is pH 6.8, Rahn hypothesizes that this normal 0.6 unit pH offset between water and blood should be maintained at all temperatures as occurs in ectotherms.[2] The blood pH should rise with cooling as does the neutral water pH, and thus, gases left "uncorrected." (Figure) There is some evidence to suggest better outcomes when this target pH is maintained through addition of CO2 to inspired gas.



The 0.6 pH unit offset from neutral water progressively diminishes with cooling if arterial blood gases are temperature corrected.

Standard cardiac life support was surprisingly successful in the multicenter survey. CPR was initiated in the field in 27 cases with nine survivors. Six of the 14 patients in whom CPR was begun in the Emergency Department survived. The profiles for the total group of 41 receiving CPR differed significantly from the others in rescue location (outdoors), presenting temperature (24.8 vs. 30.0 degrees C), and a variety of tabulated laboratory parameters in addition to pH. Ninety-seven of the 117 patients endotracheally intubated were less than or equal to 32.2 degrees C; none of the 117 developed ventricular fibrillation.

There are ample experimental and clinical data demonstrating that standard closed chest compressions can generate sufficient cardiac output to maintain viability under some hypothermic conditions. Antegrade blood flow in normothermic canine models results from phasic alterations in the intrathoracic pressure and not direct cardiac compression.[3] In a swine model in hypothermic cardiac arrest, the cardiac output, cerebral and myocardial blood flows averaged 50%, 55% and 31% of that achieved during normothermic closed compressions.[4] In this experiment, blood flow did not decrease over time, unlike in the normothermic group. If this occurs in humans, it would help explain the survival of large numbers of patients receiving prolonged closed chest compressions in the multicenter survey.

In addition to adverse rheologic factors affecting blood flow, chest wall elasticity and pulmonary compliance are progressively decreased when cold. If a frozen chest wall is not compressible, sufficient intrathoracic vascular compartment pressure gradients cannot be generated. Myocardial compliance is also reduced in severe hypothermia. Althaus observed during thoracotomy that the heart of one of his three survivors "was found to be hard as stone" and it is inconceivable how effective external cardiac massage could be performed.[5] An attractive hypothesis would consider the cold heart to be a passive conduit for a "thoracic pump" during hypothermic chest compressions.

In conjunction with the Wilderness Medical Society, the study group proposes refinements of the current American Heart Association standards for CPR in hypothermia.[6] Ideal recommendations for basic and advanced life support for hypothermic patients are still unresolved.

Reliable field assessment of the cardiac rhythm in hypothermia without a cardiac monitor is difficult. Peripheral pulses may not be palpable in a bradycardic, vasoconstricted victim and the frequency of misdiagnosis of cardiac arrest is unknown. Therefore, in this setting, any respiration or motion should be accepted as evidence of a perfusing rhythm. Avoid initiating CPR if chest wall depression is impossible, lethal injury or "Do Not Resuscitate" status is documented, or rescuer safety is imperiled.

Apparent rigor mortis, dependent lividity, fixed dilated pupils, and even localized tissue breakdown are unreliable criteria for withholding CPR. Literature review indicates intermittent chest compressions and blood flow may provide adequate oxygenation and metabolic support during some evacuations of severely hypothermic victims.[1] Therefore, withholding CPR only because continuous compression can't be assured en route is not warranted. The lowest temperature documented in an infant survivor of accidental hypothermia is 15.2 degrees C, so there is no consistent minimum temperature which excludes resuscitation. Certainly, identification of frozen intravascular contents should suffice.

Assessment of the treatment modalities reported in this database is ongoing and will be separately

reported. There are a variety of passive external, active external, and active core rewarming techniques currently in wide use to rewarm a heterogenous patient group. Statistical analysis is being done to identify the variables predicting outcome in this survey.

A Hypothermia Outcome Score is being developed which could be useful in future studies to evaluate residual effects of treatment on outcome. The relative weighing of each variable is determined with the likelihood ratio, which is the ratio of two probabilities, the true-positive fraction divided by the false-positive fraction.[7] Future efforts to prospectively validate this instrument will require a prolonged cooperative multicenter approach.

REFERENCES

1. Danzl DF, Pozos RS and Hypothermia Study Group: Multicenter hypothermia survey. *Ann. Emerg. Med.* 1987; 16:1042- 1055.
2. Rahn H, Reeves RB, Howell BJ: Hydrogen ion regulation, temperature and evolution. *Am. Rev. Resp. Dis.* 1975; 112: 165- 172.
3. Nieman JT, Rosborough J, Hausknecht M, et al.: Blood flow without cardiac compression during closed chest CPR. *Crit. Care Med.* 1981; 9: 380-81.
4. Maningas PA, DeGuzman LR, Hollenbach SJ, et al.: Regional blood flow during hypothermic arrest. *Ann. Emerg. Med.* 1986; 15: 390-396.
5. Althaus U, Aeberhard P, Schupbach P, et al.: Management of profound accidental hypothermia with cardiorespiratory arrest. *Ann. Surg.* 1982; 195: 492-95.
6. Standards and guidelines for cardiopulmonary resuscitation (CPR) and emergency cardiac care (ECC): *JAMA* 1986; 255: 2905-2984.
7. Radack KL, Rouan G, Hedges JR: The likelihood ratio: an improved measure for reporting and evaluating diagnostic test results. *Arch. Pathol. Lab. Med.* 1986; 110: 689-93.

INTERNATIONAL HEALTH AND TRAVEL

LEISHMANIASIS

Karl Neumann, M.D.

(Private practice in pediatrics; Associate Clinical Professor of Pediatrics, Cornell Medical College; syndicated columnist on travel medicine.)

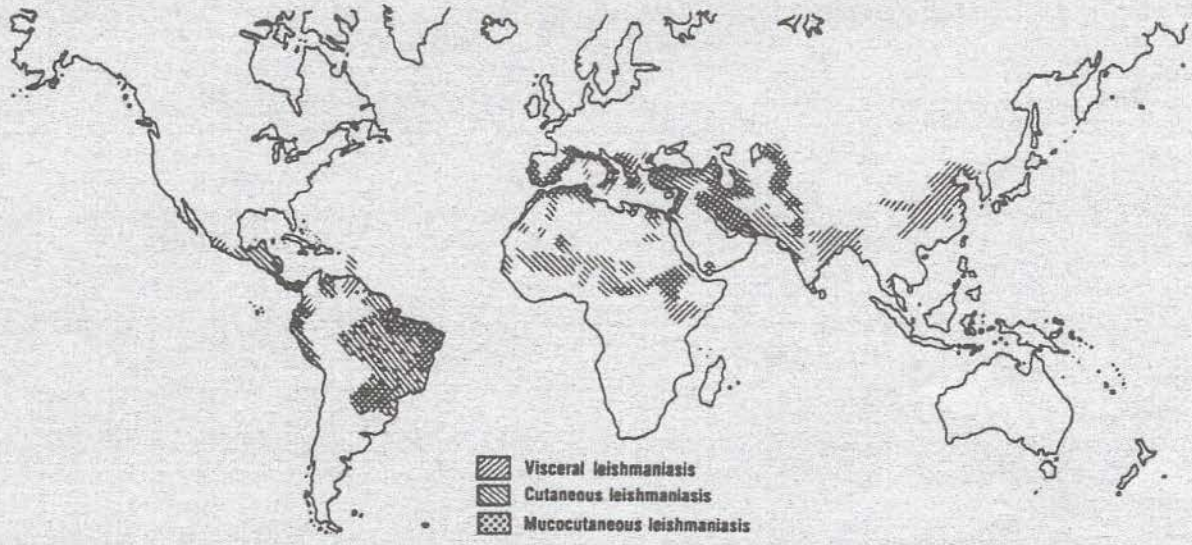
When treating skin lesions that are slow to heal, ask patients about recent trips to the tropics, subtropics and countries bordering the Mediterranean. The diagnosis could be leishmaniasis. Millions of Americans travel to these areas each year, and many immigrants come here from those regions. Statistics are lacking, but the best estimates indicate that hundreds of cases of leishmaniasis are seen in the United States each year. Unfamiliarity with leishmaniasis causes delays in diagnosis and treatment of annoying skin ulcers.

There are three major human types of leishmaniasis: cutaneous (oriental sore), mucocutaneous (espundia), and visceral (kala-azar). Often there is overlap in syndromes. The clinical picture is determined primarily by the infecting species of *Leishmania* as well as host resistance. By far, the most common type of leishmaniasis seen in the United States is cutaneous.

Leishmaniasis is caused by protozoal parasites belonging to the family Trypanosomatidae, genus *Leishmania*. Generally, four types are distinguishable: *L. tropica, L. mexicana, L. brazilensis and L. donovani.*

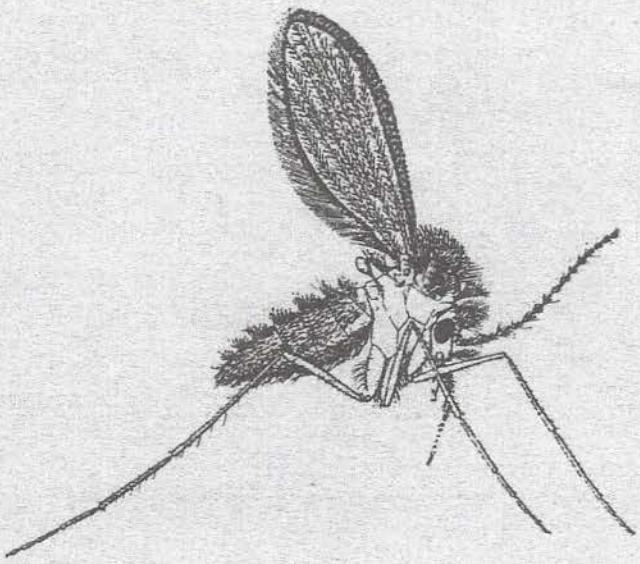
Transmission occurs via several species of sandflies, mainly of the genera *Phlebotomus*. These are 1.5 to 2.5 mm black insects that look like hairy mosquitoes. Reservoirs exist in canines, rodents and other animals.

Female sandflies inject the promastigote (flagellated form of the protozoa) into a mammalian host. In the host, a protozoan enters a histiocyte or macrophage,



Geographic distribution of leishmaniasis.

loses the flagellum, and transforms into an amastigote, which reproduces intracellularly. Parasitism is limited to reticulo-endothelial cells.



Sandflies ingest infected cells containing amastigote forms when they feed on the infected host's skin and peripheral blood. The amastigotes become promastigotes and multiply by binary fission in the gut of the sandfly. After a two to three week period, infective forms migrate to the proboscis, completing the life cycle of the parasite.

The clinical disease depends on whether infection remains localized to the skin, spreads to the mucous membranes of mouth or nose, or throughout the reticulo-endothelial system to the spleen, liver, lymph nodes, and bone marrow.

Cutaneous leishmaniasis results in a chronic ulcerative granuloma of the skin. It is most commonly caused by *L. tropica,* occurring chiefly in Mediterranean countries, Asia, Africa (old world leishmaniasis), and in parts of South America (new world leishmaniasis). There is little difference in the two types except that the new world form may be more virulent. Most of the lesions seen in the United States are contracted in the Mediterranean region – on Greek islands, in Majorca and on the Costa del Sol of Spain. The new world type is most commonly found in jungle areas not frequently visited by tourists.

Lesions generally take many weeks to become visible. Often, the traveler has returned to his home and fails to connect the lesion with the recent travel. The lesions begin as small papules at the site of the bite, usually on exposed areas of the skin, and progress successively to form tubercles, scabs, and ulcers that may grow to 5 cm in diameter with raised borders. There may be solitary or multiple lesions. Healing may occur rapidly without treatment, or the ulcer may persist for years.

The diagnosis can be made in several ways: demonstration of the organisms in smears from the lesions, from biopsy specimens or in cultures; histopathologic examination of biopsy tissue; hamster inoculation; and immunofluorescent techniques. A skin test is also available.

The leishmanin skin test gives evidence of present or past infection and is usually positive three months after onset of lesions. It is read at 48 hours after application, and a response of 5 mm or greater is positive. The test generally does not distinguish present or very recent illness from past illness. The skin test remains positive for many years.

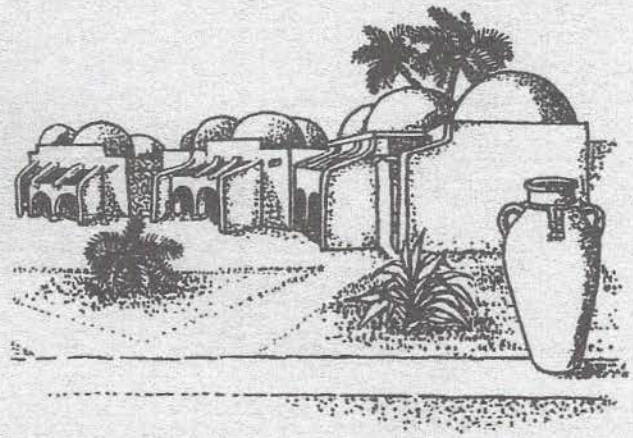
The number of organisms present in lesions varies, and *Leishmania* often are difficult to demonstrate and to identify. The best technique for diagnosis is to culture specimens from active lesions. The organism grows on Schneider's Drosophila medium in about one week or on NNM medium (Nicolle's modification of Novy and McNeal's medium) in about three weeks.

Lesions may be secondarily infected with other organisms. Therefore, positive bacterial cultures do not rule out leishmaniasis.

Although most lesions of old world cutaneous leishmaniasis lesions resolve spontaneously, requiring a few months to 1.5 years, treatment is indicated for large or multiple lesions, to avoid cosmetic scarring for persistent lesions. Treatment of new world cutaneous leishmaniasis is indicated because of the risk of subsequent mucosal disease.

Various modalities of treatment are available. Medication, surgery, and cryotherapy have all been used, and there is considerable debate which is most beneficial. Oral treatment with pentavalent antimonials (sodium stilogluconate – available from the Centers for Disease Control – and meglumine antimoniate) is most commonly used.

Infection usually gives long lasting or permanent immunity.



In endemic areas, infection can be minimized with the use of protective clothing, insect repellents, insecticidal sprays and, at night, window screens or netting around beds. Most bites occur at night.

References

Russo, G. Leishmaniasis. *Lancet,* 1986; Feb 22; 451.

Marinkelle, CJ. The control of Leishmaniasis. *Bulletin of WHO,* 58 (6):807-818 (1980).

Warren, KS and Mahmoud, AAF. *Tropical and Geographical Medicine,* McGraw Hill, N.Y. 1984.

Maegraith B. *Clinical Tropical Medicine,* Blackwell, Oxford, England, 1984.

Moschella, SL and Hurley, HJ. Dermatology, Saunders, 1985. Jong, E. *The Travel and Tropical Medicine Manual,* Saunders, 1987.

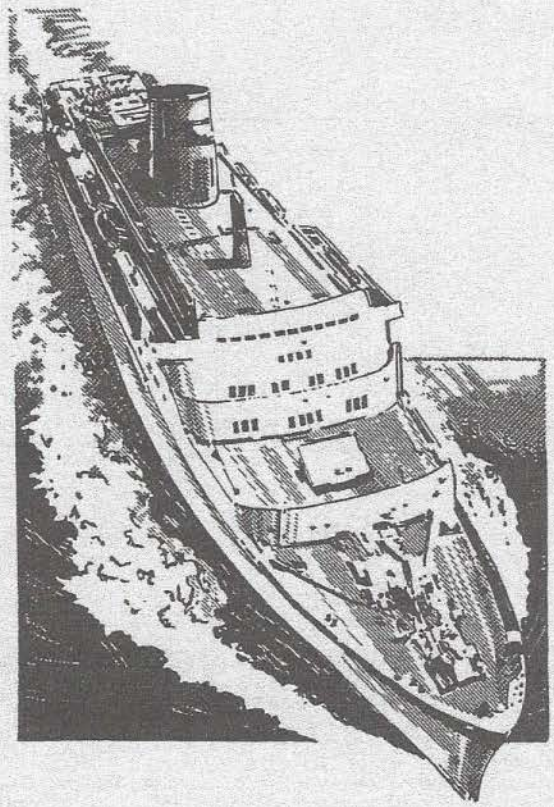
HEALTH INFORMATION FOR INTERNATIONAL TRAVEL

Norman H. Mellor, M.D.

(Family Practice, Corona, CA; International Travel and Expeditions to Study Ornithology)

Three publications offer essential information on health recommendations for international travel. The "bible" for all who give advice for foreign travel is *HEALTH INFORMATION FOR INTERNATIONAL TRAVEL* (H.H.S. No. (CDC) 87- 8280) authored by the Centers for Disease Control (CDC). It is updated every May and can be obtained from the Superintendent of Documents, U.S. Government Printing Office, Washington, D.C., 20402, for $5.00.

To keep with the latest health information and statistics on a wide variety of medical problems, the *Morbidity and Mortality Weekly Report (MMWR)*, also edited by the CDC, may be obtained from the Massachusetts Medical Society, C.S.P.O., Box 9120, Waltham, Massachusetts, 02254-9120. The cost is $26.00 for third class or $45.00 for first class mail and includes a copy of Health Information for International Travel. MMWR is recommended principally for physicians.



To evaluate cruise ships that dock in U.S. ports, review the *Biweekly Summary of Sanitation Inspections of International Cruise Ships*, also by the CDC. Write to the office of The Chief, Sanitation and Vector Control, 1015 N. American Way, Room 107, Miami, Florida, 33132. A detailed copy of the most recent inspection report on an individual vessel may also be obtained from the same office. There is no charge for these.

WILDERNESS EMERGENCY MEDICAL CARE

This is the third section in a series on Wilderness Emergency Medical Services, investigating the need for designated providers with certifiable levels of training, and identifying some of the courses developed to teach wilderness medicine to various providers.

THE NATIONAL SKI PATROL WINTER EMERGENCY CARE COURSE

Warren Bowman, M.D.

(Practice of Internal Medicine, Billings, Montana; National Medical Advisor, National Ski Patrol; Medical Director, National Association for Search and Rescue.)

The National Ski Patrol (NSP) has always been in a unique position compared to other providers of prehospital care. Although patrol first aid rooms usually contain fairly sophisticated first aid equipment, they are not usually near hospitals; at times, patrollers must manage a victim for an hour or more before turning the patient over to the EMS system. Nordic ski patrollers and ski mountaineers are often hours or even days from a doctor or hospital.

The NPS is a nationally chartered organization with 50 years' experience in cold weather and high altitude illnesses and injuries; it is the world's largest winter rescue organization.

Prior to 1985, the NPS required its members to be trained in basic first aid through the American Red Cross Advanced First Aid and Emergency Care course. This course was developed in the 1970's and

Michael V. Callahan demonstrates high angle rescue techniques at the WMS 1987 Annual Meeting in Snowmass, Colorado.

was designed primarily for urban laypersons with quick access to the EMS system. After some experience with it, the NSP found that additional training in the types of illnesses and accidents seen in the mountain environment was necessary. By 1976, the NSP Winter First Aid Course had been developed for the instruction of candidate ski patrollers in the care of high altitude illness and cold injuries, ski injuries, and the special equipment and techniques used by alpine and nordic patrollers.

In the fall of 1985, the NSP took a hard look at its training program and other available programs, including the EMT program. Since none of these was able to fulfill the needs of patrollers without major modification, the NSP decided to create an entirely new course called "Winter Emergency Care (WEC)," reflecting the Patrol's primary concern with the winter environment. A special committee was appointed to produce and oversee the course, a curriculum was drawn up, and pilot courses were taught successfully in the U.S. and in Europe during the 1986-7 ski season.

During the 1987-8 ski season the course will be taught to all 24,000 members.

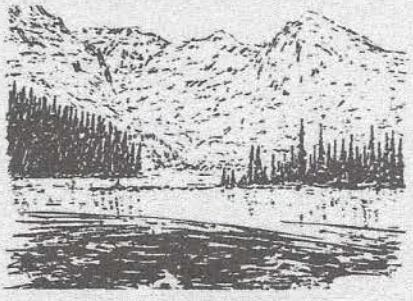
The decision to remain outside the EMS training system was a deliberate one. The NSP feels that its can achieve stronger legal and educational positions by exercising complete control over a course designed to meet the specific needs of its members, rather than by trying to adapt the course from some other organization. The WEC course will be reviewed and updated regularly by four groups: the NSP National Medical Committee (composed of the first aid advisors from the 10 divisions and chaired by the National First Aid Advisor), a committee of expert physicians experienced in wilderness medicine and first aid, and by the NSP Board of Directors, which has to approve each step in the development of the course. The knowledge and skills taught in the WEC Course closely follow the Department of Transportation's National EMS Standard Curriculum (1984 revision), differing only as necessary for application to the wilderness environment.

As National Medical Advisor, I am writing the Manual of Wilderness Emergency Care of the NSP to serve as a textbook for the course; it should be ready for the 1988-9 ski season. The manual is designed to be useful to any person who gives care in a non-urban environment. It is not limited to cold weather problems and will include the assessment and emergency care of all types of wilderness first aid problems; an appendix discusses the care of a victim when definitive medical care is hours or days away—whether due to distance, adverse travel conditions, or difficulties in communication. However, it is not a textbook of backcountry first aid.

The course is divided into 20 lessons; it is anticipated that about 60 hours will be required to complete it. A refresher course will be taught yearly at the onset of the ski season. Major topics include: introduction to winter emergency care, overview of anatomy and physiology, topographic anatomy, victim assessment, basic life support, bleeding, shock, soft tissue and musculoskeletal injuries, burns, specific injuries to the chest, abdomen, upper extremity, lower extremity, pelvis, head, neck, and back; face and eye injuries, medical illnesses, environmental injuries and illnesses, victim extrication and transportation, and legal concerns. Hands-on practical sessions are emphasized, and sufficient time for review and testing is included.

The course is performance rather than time based. Performance objectives are designed to support the learning of basic anatomy, physiology, the relationship of each body system to the function of the body as a whole; in-depth victim assessment for both injuries and medical illnesses; and the improvisation of first aid equipment and techniques. Mechanisms and common patterns of injury are emphasized.

The Winter Emergency Care course is not a complete first aid course. Heat injuries (except for burns), bites and stings, poisoning, water hazards, automobile extrication, and childbirth will not be covered. Conversion courses are being developed to enable a holder of a WEC card to get an EMT card or an American Red Cross Advanced First Aid and Emergency Care card, if desired or required by other employment. There is no prerequisite for entry into the course, other than to be registered with the NSP as a candidate patroller. At the present time, the NSP is not able to offer the course to nonmembers, although there are future plans to make it available to members of other wilderness recreation and rescue organizations upon request.



WILDERNESS FIRST AID COURSES

Steve Donelan

(Active Instructor and Committee Chairman, Local and National Red Cross; Wilderness First Aid Writer and Teacher.)

Since 1935, climber-physicians have been teaching some medical techniques to non-medical members of the Seattle Mountaineers. Since urban-oriented first aid may not prepare rescuers to manage back country accidents, the Mountaineers, in 1968, adapted Standard First Aid to their needs. The National Ski Patrol and other local groups have put together similarly specialized first aid courses. However, there is no national course in backcountry first aid for the general public.

Nearly four years ago, I designed a Wilderness First Aid program, which I teach as a volunteer at the Oakland, California Chapter of the American Red Cross. California SAR units and an increasing number of other Red Cross chapters are adopting my course, which assumes that medical help may be far away and few resources available. Urban first aid is included as a special case, in which medical help is near and resources abundant. So far, I and my co-instructors have taught 20 courses.

Wilderness First Aid (WFA) is a 24-hour course that adapts and supplements most of the American Red Cross (ARC) Advanced First Aid topics. Emergency childbirth, automobile extrication and the pool session are omitted. Graduates get ARC cards in Standard First Aid and chapter cards in WFA. Advanced Wilderness First Aid (AWFA) is a 54-hour course aimed at people who do serious back country activities, especially people responsible for the safety of others. AWFA exceeds the standards for Advanced First Aid (AFA). Graduates get Red Cross AFA cards, and chapter AWFA cards. Though AWFA does not teach people to be their own doctors, it goes beyond teaching what to do until the doctor comes. Back country accidents and their psychological effects may put the entire group (not just the victim) in a survival situation. First aiders with few resources may have to make decisions about on-going care and evacuation. What kind of first aid training prepares people to cope with problems like these?

In AWFA, students learn some physiology with the emphasis on oxygen transport and heat regulation via the circulatory system. How body and mind respond to environmental stress (heat, cold, altitude, dehydration), and the effects of injury or sudden illness on body systems is considered: first-aiders who can understand the underlying process of illness will best recognize medical emergencies and act rationally to stabilize victims.

Biological hazards are discussed, including contaminated water. Students learn how clothing can control heat and moisture exchange with the environment. They design a wilderness first aid kit that meets specific needs.

These topics take 9 hours in AWFA. Psychological responses to accidents and psychiatric emergencies (including drug abuse) is covered in 3 hours. There are 22 hours of skills practice which includes wilderness evacuation, backcountry splinting (using some ski patrol techniques) and bandaging, as well as traditional AFA skills. In all of these sessions, students learn to think and solve problems, not just to memorize information.

But, lectures and classroom skills practice still do not prepare people to perform in an emergency. Problem simulations used throughout the course focus training on overall accident response. Practice of head-to-toe assessment, then writing findings on a standard EMT accident report, teaches students a systematic approach. Simulated accidents, using stage makeup to portray injuries, bring students as close as possible to real situations. Plausible scenarios and realistic acting of victim's behavior increase believability. Psychological problems and rescuer stress are always included, since these areas are often the most difficult aspect of accident management.

In a simulation, students learn to use all their senses, some of which may fade under stress. Giving first aid while reassuring an upset victim requires a combination of skills; students trained to integrate skills in a stressful situation are better prepared to manage a real emergency.

With this kind of training, even large classes can participate actively. During simulations, monitors are silent and invisible. Afterwards, each rescuer has one minute to give an oral report of findings and treatment, followed by equally succinct critiques from the victim and monitor. Another exercise, team splinting and bandaging contests, done in complete silence, teach students to work together without arguing and to become more sensitive to the pattern of activity around them.

Teaching Advanced Wilderness First Aid effectively requires many instructors and aides. Fortunately, the course tends to produce its own ongoing support group. Current guest and assisting instructors (most of whom have taken the course) include naturalists, ski patrollers, EMT's, nurses, a physical therapist and a psychiatrist. Because of the participatory teaching style, instructors often learn as much as the students.

I would like to hear from other wilderness first aid instructors. I am interested in forming a nonprofit group to share information and ideas among persons around the country who are developing Wilderness First Aid courses. This group would focus on how to teach and develop programs in Wilderness First Aid for the general public, using standards passed down by the WMS and other national organizations. The first project would be a quarterly newsletter to communicate ideas, problems, and methods. This will be produced by the volunteer instructors from our Wilderness First Aid program (including three former magazine editors and one professional printer). If you want to receive more information, please write to:

Steve Donelan, P.O. Box 1227, Berkeley, CA 94701

WELCOME

NEW MEMBERS

Rosemary Agostini, M.D., Seattle, WA
James H. Andrisevic, M.D., Minneapolis, MN
John Anshus, M.D., Cardiff By The Sea, CA
Andrew L. Arnold, M.D., Winter Park, CO
Bob Apter, M.D., La Conner, WA
Linda Darlene Armstrong, R.D., Novato, CA
Allan Bach, M.D., Seattle, WA
Julia Ballard, M.D., Atlanta, GA
James T. Barnhart, Elmendorf Air Force Base, AK
Lyn Benjamin, Albuquerque, NM
Paul A. Berger, Jr., M.D., Sioux City, IA
Ronald W. Berry, M.D., Birmingham, AL
David H. Bizot, M.D., Louisville, KY
Leslie C. Bizot, Louisville, KY
Donald G. Bliss, M.D., St. George, UT
Marni Bonnin, M.D., Royal Oak, MI
Victor W. Brown, Lake Powell, UT
John A. Bryan, M.D., Atlanta, GA
Suzanne Brissette, M.D., Montreal, Canada
R. John Bull, M.D., Oxnard, CA
Joseph J. Casey, M.D., Ft. Lauderdale, FL
Edward W. Ceatruk, New York, NY
Suzette Aronade Chafey, Van Nuys, CA
Garrick Chang, Los Angeles, CA
Ward Colignon, M.D., Bremerton, WA
J. Alex Crane, M.D., Oakland, CA
Mark G. Constance, M.D., Tacoma, WA
Bruce Coorpender, Portland, OR
Stephen Corn, M.D., Boston, MA
Peter W. Crecelius, M.D., Orofino, ID
R. Kemp Crockett, M.D., Washington, DC
J. Ben Crumpler, M.D., Greenville, SC
Carolyn Cubberly, Powell, OH
Craig W. Curtis, M.D., Rome, NY
Helene Darveau, M.D., La Prairie, Canada
Joseph J. Deck, Omaha, NE
John Deliduka, M.D., Leavenworth, WA
Robert W. Derlet, M.D., Sacramento, CA
Jon Dragan, Thurmond, WV
Scott Duloit, Nashville, TN
Marc J. Dumas, Syracuse, NY
John I. Ellis, M.D., Hayward, CA
Mark Epstein, New Haven, CT
Tom Ettinger, M.D., Seattle, WA
Jo Fletcher, M.D., Albuquerque, NM
Arthur J. French, II, M.D., Sitka, AK
Chris Frolking, M.D., Arcata, CA
Martin T. Girling, DPM, Houston, TX
Robert Del Grande, M.D., London, Ontario
Steven M. Green, M.D., Loma Linda, CA
Marc A. Grinberg, M.D., Sarasota, FL
Michael D. Grossman, M.D., Salt Lake City, UT
Michael de Haan, M.D., Albuquerque, NM
Marek J. Hann, M.D., Madison, WI
J. Brevard Haynes, M.D., Nashville, TN
Lynne Heidsiek, M.D., Hunt, WV
Tom Higginbotham, D.O., Boise, ID
Andrew D. Horpeniuk, M.D., Irvine, CA
Daniel D. Houssiere, D.O., Arcadia, CA
Roy K. Howell, M.D., Marysville, CA
Christopher Hummel, Orange, CA
Eugene Jakubowski, M.D., Elk Grove Village, IL
Randy Kemberling, Playa del Rey, CA
Thomas Kinane, M.D., Richland, WA
Herbert F. Kincey, Jr., Santa Fe, NM
Max Lebow, M.D., San Bernardino, CA
Lewis Leibovich, M.D., Santa Rosa, CA
M. Lertzman, M.D., Winnipeg, Canada
Lucy W. Loomis, M.D., Denver, CO
Mark S. Louden, M.D., Arlington, TX
Wayne S. Ludkiewicz, M.D., Beaver, PA
Kirk Lufkin, M.D., Minneapolis, MN
Kevin MacMahon, M.D., Ormond Beach, FL
Robert R. Maisel, M.D., Presque Isle, ME
Ed Makoski, M.D., Atlanta, GA
Pablo R. Marquez, M.D., Tecate, CA
Nancy Lee Marriner, M.D., Albany, CA
E.M. McAninch, M.D., Camas, WA
Mike McConnell, M.D., Philadelphia, PA
Peter G. Markos, D.O., Newport News, VA
Giles Yancey Mebane, M.D., Mebane, NC
Kenneth T. Miller, PhD, Arlington, VA
Stephen M. Miller, M.D., Beverly Hills, CA
Steve Nash, Kentfield, CA
Karl Nicholas, M.D., Monterey, CA
Dirk F. Niles, St. Paul, MN
Larry J. Orosz, M.D., Rolling Hills Estates, CA
Andrew C. Peet, M.D., Emsworth, PA
L.H. Pendergrass, M.D., Palm Harbor, FL
James L. Pita, Jr., M.D., Wilson, NC
Kent L. Pomeroy, M.D., Phoenix, AZ
Susan Price, M.D., Seattle, WA
Gregory C. Putalik, M.D., Harbor Springs, MI
Jack Putnam, M.D., Kent, WA
Drummond Rennie, M.D., Chicago, IL
Joseph Ripperger, Tulsa, OK
Hector Santana, M.D., Rio Piedras, PR
Fred Schlachter, M.D., Oakland, CA
Kim E. Schmitt, M.D., Birmingham, AL
Richard Sedlak, M.D., Waipahu, HI
James N. Skeen, PhD, Decatur, GA
Gary Smith, Beaverton, OR
Susan Snider, M.D., Spruce Pine, NC
Daniel Spaite, Tuscon, AZ
David Spaite, Tuscon, AZ
Francis S. Stellaccio, M.D., Bayville, NY
Charlotte Strong, Hudson, OH
Laird S. Swensen, M.D., Salt Lake City, UT
Karl Taylor, M.D., Reno, NV
Joyce B. Thisse, M.D., Spruce Pine, NC
James R. Uhl, D.O., Parkville, MO
Reuben W. Uniat, M.D., Midland, TX
Arno Vosk, M.D., Williamsport, PA
Alan N. Weiner, M.D., Freeport, ME
Brent Wesenberg, M.D., Bakersfield, CA
Susan Whitaker, D.O., Olympia Fields, IL
Dennis C. Whitehead, M.D., Marquette, MI
Arthur C. Wittich, M.D., Sierra Vista, AZ
James N. Wurgler, M.D., Yosemite National Park, CA
Ray Yip, M.D., Atlanta, GA
James B. Young, M.D., Houston, TX