

The requisite clinical skills for practicing mountain medicine encompass several specialty domains. But it is the precision with which these skills can be adapted to mountain medicine's unique requirements that determines the quality of the clinical outcome. Preventing accidents and life-threatening situations and performing prompt, efficient rescues are the essence of mountain medicine.

Early scientific expeditions to high-altitude levels, beginning with the ascent of Mt Blanc in 1787 by Geneva physicist Professor de Saussure, are reviewed in the introductory chapters. Also reviewed is the evolution of our understanding of human acclimatization to altitude.

When Jean Moine landed his helicopter on the summit of Mt Blanc in 1955, the modern era of mountain medicine began. The text explains the impact that this trip had on mountain transport. In fact, it led to the first airborne mountain rescue in 1957.

Modern mountain rescue strategies are described, including communication, transport, evacuation, and medical treatment. The major climate conditionsaltitude, solar radiation, cold, ice, snow, and lightning-are defined as well. The physiology and pathology relating to the climatic conditions are presented clearly in the text and in the numerous graphs and tables featured in this work.

The photographs of frostbite prove particularly helpful. Several case presentations, with photographs of frostbite taken in its various stages through resolution, are included here, too.

Hiking and trekking, downhill skiing, and mountaineering are emphasized in Part II. The editor has also included those areas that should be studied further: the mechanics of muscle protein loss at high altitude; climbers' acid-base status at extreme altitudes; and the residual impairment of the mountain climbers' central nervous system function after ascents to very high altitude.

A well-rounded book, Color Atlas of Mountain Medicine not only details immediate and longterm treatment of specific injuries, but it also features recommendations for supplies that any physician who spends time in the mountains should keep in his or her little black bag. The comprehensive reference list is helpful to those readers wanting further details. I recommend this interesting, helpful book.

> BOB ADAMS, DO Team Physician USA Track and Field Team

medi-notes

Evaluating the Global Deterioration Scale as a means for staging Alzheimer's disease

The Global Deterioration Scale has been used extensively since its 1982 publication. Its stages are based on implicit assumptions concerning the linearity, temporality, and interdependence of cognitive, functional, and behavioral impairment in Alzheimer's disease.

In this study, the authors evaluated the validity of these assumptions and tested the hypothesis that psychopathology and functional impairment would occur in earlier stages than the Global Deterioration Scale predicts. Their analyses were based on data from 324 patients with Alzheimer's disease, selected from a registry. Among the data included were descriptive statistics on the frequency of psychiatric symptoms and difficulties with daily living activities; and logistic regression, with symptoms and functional impairment as independent variables, to test for significant changes in patients' status between stages of the Global Deterioration Scale.

More than 50% of the patients at stage 2 displayed psychopathology; 32% had two or more symptoms. The significant increase in psychiatric symptoms occurred between stages 3 and 4 and 5 and 6.

(continued on page 807)



Based on these findings, psychiatric symptoms and functional impairment occur earlier than the Global Deterioration Scale predicts. The rate of change is also different from that specified by the scale. Researchers conclude that separate scales are required to describe cognitive, clinical, and functional status to best describe this illness at least until more sophisticated, multidimensional instruments are available.

Eisdorfer C, Cohen D, Paveza GJ, et al: An empirical evaluation of the Global Deterioration Scale for staging Alzheimer's disease. Am J Psychiatry 1992;149:190-194.

Link between thiazideinduced electrolyte abnormalities, ventricular arrhythmias?

A total of 233 hypertensive men, aged 35 to 70 years, participated in this double-blind, randomized, controlled trial to test the relationship between thiazide-induced electrolyte abnormalities and the prescription of potassium-conserving therapy.

Participants were withdrawn from prior diuretic treatment and were given oral potassium chloride and magnesium oxide. They were then randomized to receive 2 months' treatment with hydrochlorothiazide; hydrochlorothiazide with oral potassium; hydrochlorothiazide with oral potassium and magnesium; hydrochlorothiazide and triamterene; chlorthalidone; or placebo.

Ventricular arrhythmias were measured using a 24hour Holter monitor; serum and intracellular potassium magnesium levels were measured as well.

Of the 233 participants, 212 (91%) completed the study. Serum potassium levels were 0.4 mmol/L lower in the hydrochlorothiazide group than in the placebo group (P < .01). This mean difference was not affected by potassium supplementation, potassium and magnesium supplementation, or triamterene supplementation. However, the supplements did prevent the occasional occurrence of marked hypokalemia; all 12 men who had serum potassium levels of 3.0 mmol/L or less were among the 90 men who received diuretics without supplementation (P < .01).

Similarly, randomized treatment did not affect the overall proportion of men with ventricular arrhythmias; however, a twofold increase did occur in the proportion of arrhythmias among the 12 men with serum potassium levels of $3.0 \, \text{mmol/L}$ or less (P = .02).

Hydrochlorothiazide did not reduce serum magnesium and intracellular potassium and magnesium levels. No relationship was found between the serum magnesium and intracellular potassium and magnesium levels and the presence of ventricular arrhythmias.

The authors conclude that hydrochlorothiazide treatment (50 mg/d) does not cause marked hypokelmia or ventricular arrhythmias in most hypertensive patients. Nonetheless, hypokalemia will develop in some patients after they begin diuretic therapy; therefore, serum potassium levels should be monitored and potassium-sparing strategies used when indicated.

Siegel D, Hulley SB, Black DM, et al: Diuretics, serum and intracellular electrolyte levels, and ventricular arrhythmias in hypertensive men. *JAMA* 1992;267:1083-1089

Treatment for adults with relapsed or refractory lymphocytic leukemia

Sixty-six adult patients were treated for relapsed or refractory acute lymphocytic leukemia (ALL). The induction treatment consisted of a Phase 1 with vindesine (3 mg/m² intravenously [IV]) on days 1, 8, and 15; daunorubicin (45 mg/m² IV on days 1, 8, and 15; erwinia-asparaginase (10,000 U/m² IV on days 7, 8, 14, and 15; and prednisone (60 mg/m²) orally on days 1 to 21. Phase 2 treatment consisted of cyta-

(continued on page 808)

rabine 3000 mg/m² as a 3-hour infusion 2 times daily on days 1 to 4. This dosage was reduced to 1000 mg/m² in patients older than 50 years of age. Etoposide (100 mg/m² IV) was administered on days 1 to 5

Side effects occurring during Phase 1 were predominantly hematologic with subsequent infections. In Phase 2, some patients also had gastrointestinal, cutaneous, ocular, and hepatic toxicity. Five patients died during Phase 1, and another patient died during Phase 2. Five of the patients had T-cell ALL. Thirtyfour (64%) of 54 patients in their first relapse had a complete remission with a median disease-free survival (DFS) of 2.9 months. The median overall survival was 6.6 months. Seven (58%) of 12 patients with primary refractory disease, a second relapse, or relapse after bone marrow transplantation (BMT) had complete remission. The complete remission rate and survival after the first relapse was significantly better in patients who had been in complete remission for more than 18 months, compared with patients with a shorter preceding remission.

The leukocyte count was a second significant, but not independent, risk factor. A negative correlation existed between the leukocyte count and the duration of the preceding remission period. Duration of the preceding remission period was, in fact, the major prognostic factor for survival in multivariate analysis.

Twenty-two patients received a BMT. At the time this study was reported, none of the nine patients who underwent autologous BMT was alive and disease-free; 5 (38%) of 13 patients who underwent allogeneic BMT were alive.

Based on these outcomes, the authors conclude that this treatment efficiently induces remissions with tolerable toxicity. Furthermore, the remission duration should be improved by optimized consolidation treatment.

Freund M, Diedrich H, Ganser A, et al: Treatment of relapsed or refractory adult acute lymphocytic leukemia. *Cancer* 1992;69:709-716.

advertisers' index

Abbott Laboratories Biaxin, 718-720

American Osteopathic Association 36th Annual Research Conference,

Ciba Pharmaceutical Company Lotensin, 706-709 Transderm Scop, 673

Curatek Pharmaceuticals MetroGel, 711, 712

Deborah Heart and Lung Center, 803

Flint Osteopathic Hospital, 754 Geigy Pharmaceuticals Lopressor, 687, 688, 791, 792

Lopressor, 687, 688, 791, 792 Voltaren, 723, 724

Helen Keller National Center, 805 Lederle Laboratories

Verelan, 761, 762 Eli Lilly and Company

Ceclor, 696-698

McNeil Consumer Products Co. Pediaprofen, 685, 686, 705 Tylenol, Cover 3

Muro Pharmaceutical, Inc. Bromfed, 703, 704 Guaifed, 721, 722

Parke-Davis Lopid, 726-728

Pfizer Labs Division Feldene, 714-716 Procardia XL, 699, 700 Zithromax, 745-753

Roche Laboratories Medical Director's Page, 713

G. D. Searle and Co. Calan SR, 771-774 Maxaquin, 675-682

SmithKline Beecham Augmentin, Cover 4 Relafen, 691-695

Student Osteopathic Medical Association (SOMA), 784

Texas College of Osteopathic Medicine, 803

The Upjohn Company Xanax, Cover 2-2

Whitehall Laboratories Inc. Advil, 670, 671