The Newsletter of the International Society for Mountain Medicine

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International Society for Mountain Medicine

The International Society for Mountain Medicine, founded in 1985, has the following goals: to bring together physicians, scientists and allied professionals interested in mountain medicine; to encourage research on all aspects of mountains, mountain peoples and mountaineers; to organize and co-organize international scientific meetings and publish a newsletter to spread scientific and practical information about mountain medicine around the world.

YOUR MEMBERSHIP FEE FOR THE YEAR 2000 IS DUE WITH THIS ISSUE. PLEASE RETURN THE MEMBERSHIP FORM WITH YOUR SUBSCRIPTION FEE.

FROM THE EDITOR

The mountains that inspired many of the members of the ISMM have been unchanged for most of the past 1000 years, and will undergo little significant change in the new Millenium, excepting some further glacial recession and the continuing scars caused by humans exploiting their environment. In the context of our mountain environment, the ISMM has had little impact for most of these past 1000 years but has now at last attained a modest decade and a half of development and influence in mountain medicine. At the start of the Millenium, it is good for all of us to reflect on the past, and to look to the future. If we remain inspired by mountains and the physiology and medicine of the people who visit them or dwell on them, we can look forward to a share in the adventure of life and science and medicine that resides in those high places.

AP

ISMM PRIZE

A prize of 1500 SFr (approximately 1000 US$) will be awarded to a young investigator for research in the field of mountain medicine, published or accepted for publication in a scientific journal between July 1st 1998 and March 31st 2000. The prize will be awarded at the IVth World Congress of Mountain Medicine and High altitude Physiology held in Arica, Chile, from October 1-6, 2000. The winner will have free registration at the meeting, and travel expenses up to 1500 SFr will be paid by ISMM. Deadline for submission is March 31st, 2000. For further instructions consult the website of ISMM.

FROM THE PRESIDENT

Looking back at the end of the millenium reminds us that high altitude medicine is a very young field that has stated to attract an increasing number of physicians only at the end of this century. The development of this new medical discipline was driven by man's exploitation of the accessible high mountain areas around the world.

Outdoor sports, among them mountaineering and trekking boom also thanks to the economic development and increased leisure time in many parts of the world. Societies for mountain, wilderness or travel medicine were founded over that last 20 years. Although chronic exposure to high altitude was a concern for many millions of people during the last 1000 years it remained mostly a neglected issue until aviation and tourism touched these areas.

It is and will be difficult for societies of mountain medicine to establish themselves and get recognition in the medical and scientific community for several reasons. First of all it is a broad inter-disciplinary area that is defined by an environmental factor rather than a medical category or skill. Furthermore its economic impacts for industry or for health care in populations living at moderate or low altitude are small - a fact that we realise when trying to raise money for projects. Finally, there are few physicians around the world who have an interest or who work in high altitude medicine or physiology. In addition these few are very unevenly distributed around the world. In some countries there are several hundreds organised in national societies, in others there are so few that national organisations do not make sense. In my opinion it will be essential for the development of mountain medicine to combine the efforts and forces of national societies and give a home for those not organised nationally. This is the role for ISMM as stated on the front page of the news letter. Thanks to the fascinating possibilities of world wide electronic communication it has become much easier to realise this goal and we should not miss this chance. I am confident that we will be successful and I hope that you share my vision of the role of ISMM and will renew your membership for 2000.

Below is some information about a new Journal that begins life shortly under the editorial direction of Professor John West. We will publish further details and a review of the first edition in the April Newsletter.

Announcement - a new Journal for the new Millenium

HIGH ALTITUDE MEDICINE AND BIOLOGY *is* a quarterly peer-reviewed journal that publishes original research articles, review articles, and short communications. Contributions are welcome in the fields of high altitude diseases and other high altitude life sciences, including physiology, pathology, comparative biology, anthropology, evolutionary biology, and human and animal ecology. The Journal has a strong interdisciplinary coverage.

Submit manuscripts by sending one original plus three copies and three sets of the original figures. Address manuscripts to: John B. West, M.D., Ph.D., Editor-in-Chief, Professor of Medicine and Physiology, Department of Medicine 0623A, University of California San Diego, 9500 Gilman Drive, La Jolla, CA 92093-0623; Tel: 858-534-4192; Fax: 858-534-4812; E-mail: jwest@ucsd.edu

CHRONIC INTERMITTENT EXPOSURE TO HIGH ALTITUDE: THE VIEW FROM MAUNA KEA

The summit of Mauna Kea is the highest point in the Pacific Basin. Rising over 30,000 feet from the ocean floor, this dormant volcano forms the bulk of the landmass of the Big Island of Hawaii and attains a summit elevation of 13,796 feet (4205 m). The summit is acknowledged as the world's pre-eminent terrestrial site for optical, infrared and sub-millimeter astronomy: exceptionally sharp celestial images are obtained because the summit is above 40% of the earth's atmosphere. A tropical inversion cloud layer below the summit isolates the observatories from interference by water vapour, which absorbs infra red and submillimeter radiation, and light and atmospheric pollution from sea level habitation. Rapid access to the mountaintop (one and a half-hour journey by road from sea level) facilitates the operation of the telescopes.

The summit area is operated as the Mauna Kea Science Reserve by the University of Hawaii's Institute for Astronomy. In the late 1970's, the Royal Observatory Edinburgh (R.O.E) constructed the United Kingdom Infrared Telescope (U.K.I.R.T), at 3.8m the largest infrared telescope in existence. As was the established practice with the existing observatories on Mauna Kea, the telescope was to be operated by staff resident at sea level. Following a case of high altitude pulmonary oedema (HAPO) on the mountain and an episode when workers were stranded at the summit by a snowstorm, concern was expressed by both the trade unions representing the work force and the R.O.E management regarding the safety and efficiency of operating a complex scientific facility at the summit altitude. Consequently, in 1980 the Science and Engineering Council of the United Kingdom commissioned a two-year study at the telescope (1). The study was supervised by the late Professor Donald Heath, author of Man at High Altitude (2) and High-Altitude Medicine and Pathology (3). This paper relates to that study and the experience of chronic intermittent exposure to hypoxia in the community of workers on Mauna Kea.

Study Details

The reduction in the optical thickness of the atmosphere at high altitude occurs concomitantly with falls in the atmospheric pressure and in the partial pressure of oxygen in the ambient air. At the summit of Mauna Kea, barometric pressure is 465 Torr (611 mbar), 61% of the sea level value, and the partial pressure of oxygen in moist inspired gas in the lung (PIO_2) is reduced to 88 Torr (115 mbar) compared to the sea level value of 149 Torr (196 mbar) (4).

By 1980, two patterns of work had evolved on the mountain. The UKIRT was operated on a shift schedule: the shift workers' schedule involved 40 days mountain work followed by 40 days office work at sea level. Mountain work comprised four shifts on the mountain with each shift lasting 5 working days plus an initial night of acclimatisation at 3000m. Whilst on the mountain workers ate and slept at the mid level facility of Hale Pohaku (3000m; 9300ft) which is the present day location of the Onizaku Center. Mountain shifts were interspersed by five days rest at sea level. (Forty-one "shift workers" were studied during a total of 134 shifts.)

Staff manning the NASA infra red telescope (IRTF) commuted daily leaving sea-level at 0800 and arriving at the summit at 10.00 allowing for 30minutes respite at 3000m; the summit was vacated at 1600 hours. (Data were collected from nineteen "commuters" during 44 visits to the summit).

Study Findings

The majority (80%) of UKIRT shift workers experienced altitude sickness symptoms on the first day at the summit. Headache and breathlessness were the commonest complaints followed by insomnia, lethargy, poor concentration and impairment of memory. By the fifth day, 60% of shift workers were free of symptoms whilst the remainder reported minimal exertional dyspnoea or headache. Day commuters fared better; 60% were asymptomatic on arrival at the summit. However after a 5 hours work at 4200m, 35% of commuters experienced dyspnoea and 25% suffered headaches. Cerebral symptoms (poor concentration, lethargy and confusion) were reported by 10% of commuters.

On Mauna Kea, one case of HAPO was diagnosed during the 2-year study. Calculated in terms of the 2000 ascents per annum to the UKIRT, this low incidence of HAPO may reflect the lack of physical exertion involved in ascent to 4200m. A single episode of high altitude cerebral oedema (HACO) occurred during the Mauna Kea study.

Arterialised capillary blood samples were collected from 28 IRTF commuters during 35 ascents after approximately 3 hours at the summit; the mean arterial

oxygen tension (PaO_2) was 39.9 mmHg (5.3 kPa). On the first day of the UKIRT shift on Mauna Kea, the mean PaO2 was 42 mmHg (5.6 kPa) measured in 27 shift workers on 40 ascents. Mean PaO_2 rose significantly to 44.4 mmHg (5.9 kPa) by the fifth day. Thus mean PaO_2 levels were higher in the shift workers (day 1) than in the commuters: a night of acclimatisation at 3000m appears to confer benefit in this regard. The benefits of the shift schedule persisted and after five days on the mountain, shift workers had a significantly higher PaO_2 compared to commuters and fewer symptoms were reported.

Psychometric tests of numerate memory (Wechsler digit span tests), a skill much prized by the scientific staff members, and of motor speed and processing of information were studied. In shift workers, performance of one of the digit span tests was impaired on the first day however by the end of the 5-day shift there was no difference between the test results on the mountain and at sea level. Deterioration in the performance of both sets of tests occurred amongst the commuters. Thus although the commuters were less affected clinically than shift-workers, commuters were only at an advantage if their exposure to 4200m altitude was limited to less than 6 hours. The disadvantage of the commuters work schedule was that the commuters did not acclimatise.

Anecdotal accounts from the UKIRT personnel suggested that tolerance of hypoxia improved sequentially through the series of shifts i.e. more discomfort was experienced during the first shift of mountain work, following a sojourn of 40 days at sea level, than in subsequent shifts after only 5 days break. An analysis of the data according to whether the shift was worked after 40 or 5 days at sea level, showed no difference in terms of symptom scores or a variety of physiological measurements (tests of pulmonary function, electrocardiographs, exercise tests, haematological indices.). The failure to substantiate the impression of a follow-on of acclimatisation between shifts could be because the parameters employed, which did not include blood gas analysis, were inappropriate or too insensitive to detect a difference.

Individual response to high altitude exposure.

Experiments at simulated high altitude in decompression chambers (5) have substantiated the impression that people react to high altitude exposure in an inherently individual manner. The circumstances of the work on Mauna Kea, involving the same individuals ascending to the same altitude by the same route on different occasions, allowed a study of this phenomenon in the 'field' (6). On Mauna Kea, PaO_2 recorded in the shift worker group ranged from 33mmHg (4.4 kPa) to 57 mmHg (7.6 kPa) at the summit . The worker with the highest PaO_2 during the first ascent (52.5mmHg; 7.0 kPa) recorded the highest PaO_2 on a subsequent ascent (57 mmHg;7.6 kPa): similarly one subject recorded the lowest PaO_2 (33mmHg; 4.4 kPa : 38 mmHg; 5.1 kPa) on both ascents. Although some workers were symptom-free, whilst others suffered altitude sickness, there was a significant correlation in the order of subjects ranked according to symptom scores in the first day of two shifts: 5 subjects were symptom-free on both occasions. Performance in memory and psychomotor tests were similar within each individual on the two ascents. Anomalies did occur - the sufferer of the documented case of HAPO had performed well on all previous occasions and continued to do so subsequently. Nevertheless, individual workers were remarkably constant in their response. One individual was so consistently affected by acute mountain sickness symptoms despite various strategies to ameliorate his distress (e.g. prolonged period of acclimatisation at Hale Pohaku, abbreviated periods at the summit) and prescription of acetazolamide prophylaxis that he was eventually excused mountain duties. In the 2 year study this was the only occasion when acetazolamide was prescribed for one of the staff: there was a reluctance to medicate the staff to facilitate work at high altitude which was shared by individual members of the staff and the managers of the telescope.

Conclusion

In June 1970 astronomical observations commenced at the University of Hawaii 2.2m optical telescope, the first major telescope built on the site. At the time of the completion of this study in 1982, there were four major telescopes at the summit. The success of these telescopes and the recognition by the scientific community, and various national grant-awarding bodies, of the excellence of the site has led to the construction of further observatories. In 1999, there are ten major telescopes, two of which are in an engineering phase, and an eleventh is under construction. No facility, including the most recently constructed, employ oxygen enrichment of the working environment although the capability exists at the Canada-France-Hawaii and the Keck telescopes; neither is this provision available at the dormitory and living facilities at Hale Pohaku.

In 1982, when the results of the study were published in medical and astronomical journals (7,8), the calculation was made that assuming a complement of 3 day staff and 3 night observers approximately 2,000 "man-days" were spent each year at UKIRT: on average, each man-day constituted 9 hours at the summit. Observing practices change as technology advances nevertheless, in 1999 staffing requirements for UKIRT still necessitate 2000 man-days per annum. Overall, 30,000 man-days per annum are spent at the major telescopes on Mauna Kea, the increased numbers reflecting the demands of engineering and construction at the three new facilities.

There has been a single cardiac death at the summit amongst the telescope personnel. A 37 year old astronomer, a smoker and diabetic, suffered a

myocardial infarction after staying at the summit all night and working through the days. Post mortem examination revealed extensive coronary artery disease. Following this fatality, revised health and safety screening protocols were established. All UKIRT mountain personnel undergo a medical evaluation at sea level, which includes physical examination, comprehensive blood screen, resting ECG, chest X-ray, pulmonary function tests and a maximal treadmill stress test. The studies are recommended yearly for individuals above age 40, every two years for those between the ages of 30-39 and every three years for those younger than age 30. Two years ago a 40 year old construction worker suffered a fatal cardiopulmonary arrest at the summit: construction company employees are not required to undergo the rigorous screening programme described. (Dr D.I Lim. F.A.C.P. Cardiologist, Hilo. personal communication).

Confidence in the safety and effectiveness of the Mauna Kea site as a major astronomical facility has grown since the first observations in 1970. The initial trepidation of the telescope staff at the prospect of working in a hypoxic and hypobaric environment has been supplanted by knowledge of the consequences of altitude exposure, and the awareness that problems can be predicted and coping strategies developed. The converse 'machismo' attitude of denial of the hazards of altitude has been replaced by respect for the mountain site and acceptance that people react to altitude exposure in an individual manner irrespective of age, fitness, 'toughness' or gender. Inevitably, the confidence of the workforce – technical, scientific and managerial – in operating the telescopes on Mauna Kea has been greatly facilitated by ready access to high quality medical care in the event of an emergency. Within 2 hours of leaving the summit by road, a victim of HAPO or HACO could be undergoing MRI or CT scanning at sea level in Hilo: two other CT scanners are available on the leeward side of the Big Island and a second MRI scanner is planned. The availability of a high standard of clinical medical practice has been a great reassurance to the visiting scientists and permanent telescope staff, both those who originate from the United States and from farther afield in Europe and Asia.

Ambitious plans are afoot to construct a telescope at an altitude of 16,400 (5000m) in the Atacama Desert in northern Chile. The Atacama Large Millimeter Array (ALMA) will image the universe at millimeter wavelengths between radio and infrared spectral regions and will consist of thirty–six 12-meter antennas arranged in a circular configuration. The telescope will be a major development for astronomical science furthering investigation of the origins of galaxies and stars and 'imaging cosmic dawn"(9). At the proposed location of ALMA (barometric pressure 419 Torr) the PIO2 is 78 Torr (102 mbar) compared with approximately 88 Torr (115mbar) at 4200m: workers at ALMA will be exposed to 11% less oxygen than the Mauna Kea telescope operators. (4). The ALMA project will reopen all the questions posed by the construction of telescopes at high altitudes – the best work schedules for the safety of the staff and the efficient operation of the facility, access to the site, rapid evacuation from the site in the event of an emergency, provision for staff in the event of a stranding , the availability of adequate medical services and the need or not for supplemental oxygen in living and working quarters etc. The success of the project will depend on the solutions to these problems as the resounding success in Hawaii has depended on the solutions that have evolved on the summit on Mauna Kea.

Peter Forster, UK

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CARDIAC ARRHYTHMIA AT HIGH ALTITUDE: EFFECT OF AGING

The impression generally reflected in mountain medicine texts that cardiac arrhythmia is relatively rare at altitude is largely based on electrocardiographic studies at rest. [1] However, a survey of studies under conditions of exertion documents the presence of some cardiac arrhythmia at simulated high altitude or in the field in young normal subjects. The incidence correlated positively with the degree of elevation, and negatively with the degree of acclimatization.[1] As there was no published information on cardiac rhythm in older normal subjects climbing at altitudes, I recorded Holter monitor electrocardiographic data on myself on ascent and descent of Mt. Kilimanjaro (5895 m) over 5 days in 1986 at age 65.[1]. A battery of cardiac studies at sea level, including coronary arteriography and thallium exercise stress showed no abnormality or arrhythmia at that time. During the ascent from 4710 to 5895 m there were frequent ventricular complexes predominantly of left ventricular origin, and multiple runs of ventricular bigeminy increasing in frequency and duration until the peak was reached. Ventricular ectopy was markedly reduced on initiation of descent, and virtually disappeared within 3 hours thereafter.

To determine the effect of age progression on cardiac rhythm under the same conditions I made an ascent of Mt. Kilimanjaro over the same route in 1996 at age 75. Sea level cardiac studies showed little change as compared to those in 1986 except that exercise capacity was less, and there were some degenerative changes in the mitral and aortic values on echocardiographic study. On ascent electrocardiogram (Holter Monitor), oxygen saturation (finger oximeter), and blood pressure (digital automatic) were recorded. A two channel Holter Monitor was employed to facilitate diagnosis of left vs right ventricular ectopy and measurement of P wave amplitude. On this occasion I turned back on reaching an altitude of 5100 m, because of increasing dyspnea and a mistaken impression that my heart rate was abnormally slow (oximeter dysfunction).

RESULTS

Holter data are displayed in Table 1. Maximal heart rate during climb was 85% of maximal rate during treadmill exercise test at sea level, and heart rate during sleep was comparable to that at sea level. Frequency of premature complexes of left ventricular origin (LV, VPC's) increased progressively at higher altitude during climb at rest, and during sleep. During ascent, at the highest altitude, a maximum of 56 complexes per hour developed, falling to 10 per hour shortly after initiation of descent. Ventricular complexes of right ventricular origin (RV, VPC's) were relatively infrequent, with no clear relations to altitude. Short runs of left ventricular tachycardia occurred during climb, rest and sleep, with increasing frequency during ascent at higher altitude, with a 14 complex run at 250 bpm during climb near 5100 m (Fig. 1). No right ventricular tachycardia occurred. Atrial tachycardia occurred predominately during sleep. No ST depression consonant with ventricular ischemia (duration > 20 sec) occurred. Amplitude of the P wave in lead V_1 rose progressively during climb, rest, and sleep with increasing altitude, falling on descent.

Blood oxygen saturation fell progressively from 92% at 1800 m to 71% at 5100 m during climb. Blood pressure levels during ascent were not significantly different from those observed at comparable heart rate during treadmill exercise at sea level min.120/60, max. 160/80 mmHg).

DISCUSSION

Duration of the ascent from 4700 to 5100 m was approximately the same, 2_ - 3 hours, as that 10 years before. Average levels of heart rate and incidence of ventricular ectopy during climb were higher in this study than previously (123 vs. 116 bpm, and 56 vs 50 VPC's/hour). During the first two hours of descent from the highest altitude reached heart rate fell little in this study vs the earlier (120 vs 102 bpm), but ventricular ectopic frequency fell strikingly in both to 10 vs 9 VPC's/hour). In contrast to the prior study when ventricular tachycardia was not observed, 9 episodes were documented in this study. The low incidence of right ventricular ectopy was comparable in the two studies, but the degree of P wave amplitude increase was somewhat greater in this study (0.5 – 1.5 vs 0.1 – 0.2 mm).

Although cardiac arrhythmia has not been observed at rest in younger men at altitudes as high as 8848 m, premature ventricular complexes, ventricular bigeminy, and premature atrial complexes have been recorded during or after exercise in healthy men over an age range of 20 – 53 years at altitudes from 4600 to 7620 m.[1] There is experimental support for the postulate that this ectopy relates to increased sympathetic neural activity. Increased sympathetic nerve traffic is demonstrable on direct intraneural recording in young subjects during acute exposure to ambient hypoxia, with a synergistic effect of exercise and rapid rise in noreprinephrine levels.[2] Sympathetic stimulation and catecholamine release may bring about delayed after-depolarizations and triggering of arrhythmia in atria or ventricles.[3] Plasma catecholamine levels for given exercise levels (% Vo_2 max) are higher in older than in young subjects, and exercise induced ventricular arrhythmia is frequent in the former group.[4]

Of note in both studies reported here is the sharp decrement in ventricular ectopy with lesser exertion during descent vs ascent at the same altitude. Greater incidence of ventricular ectopy and development of ventricular tachycardia in the second study at age 75 suggests increased sympathetic stimulation, not only during climb but during sleep as well. As far as the author is aware, there is the first report of ventricular tachycardia during sleep (6 complexes, 170 bpm, at 4700 m) at high altitude. This suggests that the sympathetic response to hypoxia as well as that to exercise may be enhanced in the older subject.

Table 1. Holter Data on Ascent 1800 m to 5100 m, and P wave Amplitude Descent 5100 m to 2700 m. Values for VPC's Represent Average Numbers Over the Period of Designated Activity.

		1800 m to 2700 m	2700 m to 3720 m	3720 m to 4000 m	3720 m to 4700 m	4700 m to 5100 m	5100 m to 4700 m	4700 m to 2700 m
MAX HR, BPM DURING CLIMB		132	138	121	121	123	120	130
Min HR, bpm during sleep ⊗		45	38	48	56			
LV, VPC'S/HR								
	Climb	4	8	7	16	56	10	21
	Rest ⊗	1	8	8	11			
	Sleep ⊗	2	3	4	6			
RV, VPC'S/HR								
	Climb	4	2	2	1	6	2	5
	Rest ⊗	0	4	1	4			
	Sleep ⊗	1	1	3	2			
LV, Ventricular Tachycardia *		4 (105) Sleep		3 (135) Rest	3 (192) Climb	3 (121) Climb	4 (207) Climb	
					6 (70) Sleep	14 (251) Climb	3 (105) Climb	
							4 (108) Climb	
Atrial Tachycardia *			6 (145) Sleep	5 (165) Rest	7 (200) Climb			
				3 (133) Sleep	8 (168) Sleep			
				9 (163) Sleep	6 (158) Sleep			
P Wave Amplitude, mm ⊥		0.7, 0.5, 0.5	1.0, 0.7, 1.0	1.2, 1.0, 1.0	1.5, 1.0, 1.1	1.5	1	1.0 0.7 0.5

* First figure refers 40 number of complexes, send in parentheses to bpm

⊥ Measurements are from lead V_1: during climb at rest, and during sleep respectively

⊗ Data secured at the higher altitude. HR, heart rate. LV, left ventricle. RV, right ventricle. VPC's, ventricular premature complexes

As regards possible implications of these findings, it may be noted that a survey of deaths among mountain bikers in Austria during the period 1985 – 1992 indicated that 30% of 210 deaths were sudden, that over 50% occurred in men over 60 years, and there was an increased risk with physical exertion.[5] In a survey of deaths occurring in British expeditions at altitudes greater than 6500 m during the period 1968 – 1987, 3 of 15 deaths were of uncertain cause.[6] Thus cardiac arrhythmia may account for a significant percentage of fatalities at high altitude, as well as syncopal episodes, and perhaps some of the deaths attributed to "falls". Thus it would seem prudent to appraise the propensity to arrhythmia at sea level with appropriate recommendations of activity in certain potentially susceptible populations such as older persons, and those with known cardiovascular disease who may anticipate ascent to high altitude.

CONCLUSIONS

The incidence of ventricular ectopy, largely of left ventricular origin, increased in association with development of ventricular tachycardia in this older normal subject at age 75 vs 65 years under comparable temporal and logistic conditions during climb at high altitude. The degree of exertion (ascent vs descent) had a modulating effect on ectopy on both occasions. These findings, together with the development of ventricular tachycardia during sleep on the second altitude exposure, suggest a progressively increasing sympathetic response to exercise under hypoxic conditions, and the possibility of increased sympathetic sensitivity to the hypoxic stimulus with advancing age. Also the focus is on sympathetic stimulation of left ventricular arrhythmia rather than pulmonary hypertension in arrhythmogenesis under unacclimatized conditions at high altitude.
(7)

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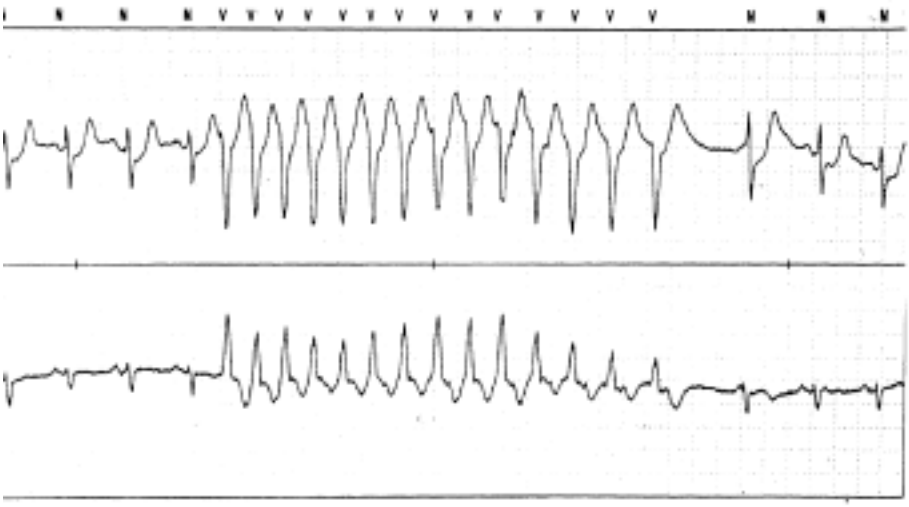
FIGURE 1 Fourteen complex run of left ventricular tachycardia at 250 bpm during climb near 5100 m altitude. ECG lead I above, lead V_1 below.



WILDERNESS MEDICINE WORLD CONGRESS CHATEAU WHISTLER, AUGUST 7TH TO 12TH 1999

Whistler is a fast growing resort in the mountains about two hours drive north of Vancouver in Canada, BC. It is better known as a ski resort but has plenty to offer the summer visitor in the way of hiking, mountain biking, climbing, canoeing etc. It provided the lovely location for the World Congress on Mountain Medicine sponsored by the Wilderness Medical Society. The WMS is a 4000 strong group of Doctors drawn mainly from the US. The WMS regularly has three meetings a year at various pleasant locations around North America and the World Congress takes place every four years. A number of members are also in the ISMM including Peter Hackett who was involved with Ken Zafren in the organisation of the Congress.

The Congress attracted about 500 registrants mostly from Canada and US but 16 other countries were represented. The international flavour was helped by the fact that the UIAA medical commission met at Whistler at the same time and many of them, well know to

ISMM members, were recruited to the faculty of the Congress. Your reporter was one of these.

The WMS also publishes a journal, now called "Wilderness and Environmental Medicine". The editorial board was in celebratory mode because Index Medicus had, since the last WMS meeting, decided to list the journal in its bibliography. This means that the journal should attract a high standard of papers for submission. Many ISMM members have published in this journal already and now that it is IM listed I am sure that members should consider this as a vehicle for their mountain medicine papers.

The Congress ran for four days and topics presented were wide ranging, from attacks by cougars to 101 uses for the safety pin (!) and from the organisation of Mountain Rescue to recent advances in Mountain Medicine. One feature of this congress was the "Small Group session". Two afternoons were devoted to four such sessions with up to 10 groups to choose from in each session. As with any conference when there are parallel sessions you find yourself wanting to go to two or three at the same time. In one of the four sessions I heard Frank Butler discuss, "Ocular disorders in diving and altitude". The latter was covered in an article by Tom Mader in an earlier ISMM Newsletter (Vol. 9 No1 p7). In another session I led a discussion on, "Nutrition at altitude". Most of the groups were more practical, many of them including demonstration of, for instance, rescue techniques, equipment etc. Titles included, Gourmet baking in the backcountry, Water disinfection, Basic swift water rescue (in the water!), Land navigation, and Improvisation for winter survival. There were also more didactic sessions on, for instance, Communication devices, Field Hypnosis, Case studies for trekkers in Nepal, Travel dermatology, High altitude medicine, Case studies in traveller's diarrhoea, Lightning injuries, etc. Unfortunately, none of these could I get to.

During the plenary sessions the timetable was quite relaxed with typically only two talks before coffee and two between coffee and lunch. On the Wednesday we had a morning on altitude. Peter Hackett led off with an admirable talk on; "Recent advances in mountain medicine". Most of the substance of this can be found in his article in the current number of Wilderness & Environmental Medicine, Vol. 10, (2) p97-109; 1999. He advanced the thesis that the symptoms of AMS as well as HACE were due to cerebral edema causing increased intra-cranial pressure. He considered the edema to be probably vasogenic rather than cytotoxic and suggests some increase in permeability of the blood brain barrier. None of this is entirely new but Peter has produced a nice review, which I can recommend to anyone interesting in the mechanisms of AMS. I had been asked to give a personal retrospective talk on "40 years research in the Himalayas" which allowed me to mix some science in a wrapping of reminiscences and mountain slides. I tried to give some idea of the varied types of research I had been involved with from the Silver Hut expedition (1960-61) to Kanchenjunga (1998).

After coffee Bruno Durer gave a gripping talk on "Mountain rescue in the Swiss Alps". The two impressions I was left with were firstly the efficiency of the Swiss rescue service compared with the amateurism of services in most other countries and, secondly Bruno's incredible wealth of experience of mountain rescue. Most of us, after a lifetime of mountaineering have one or two tales to tell of rescue epics. But Bruno, in the season, seems to have one before breakfast, another between morning and afternoon clinics, and another (night rescue) between saying goodnight to his children and getting to bed himself! Finally Franz Burghold told us about training of Mountain Medicine Doctors in Austria. He has been running these very successful courses for about 5 years. The course includes both theory and practical, the latter in the mountains, a week in the summer and another in the winter. Franz had been told, "It's harder to make a mountaineer out of a Doctor than a Doctor out of a mountaineer". However that is just what they try to do on this course. Numbers, of course, have to be limited because of the high staff: student ratio needed for the practical instruction. Students are welcome from countries outside Austria though some proficiency in German is needed. Similar courses are run in France, Germany, and Spain.

There were some 30 poster communications but no discussions sessions unfortunately. Ian Wedmore and colleagues found in a controlled trial that clonidine was effective as a prophylactic for AMS. TV Wu et al. compared Han Chinese and Japanese lowlanders with Tibetan highlanders and found that the latter had much less AMS and also that their pulmonary artery pressure (Echo) was only slightly increased whereas the lowlanders showed marked increase at altitude. Buddha Basnyat, from Kathmandu, reported a random survey of 228 Hindu pilgrims, out of 5000, at Gosainkund (4300m). They had mostly made a rapid ascent from the road head at Dumche (~2600m) and 68% had AMS, 5% had HAPE but 31% had HACE! Women had more AMS than men (odds ratio 4.34).

I had long wanted to attend a WMS meeting and Whistler was certainly a lovely place for my first. I hope it will not be my last.

Jim Milledge, Chorleywood, UK.

SYMPOSIUM ON MOUNTAIN MEDICINE AT ARMY MEDICAL COLLEGE, RAWALPINDI (PAKISTAN) ON 25 MAY, 1999

The armed conflicts have always posed special problems for the medical profession. Modern warfare fought with weapons having devastating power causing casualties on mass scale has taxed the ingenuity and resources of medical personnel all over the world. Even in peace time, the scourges like floods, earthquake, fire, avalanche, trauma, cold injuries and high altitude hazards continue to pose new challenges. It is rational for the medical corps to conduct research in high altitude medicine in addition to its service role.

It is a lamentable but nonetheless incontrovertible fact that most of the serious losses that occurred from high altitude disorders and cold injuries to the troops deployed in mountainous areas should not have occurred. It would be less than candid not to acknowledge this painful truth. The losses occurred due to cold injures because the lessons of the past (WWII) were not learnt. In order to institutionalize this medical problem, the High Altitude Medical Research Cell (HALMARC) was established. The need for such establishment cannot be over emphasized when a significant number of troops are deployed at high altitude in a glaciated terrain.

A Pak-Swiss Expedition climbed on Skyang Kangri in the Northern Area of Pakistan from May to June, 1999. Maj Gen M. Shuaib Qureshi (Retd), Professor of Medicine co-ordinated between the Swiss delegation and High Altitude Medical Research Cell (HALMARC) to organise a one-day symposium on Mountain Medicine in the Ayub Auditorium at Army Medical College, Rawalpindi, Pakistan. With the hectic efforts by Brig (Dr) Muhammad Aslam, Professor of Physiology and Officer In charge of HALMARC, it took a practical shape at a short notice. The symposium was chaired by Professor Ulrich F. Gruber, MD, Prof Emeritus of Surgery, Consultant in Occupational Toxicology Sky Instructor, University of Basel, Switzerland and the Secretarial Assistance was provided by Brig (Dr.) Muhammad Aslam. The introductory remarks were given by Maj Gen (Prof) Najam Khan, Professor of Surgery & Principal, Army Medical College, Rawalpindi, Pakistan.

The programme comprised of as follows:-
Children at Altitude. Dr. Susi Kriemler, Zurich, Switzerland.
Spinal Cord Injuries Rescue and rehabilitation, Dr. Monica Brodmann, Basel, Switzerland.
Climbing High/Diving Deep. Dr. Walter Pteithofer, Goldau, Switzerland.
Treatment of Pain and Surgical. Dr. Urs Wiget, Sion, Switzerland
Avalanche Survival and Treatment of hypothermia. Dr. Bruno Durrer, Lauterbrunnen, Switzerland.

The symposium was attended by 300 participants consisted of undergraduate medical students, Faculty members and the Swiss delegate. The concluding remarks and note of thanks was offered by Maj Gen (Prof) Mahboob Alam Shah, Prof of Pharmacology, Army Medical College, Rawalpindi (Pakistan). In the end, the college souvenirs were presented to the delegates by the Principal as a token of appreciation and remembrance.

Brigadier Muhammad Aslam
High Altitude Medical Research Cell
Army Medical College
Rawalpindi

ODE TO THE PORTERS OF OXYGEN IN THE NEW MILLENNIUM

One cannot stop wondering about the marvelous red blood cells. The only cells in the human organism that have no nucleus, because they don't need to replicate themselves and in order to save energy by not consuming their precious load of oxygen, carried to the metabolically active and oxygen hungry cells of the whole organism.
The biconcave shape permits them to be flat, yet maintain the greatest surface area. Such shape, along with the flexibility of their walls, makes their transit along the capillaries a smooth and friction free ride. Their interior is filled with iron containing hemoglobin molecules that when packed with oxygen gives them the characteristic scarlet red color. Hemoglobin is also an example of perfection in nature. This "awesome" molecule has the S-shaped oxygen dissociation curve, which curiously resembles the left half of the marvelous normal Gaussian distribution used in statistics. This shape is the key to easy loading, holding on to oxygen and easy unloading. They live on average 120 days, with not a single minute of rest, always carrying either oxygen or carbon dioxide. If we consider a 30 minute circulatory time, they traverse a distance of around 3 kilometers per day or 360 kilometers in their whole life. Sometimes and accidentally they "lock" themselves with carbon monoxide, but they are not to blame. When the skin is cut, they form the structure upon which fibrin will deposit and later healing cells will develop. And in relation to intra-vascular thrombosis, it is not they that begin the thrombus, it is the thrombocytes (or platelets) that adhere to endothelial lesions or suffer biochemical alterations and create webs that then catch the circulating red cells. They pile up in order to "plug" the lesion. Along the history of medicine, they have

been vilified. Oceans of human blood have been shed as a means to treat ailments, of which medical practitioners had absolutely no idea of what was going on.
Their most recent accusation of evilness is their role in high altitude increased polycythemia. They are blamed for headaches, physical fatigue, mental fatigue, muscular pain, tinnitus, burning sensation of hand palms, depression, dizziness, sleep disturbances, anorexia, and even shortness of breath; even though they are doing their best to carry the "scarce" oxygen available. Their increase above normal levels, allow for energy efficient high altitude adaptation. May the scientists of the next millennium, show more respect and understanding of these wonderful microscopic porters of oxygen, particularly when they marvelously compensate for lung and heart deficiencies in the hypoxic environment of mountains.

Gustavo R. Zubieta-Calleja Jr., Bolivia

THE EMERGENCY BREATHING DEVICE "AVALUNG™" BY BLACK DIAMOND LTD

International Commission for Mountain Emergency Medicine

A short time ago a new emergency breathing device was presented and launched by the media in the USA and Europe. Headings as follow give hope of an efficient rescue device: "The AvaLung, A Revolutionary Avalanche Safety Tool" (*The Avalanche Review*, USA, Feb. 1999), "Caught by an avalanche, breathe with a plastic bag" (*Medical Tribune*, Feb. 1999), "An air vest that helps avalanche victims breathe" (*The New York Times,* 1st April 1996), "The AvaLung gives you a snowball´s chance in Hell" (*Powder Magazine,* Sept. 1996).

AvaLung™ was invented and patented in 1996 by a physician, Thomas Crowley. Subsequently the device has been constructed by Black Diamond Equipment Ltd in Salt Lake City, UT, USA. The plastic device is built into a waistcoat, worn continuously as an outer garment by the backcountry traveler. A flexible plastic mouthpiece is located near the garment's collar. If an avalanche is triggered the user must move his mouth to the mouthpiece and breathe through it. A membrane (Avalung™ System), built into the front of the jacket, provides for inhalation of air from the surrounding snow and exhalation to the rear of the vest. Thus, increasing levels of CO_2 in the inspired air can be avoided.

The emergency breathing device was tested at Mt. Hood, at an altitude of 2225 metres, in 1998 under the medical supervision of MI Grissom and CK Radwin. Three volunteers were firstly partially buried (head-out) in the snow, then they were fully buried beneath well-packed snow (0,3-1 m below the surface). Pulse and respiration rates, oxygen saturation and inhaled and end-tidal CO_2-levels were measured continuously during burial. Intercom systems allowed continuous communication between the subjects and the test team during burial, a T-tube led to the surface for insufflation of oxygen at the end of the test.

All subjects showed increased pulse and respiration rates during burial. The first subject's oxygen saturation level remained above 93%, end-tidal CO_2-level rose maximally to 6%. The test continued for 63 minutes, the maximum planned duration. As with the first, the second subject's oxygen saturation remained above 92% and CO_2-level rose maximally to 6%, but he became anxious and requested to be dug out after 10 minutes of burial. During the full burial test of the third subject, oxygen saturation slowly declined to 81% and CO_2-concentration rose up to 8%. The subject was scared so he was dug free after 45 minutes. The subjects neither showed unconsciousness nor signs of dyspnea, but all felt compressed by the snow masses and reported that the weight of snow impaired thoracic movement at the beginning of burial.

In March 1999, the device was presented at Simplon Pass (CH) to members of the International Commission for Alpine Emergency Medicine.

One may criticize that with the described experiments the supposed extension of survival time after complete burial cannot be quantified. The small number of tests does not allow a statistical analysis. Moreover, two out of three tests were stopped before the planned duration was reached due to panic attacks, in one case even to increasing hypoxia. The only conclusion from the results may be that in certain cases survival time can be prolonged up to one hour. Nevertheless, the fact, that it was possible to breathe from snow without any hypoxia for this period is a surprising result. It shows the high air-diffusion through the snow, even when density is very high (600 kg/m^3). The separation of inhaled from exhaled air presents an ingenious trick of avoiding asphyxia.

There remains a basic criticism. AvaLung™ can prolong survival time of a totally buried person from 15 up to

60 minutes. Time is gained for rescue. However, to save life not only prolongation of survival time but also localization and extrication from snow masses within this time period are necessary. Rescue in time depends on particular circumstances: extension of the avalanche, depth of burial, position, equipment and instruction of non-buried companions. Rescue within 1 hour can normally carried out only by companions, not by professional help. Moreover, the jacket does not protect from injuries during the descent of the avalanche. It is doubtful whether the user will be able to correctly position the mouthpiece. The influence of hypothermia is unknown, because core temperature during tests was not registered. Reports of real accidents for a realistic evaluation of the device are not yet available.

At this time, we cannot quantify the influence of AvaLung™ on survival rate. The most important disadvantage seems to be the acceptance of a complete burial with all its risks, despite the fact that rescue in time cannot be guaranteed. Reduction of mortality with AvaLung™ can be obtained only with efficient help by uninjured companions, using avalanche beacons, probes and shovels. It should be appreciated that the producer does not conceal these negative aspects, but points to these limits of the rescue device.

It is uncertain whether mortality rate can be reduced by the AvaLung™ device. The main goal of any avalanche rescue technique should not be the prolongation of survival time, but the avoidance of burial. Buoyant systems have the advantage over AvaLung™ of avoiding a complete burial and of reducing risks of mechanical trauma. Moreover, rescue does not depend on the success or failure of localization and extrication of buried persons.

AvaLung™ could theoretically prolong survival time of complete burial in special cases, but it is unknown whether survival probability will be increased significantly. The ICAR-MEDCOM does not have enough evidence to allow a final statement at this time. AvaLung™ has the disadvantage that complete burial is being deliberately accepted. Outcome for a buried person depends on rescue, within the survival time, carried out by uninjured companions with avalanche beacons, probes and shovels. Users are likely to run the risk of minimizing the dangers of complete burial. Further developments of rescue devices which aim to avoid total burial, with certainty, should be preferred.

On April 30th 1999 this statement was discussed and approved in Fieberbrunn (A) by the International Commission for Alpine Emergency Medicine (ICAR-MEDCOM).

Hermann Brugger MD, Urs Wiget MD (president ICAR MEDCOM), Bruno Durrer MD (president UIAA-MEDCOM), Dave Syme.

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A CASE OF SERIOUS FROSTBITE IN THE MONT-BLANC MASSIF

Although we receive, in Chamonix hospital, nearly 80 cases of frostbite per year, rare are the cases serious enough to lead to amputations of the fingers or toes (8%). We report here the most serious case that we have ever had to treat. This case ended in total amputation of both the hands and feet, despite the early commencement of the reference treatment.

OBSERVATION
On Monday January 25, 1999, two British mountaineers set off to climb the north face of 'Les Droites', a difficult 1000 metre ice route, situated in the Mont-Blanc massif. On the Tuesday evening, they reached the summit ridge, at 4000m, in a storm and, too exhausted to go down, they decided to bivouac. They were equipped with sleeping bags, but did not have a tent. The snowstorm lasted until Friday, the day on which the helicopter was able to locate them but not to rescue them because of strong winds at altitude. The two mountaineers were still alive; one of them even stood up to confirm to the rescuers that they needed help. With the return of anticyclonic conditions, the temperature in the valley (1000m) dropped to -15°C. On Saturday, the wind picked up, preventing helicopter

flights. One of the two mountaineers died in the afternoon. Finally, on the morning of Sunday 31, the survivor, upright, haggard, in socks and with bare hands, welcomed the rescuer, who was descended by winch close to him, by means of some risky manoeuvres.

On his arrival at the hospital, the survivor was conscious, slightly hypothermic (33°C) and clinically dehydrated (pulse 110, BP 80/45). Both his hands were livid and frozen up to the level of the wrist. The right foot was in the same state below the ankle, with cold and cardboard-like tissues at the level of the kneecap. The left leg was frozen until the middle of the calf. A frostbite of the pavilion of the ear was also noted.

Initial resuscitation

The rise of core temperature and the re-establishment of an effective volemia were achieved by the perfusion of 3500 ml of crystalloid solutions heated to 40°C (isotonic saline and bicarbonate 1.4%). An air blanket was used at the same time. Outstanding biological results were: pH 7.29, creatinine 250 µmol/l, haemoglobin 17 g/100 ml, WBC 31000, CPK 6120.

In parallel, the early treatment of the frostbite consisted of the injection of 250 mg of aspirin and the perfusion of 400 mg of buflomedil followed by the rewarming of the frozen extremities in a 38°C bath for 2 hours. Once re-heated, the hands and the feet, cyanic, became mobile again, but insensitive, and swelling appeared rapidly in a few hours, except in the left leg where the soft parts remained tense, leading to an aponevrotomy 6 hours after admission.

Following days

The reanimation prevented the consequences of the acute tubular necrosis. Initially black, then red, the diuresis was maintained to 3 l per day thanks to the supply of 4 liters of crystalloids by 24 hours, associated with furosemide. For the frostbite, we chose the theoretically most effective protocol, associating aspirin, prostacyclin and heparin.

The following day, February 1, the hands were swollen, cyanosed, with small darkish blisters. Insensitivity was complete, and the patient was encouraged to move his fingers, in particular during the twice-daily baths. The feet presented an identical aspect. From the first day, the patient remained feverish, at around 38°. The CPK reached a peak of 43000, without deterioration of the renal function.

On February 3, the temperature reached 39°C. We introduced antibiotics: penicillin 10M and metronidazole 1.5g. The patient underwent a surgical excision of the blisters from where we extracted some darkish and 'nauseating' serous fluid. Regarding the maintenance of the renal function, and the pain and the swelling above the frostbite, we decided to introduce ketoprofen (100 mg/d).

On February 4 the patient was sufficiently stable to withstand a long investigation (6 hours), so we sent him to the service of nuclear medicine for osseous scintiscanning. This identified an absence of fixing at the osseous time at the midway level of the left leg, below the right ankle, and below the left wrist. Only the right carpus fixed a little.

On February 5, his clinical condition was very worrying (wet gangrene) and we decided to cut the left leg. We performed a guillotine amputation, carried out at the mid-level of the tibia. The surgical wound was left open. The patient remained voluntary and participating, but increasingly weak, and anxious.

On February 7 in the morning, we noted 3 episodes of shivers with a fall of blood pressure. The first blood cultures returned positive for *Escherichia coli*. One of the shivers began with the introduction of the prostacyclin.

On February 8, the patient still shivered at the beginning of the perfusion of prostacyclin. Taking into account the antibiogram, we introduced a combination of cefotaxime, gentamicin, and metronidazole. The chest X-ray showed bilateral pulmonary oedema, and the hypoxia worsened (PAO_2 39 mmHg, PCO_2 33mmHg).

Amputation

On February 9, the radiographic appearance and infection worsened. It was finally in the evening, in full period of hyperkinetic endotoxic shock that the patient went to the operating room for amputation of the 4 extremities. The right foot was cut in the healthy zone, 5 cm above the ankle and the left hand 1 cm above the wrist. After exploration, the right hand was also cut in the same way, since none of the tissues covering the carpus appeared viable. All the sections were of the guillotine type, with covering of the bones by the muscles, but the skin remained open.

After the surgery, the patient remained under anaesthesia with mechanical ventilation and PEEP. His general condition improved quickly, the PAO_2 reached 75 mmHg the following day morning. The oedemas disappeared in 48 hours (diuresis 4500/ d). The patient could be extubated on February 13.

Later evolution

The stumps were revised and 3 of them closed within the following week. The left leg could not be closed, but the muscular cover on the osseous section was satisfactory. The patient was transferred to Scotland on February 23. By May 1, the patient already had prostheses for both legs and the left hand. He was able to walk with crutches.

DISCUSSION

This accident takes us back 42 years to the tragedy of Vincendon and Henry, when two mountaineers died 'live' (they were visible with binocular from Chamonix) because there was no means of evacuation. In 1999, the Mont-Blanc massif remains a high mountain area; in spite of vastly improved rescue facilities, storms still prevent the helicopters from flying and heavy snow fall render the slopes inaccessible to the rescue parties. These two mountaineers decided to climb, in winter, on a particularly difficult north face, in spite of an unfavourable forecast. Experienced Scottish climbers, they were used to bad weather, but at a lower altitude. Some of their other decisions also don't seem very rational: bivouacking in a place which quickly became inaccessible to rescue helicopters in high winds; taking off their boots to get into their sleeping bags which meant that their boots were rapidly lost in the storm as were the survivor's gloves which were taken off to bring a little heat with naked hands to the chest of his dying companion ... And in this easily accessible massif, over-covered by the media and over-secured, one of them died, less than 5 minutes' flight from downtown Chamonix.

On arrival, the patient was conscious in spite of a temperature of 33°C; we can call this subacute hypothermia. He had resisted 4 days and 5 nights of icy hurricane, which shows the excellent physical condition he was in at the beginning. He was very dehydrated: due to a lack of water supply (a stove doesn't heat in a satisfactory way in high wind); the dryness of the air; and the hypothermia which, at the time of its onset, by blood redistribution towards the core, causes a relative hypervolemia followed by hyperdiuresis.

The aim of the initial treatment was to re-establish an effective blood volume at the renal and microcirculatory levels. The use of heated perfusions, moreover, helped to heat the patient more quickly, along with an air blanket, which is the usual treatment for this type of moderate hypothermia.

The physiopathology of frostbite describes a phase of cooling during which the adrenergic vasoconstriction causes a closing of the small precapillar arteries and the opening of shunts upstream. Clinically, the affected extremities are livid. In addition, the blood flow is decreased in the largest vessels by the increase in viscosity related to the cold. The hemoconcentration and dehydration are auxiliary factors. This period is followed of a phase of freezing, initially extracellular with an increase of osmolarity, then intracellular with a phenomenon of supercooling in the cytoplasm until tissue temperature becomes as cold as -15°C. This mechanism explains why some frozen tissues heal totally when an effective treatment is correctly and promptly undertaken. However, when the exposure time to freezing is prolonged and for temperatures even colder, the crystals appear in the cytoplasm, destroying the remaining intact ultrastructures.

Because of the depth of the freezing in this case, the reheating of the hands and the feet took 2 hours, which is unusual since a bath of 30 min generally makes it possible to recover flexible tissues. At the end of this bath, the frozen extremities appeared red, a red which turned blue in a few hours. Indeed, during the reheating, the arteriolar vasoconstriction usually gives way to a reactional hyperhemia related to the appearance of vasoactive substances locally during the ischaemic phase. This hyperhemia facilitates the passage of liquids towards interstitium, which causes an increase in the blood viscosity followed by a slowdown of the microcirculatory flow. The desquamation of the endothelial cells and the deterioration of the basal membrane generate an activation and an adhesion of the leukocytes, mast cells and platelets. This progressive vascular thrombosis leads in a few hours to a complete shutdown of the microcirculation [1].

All the therapeutic protocols are based on these concepts. Aspirin and buflomedil were given before the reheating in order to be present in the tissues at the return of circulation to prevent these phenomena. Thereafter, the theoretically most effective molecule was employed: Iloprost which is the stable analogue of prostacyclin, or PGI2. It inhibits, by antagonising thromboxane A2, all the consequences of the platelet activation, especially the vascular wall aggregating and adhesive mechanisms. It is, by its direct relaxing effect on the smooth muscular cell, one of the most powerful vasodilators known today. It inhibits the leukocyte activation and the freeing of cytotoxic factors and is cytoprotective [2].

Haemodilution was favoured to increase the distal flow to the maximum. When the renal function was stabilized, we added ketoprofen to reinforce the cyclo-oxygenase inhibition and, auxiliary treatment, polyvitamins were given for their anti free-radical effects. Only, the thrombolytic drugs were not employed. Their effect would be best after of a slow and spontaneous remarming of frostbite [3], which was not the case here.

All the paradox of frostbite is seen in this case: although blood flow was restored in the hands and the feet, still macroscopically intact at the time of admission, and although physiopathology tells us that the cells can remain viable for very low temperatures and although the reference treatment was employed, these extremities evolved to necrosis [4]. The duration of frostbite was certainly a determining factor. Conversely, the skin above the right kneecap, which was also cardboard-like and frozen, but exposed to less cold temperatures, being protected by clothing, and

with a non-terminal vascularization, totally recovered in a few days.

From the first hours, the clinical aspect of the left leg was worrying, with tense muscles, which justified the sub-cutaneous aponevrotomies, the incision (5 cm) being in healthy zone. This technique was promoted by Mills with good results [4], but it is rarely performed because of the risks of traumatizing hypoxic tissues.

On day 4, the vascular necrosis being well established, the osseous scintiscanning with technetium 99m showed the extent of the lesions. This examination is done in three stages, with an immediate or vascular phase, an early or tissue phase and a late phase (after 3 hours) or osseous. According to a study carried out here on 95 cases, the specificity of this examination is 0.99 and its positive predictive value (of amputation) is of 0.84 [5]. It was thus legitimate, in view of the pictures, to cut the clinically worrying left leg without doing an arteriography. Only the right carpus left some hope.

In the case of absence of fixing of the tracer at osseous time, some authors propose an intervention of rescue: cutting down the soft parts, leaving only the tissues with a less demanding metabolism, such the tendons, the nerves and the bones and covering them with a free or pediculated flap [6]. The idea was evoked, but quickly drawn aside, given the septic context and the size of the flaps which would then have had to be used.

The right foot and left hand were quickly mummified, with not very inflammatory tissues above. The soft parts of the right hand remained flexible but of doubtful viability. On February 7, 8 and 9, with the onset of the perfusion of iloprost, the patient shivered, which evokes the symptoms appearing in arteritic patients after reperfusion surgery.

In spite of the aggravation of his respiratory function, the patient remained lucid, although exhausted, until the 9 February in the afternoon, and could take part in the decision-making. Indeed, the moment of the amputation was psychologically difficult to determine. The norm is to cut as late as possible, when the demarcation line is well marked, which allows for minimum cutting in already cicatricial tissues [4][7]. This amputation is usually carried out in a guillotine manner and revised later on.

Finally, the radical amputation was imperative in the context of hyperkinetic septic shock. Perhaps it would have been better to cut the right foot and the left hand earlier as this could have decreased the quantity of necrotic tissues and their general inflammatory consequences. Moreover Seim speaks of an delayed immunological phase [8].

Once the necrotic zones were withdrawn, the treatment of this septicaemia with sensitive E-Coli was easy and fast. Four days of controlled ventilation stamped out the ARDS. The renal function was preserved and the inflammatory and oedematous syndrome was quickly amended. It is true that in mountaineering frostbite tends to affect young subjects in good health, with a strong potential of recovery.

CONCLUSION

This case is exceptional in more than one way: firstly because of the incredible survival of the victim under such harsh conditions; secondly by the extent and the depth of frostbite; and finally in view of the early commencement of the reference treatment, its failure and the difficult physical consequences. Serious frostbite is, and remains, a frightening condition

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JANUARY CASE DISCUSSION

I am passing my military service as a doctor in Andermatt (1600m), where young recruits are instructed to become mountain specialists.

On Wednesday, 3 March 1999, three weeks after begin of their training, the following incident happened. The troop, for the purpose of mountain instruction, were staying that day and the day before

in the Nätschen-Gütsch area, at an altitude of 2300m. That night they spent in a snow-bivouac. In the afternoon, at about 14:00, the condition of a corporal increasingly deteriorated. He became somnolent within minutes and had several apneas with a maximal duration of 10-20 seconds. The corporal was a very fit man, but since two days before this incident he had been suffering from a common cold. The rescue was immediately organised. First with the help of his comrades and then by snow-cat, he was transported down to the valley. When I saw him at 15:30 he had a GCS of 8. Heart rate, blood pressure were normal. Auscultation of both lungs was without abnormalities. The pupils were dilated, but still reacting to light.

I informed the Swiss Air Rescue Guard; within several minutes they arrived by helicopter. The rescue guard doctor intubated the patient and transported him to the University Hospital of Zurich suspecting a subarachnoid hemorrhage. In Zurich the condition of the patient improved. Extubation at 18:00, CT scan of the head was normal, no signs of infection (including lumbar puncture). Normal blood sugar, no drugs could be detected. The next day the patient was fully conscious but he had a complete amnesia of this incident. He could leave the intensive care unit and returned then to Andermatt; he was very tired but otherwise healthy. Four days later I examined the patient again. He still suffered from a little headache and felt pain while breathing deeply. I could not find any pathologies in the clinical status.

Can such a condition have occurred due to exhaustion, in this case triggered by the common cold? Alternatively, could it have been a sort of high altitude cerebral edema (HACE), although the corporal was well acclimatized and it happened in a lower altitude zone (2300m). The patient fears that such an incident may occur again, as, in his civil life, he wants to become a mountain guide, it is understandable that he would like to know more about the causes of this occurrence.

May further examinations lead to a diagnosis? Is there any form of prevention necessary?

Bernard Marsigny, France

What was his central body temperature? We regularly receive, in the emergency ward, injured patients with a coma and mydriasis, whom body temperature is under 30°C and who recover fully within a couple of hours. Exhaustion greatly increases the risk of being hypothermic.

Gustavo Zubieta Jr, Bolivia

Interesting case. Unfortunately, no arterial blood gases, pulse oximetry or ventilation measurements at high altitude were made. This may be similar to a case of hypoventilation on arrival to high altitude we previously reported (ISMM Newsletter, Vol 8 #1, 1997/98 [resume] & http://www.geocities.com/CapeCanaveral/6280 [complete]). A Japanese subject that while in transit arrived at the La Paz airport (4100m) and within hours felt very sleepy, although responsive to stimulus with a GCS of 11. He had a very low alveolar ventilation 2384 cc/min BTPS, with a respiratory frequency of 8 per minute and a PaO_2 of 41 mmHg. When given oxygen during 24 hours he felt much better and his blood gases improved. These patients also hyperventilate on hyperoxic tests, which makes treatment much easier. A possible explanation is a temporary alteration of the respiratory center, possibly caused by bacterial or viral disease (in this case the common cold), thereby producing a limited respiratory response to hypoxia.

These sudden hypoventilators on acute exposure to high altitude may confuse the physician, but there seems to be no reason to panic.

Intubation is definitely not needed. Oxygen administration and monitoring of pulse oximetry along with clinical observation, during 24 hours are necessary. There would probably be no risk in going back to altitude.

Jim Milledge, UK

Again this is an extra-ordinary case. I can only assume, in view of the rapid and complete recovery, that it must be a case of HACE. Possibly the virus infection, a common cold, was enough to cause a low saturation. This triggered the somnolence and periodic breathing thus lowering the O_2 saturation even more, causing HACE. However, at only 2300 m this sequence of events is most unusual.

Advice for the future is difficult. There is an impression that HACE is less a matter of susceptibility than is HAPE. But never-the-less he must be considered as being at higher risk of HACE than the average and future trips on to altitude he and his companions must be on the look out for trouble.

John Severinghaus, US

There is little one can do here except speculate. When the first physician saw him, was he cyanotic? Was a pulse oximeter used? One wonders if he had a severe cyclic apnea, preceded by hyperventilation such that a long apnea would occur before CO_2 rose enough to start him breathing. If so, it implies a low HVR, and he should have that tested before deciding to work at altitude. Also, if so, acetazolamide would be good to prevent a recurrence.

If a careful CT scan was really examined, and found no evidence of white matter edema, it will be hard to call it HACE. Did he have retinal petechiae? I can't relate this to a cold virus. So I guess he had post-anoxic unconsciousness. This is similar to the long period of unconsciousness we used to see after using hypothermic cardiac arrest for cardiac surgery in the 1950's. Patients whose circulation was stopped for about 15 min at

30°C often took 8-16 hours to really wake up after all anesthesia was thought to be out of the system.
We assumed it to be a post anoxic stunned brain (although we hadn't used stunned then).

Ken Zafren, US
This is a mysterious case. It doesn't really sound like HACE, except for the fact that it resolved after descent. Carbon monoxide poisoning is a possibility although the onset sounds quite rapid for this and we don't know from the history about exposure of others. Could this have been a seizure? An MRI of the brain might be helpful, even at this late date. The workup which included CT scan and lumbar puncture would exclude most cases of subarachnoid hemorrhage. The role of the upper respiratory infection in this case remains elusive.

Peter Hackett, US
I think this man's condition had nothing to do with altitude illness, and probably was not related to altitude at all. Assuming he had been at 1600 m for the 3 weeks of training before ascending to 2300 m, he was no doubt well acclimatized. In addition, 2300 m is barely high altitude. Since he had been staying in a snow shelter, could carbon monoxide poisoning have been a possibility? The clinical description is consistent with CO. Other possibilities include a drug not revealed by the testing undertaken, asphyxia (did one of his comrades try to suffocate him?), and atypical seizure disorder. Was he hypoxic at any time? Apparently hypoglycemia and other metabolic disturbances were ruled out, as was SAH, and presumably cerebral edema. Was an MRI done? Was MRA done to exclude any vascular abnormalities? His episode should not be considered due to altitude and warrants a thorough search for other etiologies. I would advise him to go to a higher altitude such as the Capanna Margharita or the Vallot Hut and his response to high altitude could be studied by one of our European colleagues.

Oswald Oelz, Switzerland
A Swiss soldier and mountain guide at an altitude of 2300 m. I have no idea what this guys problem was. However, I would definitely exclude HACE. The only thing that comes to my mind is some sort of seizure and I would recommend the appropriate examinations.

Robert Schoene, US
Have to think that this fellow had HACE. Unusual altitude to be sure. In HAPE there are good data (ours and the pediatric folks from Colorado) that a viral infection may predispose folks to getting sick and a large number have evidence of LTE4 in their urine as a marker of some inflammatory response that may make the endothelium more vulnerable to the subsequent pressures. Don't know about the brain but is possible. Was his serum sodium checked?
I have a couple of cases who drank a large amount of water (as read from the usual AMS brochures) and got HACE at very moderate altitudes (~2500m or so). I think that there may be a relationship between over-drinking of pure H_2O and HACE. My patient had a Na^+ of 121 and was comatose.

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