SESSION I

Chairman: PROFESSOR R. HOFFENBURG

Introductory address: Lessons to be learned from high altitude

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Summary

A historical account of the important landmarks in man's experience with the high altitude environment is followed by comments on the important stages in the understanding of its physiological effects. The work of The Mount Logan High Altitude Physiology Study on acute mountain sickness is reviewed from its inception in 1967 until the present.

For a featherless biped without wings, man (or woman of course) hasn't done badly at getting off the ground and into the air. Brain rather than brawn has sent a few hundred persons higher into space than any except the most avid science fiction buffs thought possible, but these celestial explorers are cocooned in the familiar earth's atmosphere, not exposed to the thin cold void of space. Some day, technology may find a way to move us toward infinity without encumbrances, but not for a time. Today, those who go a few miles above the earth must either take their air with them or risk a variety of unpleasant to fatal consequences. The same is true of unfortunates whose oxygen delivery systems fail – either from disease or injury, or failure of mechanical safeguards.

It is my privilege to discuss with you a few aspects of man's ventures to altitude initiating other presentations which will show how far we have come since Icarus, Cavallo, Acosta, Bert, Haldane, Barcroft and Whymper, Odell, and Mallory. First I wish to review some important landmarks in our exploration of the atmosphere in which we live much like fish in water. Then I will touch on our awakening awareness of what too little oxygen will do to unprotected man, leaving to others a detailed description of altitude illness as we know it today. I will review some of the work which my

colleagues and I have been doing high on Mount Logan in the Canadian Yukon for the past 10 years, and finally I shall try to relate what happens to healthy persons at altitude to the signs and symptoms of lack of oxygen due to disease or injury at sea level.

Only in this century have we been able to move almost at will wherever we wish in the thin envelope of air about the earth, and beyond where molecules are miles apart. Before now wax wings have melted in the heat of the sun, evacuated copper spheres have collapsed, 'airships rowed by oarsmen' have refused to lift off, and balloons moved at the whim of many capricious influences. Until this century we went above sea level on foot or on horse-back, very rarely by elephant or camel or yak. One wonders but cannot even approximate, just what percentage of earth's population in any given century in the past has tried to go high into the sky. I suspect that the incidence of astrophilia may have been constant throughout our complicated evolution.

Noah, the first and perhaps only mountaineer to reach his goal by boat, under circumstances which are not expected to recur, took 40 days to get there from sea level. He and his passengers did have motion sickness and some are alleged to have had bad hangovers, but none suffered from altitude illness (often confused with the fruits of over-indulgence), even though Arrarat is 5500 m high. Moses of course climbed on his own two feet but to a lower and innocuous altitude: Mount Sinai is only 2200 m high, an altitude noticeable perhaps to jet-propelled vacationers but rarely if ever to climbers. There were others. We know that the Altiplano of South America, an area of some 170 000 square kilometers, most of which is above 3500 m, has been densely populated at various periods in

the last 10 000 years and perhaps much longer. We know that the high Tibetan plateau has supported tribes of nomads for many millenia. Their descendants the Sherpas appear to have inherited a mutant haemoglobin, or at least a variant oxygen transport system which enables them to work harder and longer at altitude than can even well acclimatized sea level man. Whether there has evolved in the Andes a special 'altitude man' is still disputed, but with reasonable certainty we know that the descendants of altitude residents are better adapted than those who have been there for only a few years or decades. Marco Polo commented very briefly on this during his crossing of what were then called 'the headache mountains'. José Acosta, the most famous raconteur of the horrors of altitude sickness, described what happened when he crossed the high steps of Periacaca (about 3500 m), calling them 'puña' – the term for high barren desert. Today, Peruvians speak of 'soroche' (the word for lead or antimony) because centuries back the illness was blamed on emanations from these ores (and from mosses, primroses, and certain herbs as well).

Acosta's book was published in 1590, some 40 years after his journeys ended, and one can never be quite sure how much may have been added or forgotten, but his account remains the best one of altitude illness to appear until several centuries later. He noted that 'the air is there so subtle and delicate, as it is not proportionable with the breathing of man . . . and I believe it is the cause that doth so much alter the stomacke and trouble all the disposition'. Remember, that in Acosta's day the precepts of Aristotle dominated science. That great philosopher scientist had taught that a vacuum could not exist even though he recognized that ' . . . in its own place every body has weight, except fire, even air', and thus it could have been heretical to suggest that the atmosphere grew progressively thinner at greater heights.

Indeed, some advanced thinkers paid by death or torture for questioning the beliefs of the church so many of which sprang from Aristotle. Soon after Acosta, though, the intellectual and scientific atmosphere began to change, slowly, tentatively, then with a rush. Appreciation that air had weight led to proof that it could be compressed, confirming much of what Democritus had written well before Aristotle's time, about the smallest particles, which he named 'atoms'.

A major landmark in our studies of altitude was the evolution of the barometer, probably conceived by Torricelli though first demonstrated by Berti in 1643 or thereabouts. Even before then (c. 1630) Baliani wrote 'the higher we go into the air, the less weight it has' a remarkable perception proved

correct in 1648 when Perier, with several reliable witnesses, climbed the Puy du Dôme (1100 m) and proved that the mercury in his barometer stood lower on top of that hill than at the bottom. Thus, the fact that atmospheric pressure decreased as altitude increased was established, and a major step taken toward understanding hypo-barism. At that time, the atmosphere was considered to be an 'aether, a spiritus mundi or subtle substance' which could pass through pores in solid substances. Not for some time would it be recognized as a mixture of many gases, a word coined by Van Helmont (from the German 'geist' or spirit) to replace the earlier term 'archaeus' which Paracelsus used to describe the hydrogen he generated in 1530. Robert Boyle planned a long and logical series of experiments which would probably have led him to the discovery of oxygen a century before Lavoisier and Priestley, but he was not able to complete them before his death. His gas laws, of course, laid the foundation for most of what was still to come. Mayow and Borch were busily exploring one of the substances which made up air which Mayow called 'nitro-aerial particles necessary for the support of life and of combustion'. Sealing a mouse or a candle under a bell jar, both these pioneers showed that either mouse or burning candle 'consumed' about one-fourteenth of the air before succumbing. Mayow wrote: ' . . . it is quite certain that animals in breathing draw from the air certain vital spirits . . . ' and 'that nitro-aerial spirit is by means of respiration transmitted into the mass of the blood and the fermentation and the heating of blood are produced by it'. Not for a long time would a better description of metabolism be developed.

Around 1730, George Stahl, court physician in Berlin, proposed the ingenious theory that a substance he called 'phlogiston' had the property of levity (the reverse of gravity) which explained why substances became heavier when they were burned and lost their phlogiston. Surprisingly the phlogiston theory pre-empted attention for half a century before Lavoisier laid it to rest by the studies which he did at the same time as Priestley. Such was the impact of their demonstrations of oxygen that earlier work – a century earlier in fact – has been almost forgotten. As the nineteenth century began, the major components of air were identified and the principles of oxidation were understood.

Meanwhile, as the atmosphere was being dissected, travellers were passing the edges of the all-encircling seas and beginning to go higher into mountains which until the Renaissance were off limits for one reason or another, to all but a very few. Perhaps it

was fear of dragons, catalogued meticulously by Scheuchzer in 1712 (a century after America was settled), which kept most people in the valleys. Or perhaps cold, avalanches, crevasses, or simple lack of curiosity was the barrier. There were superstitions such as the fear of the spirit of Pontius Pilate whose presence in the lake on Mount Pilatus caused such natural disasters that for a century persons who disturbed it could be executed! Although Conrad Gesner took up mountain climbing simply for the joy it brought him, and wrote beautifully about the spiritual embraces of mountains, it was not until the end of the eighteenth century that many people ventured on to the peaks and not until 1850 did the golden age of mountaineering for sport begin. By then, enough was known about the atmosphere and about human physiology to open the way to understanding how high altitude can affect us.

Not far behind exploration of the environment about us came equally wonderful understanding of how the human body functions. Galen had an approximate understanding of at least part of the circulatory system 150 years before Christ, for he knew that the heart pumped blood through vessels, but believed that the lungs served only to cool a portion of the blood, and that blood passed from the right to the left side of the heart through tiny pores. Galen's teaching was established doctrine until Servetus described the pulmonary circulation, paying for this unorthodoxy at the stake in 1553, but confirming observations made about 1250 by a mysterious Persian Ibn-an-Nafis, and paving the way for Colombo's great book *de re Anatomica* in 1559 and for the giant step taken by Harvey in 1628.

The cellular components of blood were recognized immediately by Leeuwenhoeck when he developed his microscopes. Lower reported that blood changed from dark to bright red when exposed to oxygen and the functions of haemoglobin were further analysed by van Liebig (1850) and Hoppe-Seyler (1870) who was a friend of Paul Bert whom we shall soon meet again. Once the circulation of the blood had been clearly understood, the functions of its components could more rationally be grasped, and concurrent developments in chemistry and analytical techniques led to a rush of discovery which is still accelerating.

All of this is fascinating evidence that priority in science is a will-o'-the-wisp, that knowledge is rarely if ever absolute, and that frontiers exist to be breached. Most advances are made on the shoulders of those who have gone before and often – perhaps usually – those who get the credit are not the lonely, inspired pioneers who first saw the light, but those

who came later, recognized and put together the bits and pieces or better publicized or safeguarded their contribution. Lavoisier, for example, carefully instructed the secretary of the French Academy of Science to keep under lock and key the records of his (Lavoisier's) work until it was completed and ready to publish, lest others claim the credit. Priestley was far more open.

Paul Bert was not an opportunist, but a great investigator of prodigious energy who came on stage at an ideal moment to bring together a vast collection of travellers' tales, myths and legends, serious experiments on mountains and in laboratories and decompression chambers, weaving these into a fabric of knowledge which was unappreciated until long after his death. He was a remarkably diverse man: plastic surgeon of note, politician, diplomat, teacher, and imaginative experimenter. Thanks to Dr and Mrs Hitchcock who translated Bert's great book *La Pression Barométrique* in 1942, Paul Bert can be identified as the father of altitude research, whose tracks are found in much of what we know today. He distinguished well between what was known for certain and what was speculative, and anticipated much of what has become an accelerating explosion of information, as others will show us at this meeting.

I must reluctantly leave the charms of history, omitting the great impact of the scientist-mountaineer De Saussure, the physician Mosso, explorers like Whymper, the Schlagintweit brothers, Conway, Zuntz and Loewy in their mountain laboratory, and scores of others.

A great share of what we know about human response to lack of oxygen has come from mountain studies. True, Barcroft worked in the 'Glass House' in Cambridge living for days in an atmosphere equivalent to 4500 m; Haldane and Douglas went above 7000 m in a decompression chamber, and Paul Bert's colleagues made observations in chambers and balloons. But response to altitude (and *pari passu* to other causes of hypoxia) was best studied on mountain summits. Mosso inspired the Regina Margherita Hut on Monte Rosa, Yandell Henderson brought Haldane and Douglas to Pike's Peak, Hurtado and Monge initiated their studies high in Andean mining communities, Pugh and his brilliant cast lived for months in the Silver Hut high in the Himalayas, and Singh, Roy and others have produced an almost indigestible mass of data from studies of soldiers on the high Himalayan frontier with China.

With the conviction that hypoxia could best be studied in healthy persons exposed to lack of oxygen in as near normal an environment as possible – and incidentally in a beautiful and inspiring setting –

we have followed the lead of many of our predecessors by working in a high mountain laboratory on Mount Logan. I would like to give you a brief summary of this programme and some of the highlights of our 10 years of work there.

The Mount Logan High Altitude Physiology Study (HAPS) began at a small meeting of American and Canadian scientists in the autumn of 1966, when the Arctic Institute (a joint Canadian-American organization) wished to expand its years of broad environmental studies in the St Elias range of northern Canada, to include human physiology. Mount Logan appeared a suitable site for establishing and supporting a well equipped laboratory on the large summit plateau which ranges in altitude from 4200 to 6000 m. A site was selected at 5360 m and the first building erected and occupied in 1967. Except for 1972 and 1978 we have worked there each summer, supported at first by private foundations, but mainly by the National Institute of Health (U.S.) and by the Canadian Forces. It has been from the start a bi-national effort with scientists from many universities participating, and with year-round work now evolving to supplement the 2 concentrated summer months. Without going into much detail, I will outline the procedures we follow, present our original and current objectives, and summarize our most important findings, emphasizing parts of the work which are most original.

At the start we proposed to study acute altitude illness in its various forms; this proved impracticable and perhaps even dangerous because flying persons to 5360 m from our well equipped Base at 790 m caused many severe symptoms. This procedure and this objective were changed after several years of sometimes frightening effort. We modified the procedure by inserting a staging period of 3–5 days at 2700 m and then of course experienced less difficulty with illness, but more difficulty in finding clear flying weather on just the days when it was most needed. Since Logan High can be supplied only by air from our base 90 miles away, we are at the mercy of weather, snow conditions at the landing site, and depend especially on the skill of our pilots and the serviceability of the tough STOL aircraft which belong to the Arctic Institute. Helicopters are available – at a sickening price – for emergencies. We experienced the dangers of affluence of supply when for several years the Canadian Forces air-dropped anything we wanted, taking delight, so it seemed, in trying to hit our laboratory with propane cylinders. Being able to have almost anything we wanted (and could pay for) brought the usual penalties of surfeit and we have reverted to the austerity imposed by flying everything up in 400-lb loads in our own small aircraft.

During the early years we drew our subjects from volunteers from the Canadian Forces. They were fit, strong, tough, independent and obeyed orders to the letter, but they weren't particularly interested in what we were trying to achieve. To handle house-keeping chores, including shovelling out after storms which often dropped 6 to 12 feet of snow in one day on the camp, we recruited a half dozen mountain types who climbed to the laboratory one month in advance and readied it for use. This arrangement worked well enough but was far from ideal and failed to realize the full potential of the unique and remarkable opportunity offered at Logan High. Consequently we changed the procedure several years ago to what we believe is the optimal design. Since many young people were interested not only in the chance to be on a big mountain but also in the work itself, we realized what an exceptional chance this would give them to learn about cardiopulmonary physiology by working with us, by being subjects, and by talking and reading as the work advanced. For the past few years we have selected 14 to 16 young men or women from a rapidly growing pool of qualified applicants, and we use them as subjects, collaborators, and support personnel. It is a happy and fruitful relationship which we plan to continue.

The volunteers are divided into 2 groups of 8. Group I is flown to the Kluane Lake Base camp (by Canadian Forces aircraft) after 3 days of baseline tests at sea level. Usually they are ready to fly to the staging camp (3280 m) by mid-May and, after a brief stay, they climb slowly to Logan High, and spend 3 or 4 weeks preparing the buildings for the work to come. Group II follows the same pattern 5 weeks behind Group I. The scientists – usually 6 or 8 – are also tested at sea level and fly to Kluane Lake and then to the 3280 m camp with Group II. The scientists then fly directly to Logan High, while Group II climbs. The scientists expect to be mildly ill for a few days, during which they repeat the studies on each other, and then on Group I, who are by then well acclimatized. About the time all of this has been completed, Group II arrives, having taken 8–10 days for the ascent, and the scientists study them, while Group I prepares to walk or ski down to the pick-up site at 3280 m.

This plan gives us 3 populations: Group I, well adjusted by 5 weeks of residence before study, Group II, freshly arrived but somewhat acclimatized by the 7 to 10 days of climbing, and Group III (scientists) only slightly adjusted to 3280 m and studied acutely on arrival at 5360 m. This procedure is safe, much less weather dependent, and richly productive; furthermore it can be replicated year after year.

The majority of volunteers are medical students or in some field related to human physiology; all are between 20 and 29 years of age, in excellent health, with some big mountain experience. Men and women are equally eligible. We have had an exceptional group of people and the project has become not only rewarding, but fun as well. The scientific core group has been together for many years, although new people are added each year. Over the last 10 years our work has included the following broad topics:

1. Distribution of water and electrolytes between the 3 body compartments.
2. Changes in hormone and electrolytes in blood and in urine during acute illness and after acclimatization.
3. Alterations in blood platelets.
4. Changes in pulmonary mechanics and in ventilation.
5. Factors limiting exertion at altitude.
6. Prediction of susceptibility to altitude illness by measuring respiratory response to various stimuli.
7. Examination of stages of sleep by EEG and association of sleep stage with arterial oxygenation.
8. Modification of altitude illness by medication, including alterations in sleep desaturation.
9. Evaluation of frequency, severity, precipitating factors and course of altitude retinopathy.

After several years of effort, but only some data which we consider reliable and reproducible, we abandoned the first 2 studies because of the unpredictable effects of daily variations in sodium and potassium in diet and the difficulty of accurately measuring fluid intake and output. Twice we made heroic efforts to obtain exact measures of potassium, sodium and water intake, but reluctantly concluded that this was simply not practical at Logan High. Then last year, 4 dedicated volunteers tried a rigid formula diet, which I had found quite acceptable for one week at sea level, but they could not accept it for more than a few days at 5360 m and were depressed, irritable and weak after 24 hr. We have concluded that proper metabolic studies are not feasible at a site such as Logan High because, unless intake and output of electrolytes and water are carefully controlled, contradictory and variable data will emerge. Though some of our observations match data in the literature, others do not; furthermore there is wide variation and contradiction among published reports by others.

One theory of aetiology of high altitude pulmonary oedema rests on the observation that platelets tend to clump to form micro-emboli when ambient pressure is reduced (either in divers returning to surface or persons ascending to altitude). Such

emboli have been found in the lungs at post-mortem of persons dying from altitude illness, and using radioactively tagged platelets we have demonstrated such aggregates by scanning the lungs of persons taken to altitude in a decompression chamber. We have also shown a fall in platelets during ascent and stay at Logan High, but a great deal more needs to be done before the platelet emboli theory is proved or disproved. We are continuing the work in a chamber and on Logan.

We have also made a variety of preliminary studies of pulmonary mechanics at various stages in adjustment of altitude at Logan High, supplemented by short term decompression chamber runs in which lung density can be determined non-invasively. Unfortunately, use of this technique is not possible in our mountain laboratory at present. Our work on Logan and in the chamber supports our belief that interstitial oedema often develops early in altitude exposure, well before clinical alveolar oedema is detectable, and may either subside spontaneously or progress to frank pulmonary oedema, a serious, often fatal disease at altitude.

We are actively examining the factors which limit exertion at altitude, a subject which fascinates all climbers and many physiologists and of which we will hear more at this meeting. We find that maximum power output is reduced by half at 5360 m in fresh arrivals as well as in those partially adjusted by 5 weeks of residence. Both recent and earlier arrivals achieve maximal pulse rates 15–20% lower than achieved at sea level. Inability to move enough air limits exertion in most subjects at altitude, whereas muscle fatigue is limiting at sea level.

We have found no reliable predictor of those who would be most or least affected by altitude.

Major alterations in breathing occur during various stages of sleep, and these result in significant arterial oxygen desaturation. The fall in saturation during sleep parallels the severity of morning headache but is not related to other symptoms. Length of stay at altitude does not alter the extent of arterial desaturation nor affect breathing irregularity. Acetazolamide abolishes the periodicity of breathing, raises arterial oxygenation appreciably, and produces sleep which is, so to speak, more restful. However, acetazolamide does not alter awake oxygenation, a finding in accord with data which show no significant increase in pulmonary ventilation attributable to acetazolamide. Others have reported a decrease in arterial oxygen saturation during sleep at sea level and in patients with pulmonary insufficiency, but to my knowledge our studies at high altitude, combining saturation measurements with electro-encephalograms, are new. Not surprisingly, correlation between the severity of morning headache and the

extent of sleep desaturation fits experience gained from sleeping with low flow oxygen high on Everest.

Since we first saw retinal haemorrhages in 2 persons after a short stay at 5360 m in 1967, elucidation of this puzzling phenomenon has been a high priority in our programme. We have examined and usually photographed the retinas of more than 100 persons, some of them repeatedly, during the last 10 years. We have baseline photographs on 25% of these and have done fluorescein studies looking at vascular leakage on many. In our experience the following statements are justified by data now in hand:

1. All persons develop hyperaemia of the optic disc, both venous and arterial vascular engorgement and greatly increased retinal blood flow at this altitude and to a lesser degree somewhat lower. These changes occur within hours of arrival at altitude and disappear more slowly after return to sea level.

2. Retinal haemorrhages occur in different shapes, sizes and at various levels in the retina in more than 50% of all persons going to this altitude, and the more diligently they are sought, the more often will they be seen. Others have found a lower incidence at lower altitudes; we have seen none at 3280 m.

3. Retinal haemorrhage like the vascular engorgement is rarely symptomatic and is almost never detected by the individual unless the haemorrhage occurs in or very near the macula. Then a central blind, or more often blurred, spot is noticed.

Visual field studies have been done on 13 persons at altitude. Four of these showed physiological angioscotomata which did not correspond to retinopathy, if present. One person of the 80–90 individuals photographed has shown ‘cotton-wool spots’ (considered to be due to retinal oedema) and field defects which matched 2 of these spots and which, though smaller and asymptomatic 6 months later, were still present on visual field examination. Four persons have had macular haemorrhage which was noticeable to the individual at the time and still noticeable a year or 2 later although asymptomatic today; follow-up field studies are being done on them.

Although papilloedema appeared to be present by direct ophthalmoscopy of a number of persons (as evidenced by hyperaemia and blurring of disc margins), stereo-pair photographs did not confirm this. Many consider papilloedema of any origin to be due to ischaemia and thus to hypoxia of the optic nerve and thus it might logically be anticipated at altitude. Leakage of fluorescein around disc margins was photographed in a few persons, usually those with the worst headache, and could perhaps be

attributed to minimal papilloedema.

Altitude retinopathy is only weakly associated with other symptoms, with exertion, and speed of ascent, but not with length of stay. We do not yet know its cause. Retinal haemorrhages occur in 30–35% of newborns, although they are uncommon after Caesarean delivery. Hypoxia *in utero*, aggravated during delivery, may be the cause of neonatal retinopathy. The fact that neonates do not have permanent field defects is reassuring to mountaineers though of course not conclusive proof that persistent field defects do not occur. Retinopathy similar to that seen in healthy persons at altitude also occurs often in sick patients chronically hypoxic at sea level owing to disease.

Others are now finding retinopathy and haemorrhage in 33–50% of mountaineers in the Himalayas and elsewhere, and one presumes that this incidence has not changed over the years. Since many thousands of mountaineers have been at risk of haemorrhage I personally doubt that any significant defect in vision persists after exposure to altitude, and my colleagues and I do not consider retinal haemorrhage an indication for descent unless the macula is affected, or the haemorrhages are very massive and symptomatic, or unless evidence of brain oedema – itself an absolute indication for immediate descent – is suspected.

What then does all this mean? Barcroft 50 years ago pointed out how relevant are the lessons from high altitude to the understanding and management of persons with hypoxaemia at sea level. Severinghaus in 1971 agreed. Though ‘pink puffers’ and ‘blue bloaters’ differ in many important respects from the Boningtons, Odells, and Messners of mountaineering fame, there is something in common between them, or more accurately between healthy mountaineers and persons with hypoxia at sea level.

Carbon monoxide poisoning causes cerebral oedema much like that seen in persons who rashly go too high too fast. Asphyxia results in brain oedema; neurosurgeons see the brain bulge in the operating theatre if the anaesthetist skimps on the oxygen. Patients with chronic pulmonary insufficiency desaturate at night and feel worse in the mornings; their retinal fundi look much like those seen at altitude. If hypoxia develops more abruptly, papilloedema, and striking vascular engorgement occur. Victims apparently recovered from near drowning, often develop pulmonary oedema much like that seen in ‘shock lung’ and in heroin overdose. These conditions all produce severe hypoxia, much like too rapid an ascent. The mental dullness, irritability, and sluggishness of the patient with chronic lung disease are very similar to the symptoms seen high in the mountains. One must add too that

cerebral arteries clogged by age may cause brain hypoxia and McFarland has drawn many similarities between ageing and hypoxia. We are beginning to appreciate that the responses to oxygen lack may not all be beneficial: there are bad adaptations and good ones. The former may produce secondary symptoms at altitude as at sea level, abnormalities in blood coagulation being most striking. We are also coming to see that like any eco-system, changes anywhere cause changes everywhere; acclimatization over years, or adjustment over hours both set in train a series of integrated changes whose sum tends to restore oxygen supply toward normal. Some changes are temporary or 'struggle' responses, such as hyper-ventilation and increased cardiac output.

Others are more subtle, take longer to develop, and occur mainly at the tissue level – these are the true acclimatizing changes and as they are perfected, the struggle responses fade.

Indeed, as one ponders the marvellous adaptations which enable the human body to maintain the constancy of its internal environment, despite illness, injury, and abuse, one wonders why we do not learn how to live within our greater global environment, individuals and communities adapting to one another's variations rather than striving to dominate or control. There are many lessons to be learned high on big mountains, not all of them being physiological.