Operation Everest II – 1985

CHARLES S. HOUSTON

Soon after the height of Everest was announced in 1864, mountaineers wondered if it could be climbed, at a time when it was generally believed that a man could not sleep higher than 6000m or climb much above this. In 1892 Clinton Dent, President of the AC and a distinguished mountaineer, surgeon and physician predicted, in a carefully worded paper, that Everest could and would be climbed. Siding with Whymper, and in opposition to Paul Bert, he believed that lack of pressure, rather than lack of oxygen, was the problem at high altitude, and he concluded that supplementary oxygen would be neither necessary nor helpful. We know today that lack of oxygen is the prime, if not the sole, cause of altitude illness and stimulates the acclimatization processes.

In Paul Bert's day the dangers of high altitude had been tragically revealed during a few balloon flights which resulted in death, although curiously a few others went equally high without difficulty. Climbers were describing unpleasant and occasionally fatal problems during the ascent of Mont Blanc and other high peaks. By the start of this century it appeared that the speed of ascent as well as the altitude reached determined the presence or absence of altitude illness. The slower the rate, the less the distress and the better the acclimatization.

Longstaff's ascent of Trisul in 1907 set an undisputed altitude record which has been steadily advanced, until today a score of men have reached the highest points on earth without supplementary oxygen. Whether they have used siege or rush tactics, success, even survival, have depended on the perfection of their acclimatization.

The earliest mention of acclimatization seems to have been in 1550 by Haidar who described illnesses in the mountains of Central Asia and pointed out that only newcomers were affected: altitude residents seemed immune. Mountaineering doctors in the late 19th century adduced several intriguing theories to explain both illness and well-being, though we know today that many were quite wrong.

In1920 Kellas calculated the physiological adjustments which would be needed to climb to a height where there was less than a third of the amount of oxygen available at sea-level, and he concluded that Everest could and would be climbed. Four years later, Norton came close to proving Kellas right when he actually climbed to within a few hundred feet of the summit. Haldane, Barcroft and later Dill, Keyes and others outlined the framework of this adaptive protection.

Hingston, Greene and Henderson soon agreed with Kellas, but not until 1946 did men actually work at an altitude equivalent to the summit after a slow ascent in a decompression chamber. This study (called Operation Everest)

provided rich data defining the beautifully integrated changes of acclimatization which made this possible – under laboratory conditions.

The most thorough and productive study of acclimatization on a mountain was that done by the Silver Hut Expedition of 1961, whose members lived and worked for four months at 5800m near Makalu, measuring various components of acclimatization.

In 1985 Operation Everest was repeated in a decompression chamber at the US Army Research Institute for Environmental Medicine, using state-of-the-art science to improve our understanding of acclimatization, not only to satisfy curiosity as to how mountaineers can achieve the summit but, more importantly, to see what lessons learned at altitude could be applied to sick men at sea-level – a question Barcroft had asked 50 years earlier.

It took five years to perfect plans for Operation Everest II during which eight subjects lived in a large three-roomed decompression chamber. Following a three-day control period at sea-level pressure, the chamber was 'climbed' at a rate similar to that used in Operation Everest I and roughly that of several successful Everest summit parties. The subjects lived for 12 days at 7300–7600m, and each day one or two of them made a rapid ascent to 8800m where they remained for several hours while measurements were made on the subjects at rest and at work.

OEII was a true team effort in which 25 scientists and 40–50 support people shared the effort of several dozen discrete studies. Our overall goal was to examine all stages of the oxygen transport system during acclimatization to lack of oxygen. This includes acquisition of air by breathing, passage of oxygen from lung to blood by diffusion, carriage of oxygen from lung to tissues by circulating haemoglobin, passage of oxygen from blood to cells by diffusion, and utilization of oxygen by mitochondria within the cells. We also looked at the reverse process – the carbon dioxide transport system – but in less detail. We studied the relationship between ventilation and circulation in the lungs and calculated the ventilation-perfusion ratio. We matched the stages of sleep to blood oxygen levels and electroencephalographic tracings.

Subjects were weighed underwater and CAT scans were done to determine their percentage of body fat before and after the project. All food and fluid ingested and the total work done were recorded to obtain the energy balance at altitude. Retinal photographs were taken regularly. Many tests were run on blood, and muscle biopsies were taken to learn whether muscle structure changes during acclimatization and how muscle uses oxygen at rest, during and after strenuous work. How much work a man could do when well acclimatized was determined by measuring the maximal oxygen uptake (VO_2Max) while cycling against increasing loads.

The most ambitious study required passing a small tube (Swan–Ganz catheter) through an arm vein, into the heart, past its valves and into the lung. This enabled us to measure blood pressure in the pulmonary artery and heart, cardiac output, and (especially important) the amount of oxygen remaining in venous blood returning to the heart from all parts of the body. The ease with which oxygen passed from air to blood in the lung (diffusing capacity) was calculated by injecting into a vein a solution of inert gases and measuring their

concentration in expired air. Together with the cardiac catheterization studies, this gave the ventilation-perfusion ratio. Other tests included psychometric, neuro-muscular and endurance studies.

To our surprise and disappointment, these men did not acclimatize as fully as we had hoped. This may have been because: (1) the close confinement made strenuous exertion difficult; (2) the rate of ascent may have been somewhat too rapid; or (3) other stresses experienced on high mountains may contribute more than we believed to acclimatization.

At 6000m headache and inability to sleep nagged most of the men to a degree which led us to 'go down' a thousand metres or so for two days; this brought great improvement, and above 6000m these symptoms were less. Appetites decreased above 3000–3700m and all lost an average of 10% of their starting weight, most of it muscle rather than fat. This was also a surprise because they were given anything they chose to eat, and because calculated energy intake was actually greater than energy output. We concluded that hypoxia, in addition to causing anorexia, contributed to weight loss.

Sore throats became a serious problem above 6700–7000m, as is the case on high mountains. But the chamber was kept at a comfortable temperature (72F) and the humidity was held above 80%. No infection was found. We concluded that not the dry cold air (as had been thought), but some element of hypoxia caused the sore throat and cough so often complained of by climbers.

In medical terms the physiological data can be summarized as follows:

Heart and Circulation

As altitude increased:

Right atrial and pulmonary wedge pressures remained normal.

Resting pulmonary artery pressure increased from 15 to 34 Torr.

Cardiac output increased with level of work but the relationship to a given oxygen uptake was maintained.

During work: the pulmonary artery pressure increased from 33 to 54 Torr.

At rest: the pulmonary vascular resistance increased from 1.2 to 4.3 Wood units.

At extreme altitude: 100% oxygen lowered cardiac output and pulmonary artery pressure but not pulmonary vascular resistance.

Left ventricular function was sustained or even enhanced despite right ventricular hypertension and reduced preload.

Heart rate for a given oxygen uptake increased with altitude and was only slightly slowed by breathing oxygen.

Systemic blood pressure did not change as altitude increased, but did increase with 100% oxygen, suggesting different control mechanisms for pulmonary and systemic blood pressures.

These observations suggest that the heart is not a limiting factor at extreme altitude.

Ventilation

As altitude increased:

The volume and rate of breathing at rest and during work increased progressively.

Increasing ventilation was effective in ensuring adequate tissue oxygenation during acclimatization. Respiratory muscle fatigue was not a limiting factor at the highest altitudes.

Ventilatory drive was increased rather than depressed.

A variable but increasing ventilation-perfusion mismatch was found which correlated with pulmonary artery pressure. This was consistent with increasing interstitial alveolar oedema.

These studies suggest that at extreme altitude the lung is an important factor limiting the ability to work.

Work

The relationship of oxygen uptake to workload was consistent throughout.

Maximal oxygen uptake on the summit was roughly 25% that at sea-level.

For a given level of work, anaerobic metabolism contributed more energy as altitude increased, though at exhaustion its maximum contribution was less than at sea-level.

Peak blood lactate during exercise at extreme altitude decreased.

These observations suggest that acclimatization to extreme hypoxia did not maximize oxidative function.

Oxygen Transport and Delivery

As acclimatization progressed:

The number of capillaries per muscle cross-sectional area increased, but the number of capillaries per muscle fibre did not change.

Myoglobin content of both Type I and II muscle did not change.

Oxygen transport at extreme altitude was defended principally by increased ventilation and a decrease in mixed venous oxygen.

A given oxygen uptake was achieved by lowering mixed venous oxygen in preference to increasing cardiac output.

During maximal exercise at altitude mixed venous oxygen pressure fell to between 10 and 14 Torr; at sea-level it did not drop below 20 Torr.

At sea-level more than 80% of oxygen O_2 delivered was used and at higher altitudes extraction was similar though less on the summit.

Tissue diffusion may be an important limiting factor at extreme altitude.

Nutrition

Although ample appetizing and nourishing food was provided, appetite and caloric intake declined.

All subjects lost more weight than accounted for by comparing calculated

energy expenditures with measured caloric intake.
CT scans and hydrostatic weighing showed that most of the weight-loss was muscle mass.

Sleep

All subjects showed greater arterial desaturation during sleep than when awake at altitude and this was unaffected by sleep stage.
At the greater altitudes all subjects showed periodic breathing and apnoea during sleep; although apnoeas were abolished by breathing oxygen or carbon dioxide, periodicity was not.
The hypoxic ventilatory response during sleep did not increase as much with acclimatization as did the awake HVR.

Could these men have climbed the Hillary step near the top of Everest? Could they have reached the summit and still have had some reserve? Only a few have done so without extra oxygen on that mountain. How do the OEII subjects compare with the Everest summiteer? Are they physiologically different from you and me, or from Messner, Roskelly, and many others? Our findings agree with others: they are not!

It is important to remember that in 1946 and 1985 we doctors were watching and studying these acclimatizing men with our normal sea-level eyes and brains; no one who is unaffected by hypoxia can observe men on top of Everest. Those who have climbed without oxygen have told us that they are close to the limit. They hallucinate, they stagger, they gasp for breath, every step is described as a tremendous effort, the world seems unreal. Our subjects, as we saw them clearly, were not so badly affected.

On the other hand they were not as well acclimatized as members of an expedition who have spent months on a big mountain. Why, in such a pampered state, did they not acclimatize better? Was ascent too fast, time at altitude too short? Are the stresses of a mountain a help rather than a hindrance in acclimatization? Perhaps the extreme work of climbing, or the bitter cold, or the anxiety and tension all enhance acclimatization more than we anticipated. We don't know.