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Preliminary Report on Studies of Erythropoiesis and Adrenal-Cortical Function on Mt. Everest

William E. Siri

Men engaged in climbing the highest Himalayan peaks are unique subjects for research on physiological adaptation, particularly the body's regulatory mechanisms. Driven by extraordinary motivation, climbers voluntarily subject themselves for extended intervals to submarginal environmental conditions that could not long be tolerated by subjects in a laboratory research setting. The state of stress induced by prolonged exposure to severe hypoxia and further aggravated by fatigue, cold, anorexia, insomnia, and psychogenic factors often leads to maximal adaptation followed by progressive deterioration when the homeostatic balance can no longer be maintained. Certain features of regulatory systems may become more evident under these conditions than in the more regulated circumstances of a laboratory experiment. This was the premise for the studies conducted on members of the American Mt. Everest Expedition in the Spring of 1963.

Among the more familiar responses to high altitude is the increased rate of red-blood-cell production. With continued exposure, this leads ultimately to a stabilized polycythemia that is related in an ill-defined way to severity of hypoxia. The processes, and more specifically the hormone erythropoietin, that accelerate erythropoiesis under hypoxic stimulus are presumed by most investigators also to be responsible for regulation of red-blood-cell volume under normal conditions.

Numerous studies of hematopoietic activity have been made at altitudes up to 15,000 ft, notably those of Hurtado and Delgado (1), Merino (2), Lawrence et al. (3,4) and Reynafarje et al. (5). More recent observations by the author at 17,000 ft in Bolivia suggest the possibility of an anomolous increase in hemoglobin synthesis relative to that anticipated by projection of observations made at lower elevations. The first object of the work on the Everest Expedition was to extend these observations to high altitudes corresponding to levels of hypoxia well below that for which human adaptation seems possible. The intended studies included observations on plasma-iron turnover to determine rate of hemoglobin synthesis, assay of erythropoietin in 24-hr urine collections, and changes in blood elements and chemistry as a function of altitude and duration of exposure.

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The second object of the physiological studies was to examine indirectly the responses of the adrenal cortex to demands for maximal adaptation by the body to conditions that exceed physiological limits for chronic exposure. The vital roles played by the adrenal cortex as a regulatory organ and in adaptive processes are firmly established. Secretion of cortical hormones, mediated in part by ACTH, exercises a strong and often a controlling influence over metabolism, the cardiovascular and respiratory systems, and the body's chemistry.

The assessment of adrenal-cortical activity during the varied stresses imposed by the Expedition was based primarily upon urinary excretion of cortical steroids and their catabolites. Twenty-four-hour urine collections were obtained under conditions and at times when the climber was severely stressed, and particularly when he exhibited signs of deterioration. In each instance a second 24-hr urine collection was obtained following administration of ACTH. The differences in urinary ketosteroids before and after ACTH should provide a rough measure of adrenal cortical reserve. Whether or not the relative as well as total excretion rates of the several major adrenal corticoids is altered by stress may become evident on fractionation of the urinary ketosteroids.

Collateral tests of adrenal-cortical function were necessarily limited to simple procedures that could be performed at intervals under the restrictive conditions of the Expedition and environment. For the most part these included white-blood-cell differential, cardiovascular response to exercise, urine chemistry, and other tests described in the next section.

Base-line values of all the physiological parameters measured in the field were obtained from each climber at Donner Laboratory prior to departure of the Expedition.

Conditions conducive to severe hypoxia and prolonged stress were encountered in the course of climbing Mt. Everest, but it is difficult to assess the actual level of tissue hypoxia. The length of exposure was sufficient to acquire a high degree of acclimatization to hypoxia and to develop signs of general deterioration. Most climbers remained at 21,500 ft or higher for several weeks and one remained at this altitude for six weeks. The most telling effects of hypoxic exposure and exhaustion were evident in the five climbers and one Sherpa who reached the summit of Mt. Everest (29,028 ft), all of whom spent intervals ranging from three and a half to 8 hr above 28,000 ft without oxygen. Normally, however, supplemental oxygen was used above 23,000 ft at rates from 2 to 4 liters per minute for climbing and one half to 1 liter per minute for sleeping.

The description presented here of the studies on Mt. Everest is intended as no more than a preliminary report. Neither findings nor conclusions can be

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offered until data and samples collected in the course of the Expedition are fully analyzed.

PRELIMINARY RESULTS

Erythropoietin, the hormone that stimulates red-blood-cell production, can neither be detected in the healthy person at sea level nor in persons chronically exposed to moderate altitudes where, presumably, the erythropoietin concentration is increased in response to hypoxia. Even at 17,000 ft, the author in collaboration with Dr. Donald Van Dyke was unable to detect erythropoietin in Bolivian natives who lived at this altitude (data to be published). The sensitivity of the bioassay for the hormone is inadequate for the low concentrations normally present. On the other hand, Van Dyke and Garcia, in this Laboratory, and others (6-10) found a transient high concentration in rats and mice during the first few days exposure to severe hypoxia. A clear indication of a similar response in man was reported from this Laboratory, describing results obtained when a human subject was acutely decompressed to a pressure equivalent to 16,400 ft and exposed for four days (11).

In pursuing this line of investigation on the Mt. Everest Expedition, 24-hr urine collections for erythropoietin assay were obtained from climbers two or three days after they had for the first time made an ascent from 17,800 ft to 23,000 ft or higher within the space of two days. Although this is a difference of only 5,000 ft the possibility existed that it would produce a tissue hypoxia comparable to that resulting from decompression from sea level to 16,400 ft and hence induce a detectable, if transient, concentration in erythropoietin.

Additional 24-hr urine collections were obtained from climbers who had spent more than a week at 21,500 ft and at 23,000 ft to determine if detectable steady state concentrations exist at these levels of hypoxia.

Urine collections were passed through collodion membranes for erythropoietin extraction. The membranes were dried, sealed under nitrogen, and sent to Donner Laboratory for assay by Dr. Van Dyke and his associates. None of the samples assayed thus far contained detectable amounts of erythropoietin. From this it must be concluded that: a) the level of tissue hypoxia in an acclimatized man at these altitudes is less severe than anticipated, or b) the erythropoietin was inactivated in the collection procedures, or c) erythropoietin makes an appearance only in response to acute exposure in unacclimatized subjects.

The rate of hemoglobin synthesis can be roughly determined from the turnover rate of iron in the plasma. These rates were measured in the climbers at Berkeley (sea level) prior to the Expedition's departure to establish normal values, using Fe^{59}-labelled plasma. The measurements were repeated at 17,500 ft and at 21,500 ft to determine the relative increases in hemoglobin synthesis for

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these levels of hypoxia. In general, the rate of plasma-iron disappearance observed in the climbers at 17,500 ft, and therefore presumably the erythropoietic activity, was about double that for sea level. This is in agreement with earlier observations made in Bolivia on subjects at 17,000 ft, including residents at this altitude and also persons brought up from lower altitudes. The plasma-iron disappearance rate found at 21,500 ft was less clear. In one subject, it was nearly three times that for sea level; in another it was twice the rate.

Blood smears for reticulocyte counts were taken at intervals during the course of the Expedition. A preliminary examination of the slides suggest that these immature red cells may be elevated at 2 to 3.5% about the time the climbers reached Base Camp (17,500 ft) as compared to 0.5 to 2.0% for the pre-expedition values. Later when the climbers had been to much higher altitudes and returned to Base Camp they appeared to be devoid of reticulocytes. Slides for white-cell differential count were also taken but have not been counted.

Hemoglobin concentration increased rapidly on arrival at 17,500 ft to about 18 g percent. With continued exposure to this and higher altitudes, the concentration increased slowly to the range 20 to 23 g percent, but showed little tendency to rise significantly higher, although several climbers spent a month or more above 21,500 ft. Hematocrit and red-blood-cell count paralleled more or less the changes in hemoglobin concentration. There was no significant change in the platelet count.

A more striking change, because of its rapidity, was the reduction in hemoglobin concentration to the normal range on returning to low elevations. Within two weeks after leaving Base Camp, hemoglobin concentrations that were 22 g percent had dropped to 16 g percent. Rehydration does not appear implicated in this rapid reduction in hemoglobin concentration. It remains to be determined if complete cessation of red-cell production would account for the apparent rapid reduction.

Twenty-four-hour urine collections for adrenal-cortical-steroid analyses and urine chemistry were obtained under a variety of conditions, all of them involving stressful circumstances. At the end of the first collection, 80 I.U. of a long-acting ACTH preparation were given to the subjects, intramuscularly, before starting the second 24-hr urine collection. Such collections were obtained from all the climbers who reached the summit and from the support teams that went to the highest camps. The collections were started while the climbers were still high on the mountain and completed about the time they reached Base Camp. In one instance, a collection was started before a climber reached the summit and extended through the night in which he was forced to bivouac in the open at 28,000 ft without oxygen. Other climbers provided collections after prolonged stays at 21,500 ft and 23,000 ft during which they showed signs of

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Medical officer Gilbert Roberts' and William Siri, scientific coordinator of the American Mt. Everest Expedition, measure cardiac output with radioactive iron 59 in psychologist James Lester at Base Camp, using a portable scintillation probe.

progressive deterioration. The cortical steroids were concentrated from the urine collections by ether-ethanol extractions whose volume was then reduced by evaporation, and the concentrate was sealed under dry nitrogen. Results of cortical steroid assays and electrolyte analyses will be presented in a later report.

A variety of simple tests was made at intervals on members of the Expedition to detect gross responses to hypoxia and possibly to changes in adrenal cortical function. Among these were tests of heart rate and blood pressures at rest and following exercise, of breath-holding times, muscular strength, body weight, total body water, electrolyte excretion, and the common hematological measurements.

Basal heart rate and resting blood pressure increased almost immediately as the Expedition moved to higher altitudes. They remained elevated for several weeks after arrival at Base Camp but as acclimatization progressed, heart rate and blood pressure slowly returned toward pre-expedition values at sea level. After two months at altitude, the readjustment seemed complete, and for several climbers heart rate and/or systolic pressure were significantly lower than their normal values at sea level although diastolic pressure remained consistently elevated.

Cardiovascular response to moderate exercise was tested by observing the elevation and the recovery patterns of both heart rate and blood pressure following a three-minute exercise on a 15-inch-high step at the rate of 22 complete steps per minute. Measurements were made continually for five minutes after the exercise ended. In general, the rate of recovery to pre-exercise resting values did not appear to change significantly throughout the course of the Expedition, although there were notable exceptions. On the other hand, the elevation in

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heart rate and pulse pressure produced by the exercise changed progressively with acclimatization. In the first week at altitude when symptoms of hypoxia were most evident, the exercise pulse pressure and heart rate were slightly higher than at sea level. During the two months that followed, these responses to the exercise decreased steadily until at the end of the period the elevation in pulse pressure was uniformly smaller, and for some climbers it was less than half that seen in the test at sea level. A similar trend was also observed for the heart-rate response to the test although in four climbers heart rate was hardly stimulated by the exercise. Among the half dozen Sherpas who were tested at 21,500 ft, heart-rate response and recovery were comparable to those for the American climbers, but four of the six Sherpas exhibited almost no elevation in pulse pressure. One of the remaining two showed responses comparable to those of the climbers, but the other was clearly hypertensive.

Breath-holding times were obtained after a normal expiration and after a maximal inspiration. Holding times under both conditions decreased markedly with altitude up to a point but did not appear to be greatly affected by length of stay at altitude. At low altitude(6,400 ft) breath-holding times were about 30 and 90 sec, respectively, for normal expiration and maximal inspiration. These times were reduced to 10 and 25 sec at 17,800 ft and were not substantially shorter at 21,500 ft. Degree of motivation did not seem to affect the holding time by more than a few seconds at altitude but may have had greater influence at low altitude where the end effects were less disagreeable and their onset slower.

Muscular strength was measured with a hand dynamometer by observing the maximum force exerted and the duration that one half the maximum force could be held. Some improvement occurred initially, probably the result of "training" in the use of the device, but no significant impairment of strength, as measured in this manner, developed later in the course of the Expedition as had been anticipated. In contrast to this it was uniformly the subjective feeling among the climbers that their capacity for heavy work diminished steadily during their stay on the mountain and that it was significantly impaired at the end of the Expedition.

Weight loss during the Expedition and particularly when climbers were higher than Base Camp was continuous and in a few instances severe. For all but two men losses ranged from one-eighth to one-fourth of body weights recorded in Berkeley prior to departure. This was also approximately the range in proportion of depot fat at the outset as determined from body density and total body water. While the bulk of the weight loss was unquestionably depot fat there were also strong indications of lean tissue wasting. This is supported by the significant reductions in total body water observed toward the end of the Expedition. Diminished appetites, which also grew more selective at high altitude,

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and the relative deficiency of acceptable foods high in proteins may have resulted in negative nitrogen balances as well as insufficient caloric intake for most of the climbers. The exceptions to this were two climbers who maintained their weights by deliberately forcing themselves to eat adequately. It is perhaps significant that these were the two men who climbed Mt. Everest via the difficult West Ridge.

The absence of obvious signs of cerebral damage among the six men exposed for hours to altitudes as high as 29,000 ft without oxygen is perhaps remarkable. In the history of Himalayan mountaineering there is suggestive evidence of brain damage, in some instances perhaps irreversible, in climbers exposed to somewhat lesser altitudes. The effects are said to be expressed as impaired memory and personality changes, particularly chronic depression. These and other signs were not evident in the American Everest climbers, but it must be emphasized that a careful search for evidence of brain damage was not conducted.

EXPEDITION OPERATIONS

APPROACH MARCH. The 18 climbers who left Kathmandu on February 20, 1963, on the 180-mile march to Mt. Everest had been carefully selected for experience, skill, and character. Many of them had climbed to altitudes greater than 20,000 ft and were therefore familiar with hypoxia and known to tolerate it well. Sherpa climbers, 34 in number at the outset and later increased to 47, accompanied the Expedition as a service group. Their principal function was to carry equipment and supplies to camps on the mountain.

Members of the Expedition could not carry all the necessary supplies because of the quantity of materiel needed at the nine high-altitude camps and also due to the climbers' inability to carry heavy loads to high altitudes without supplemental oxygen. About twenty-five tons of Expedition supplies and equipment were first transported from Kathmandu to Base Camp on the backs of 909 porters. Later five tons of this materiel were carried to high camps by the Sherpas.

Acclimatization stops at intermediate altitudes during the approach march were essential. To proceed directly to Base Camp at 17,800 ft from the relatively low level of the approach march would have incapacitated the Expedition from mountain sickness, the symptoms of which include headache, nausea, vomiting, muscular weakness, loss of sleep and appetite, and a variety of mild mental abberations. From Namche Bazar, the Sherpa village at 12,000 ft, the Expedition therefore proceeded by easy stages to Base Camp. Although greatly reduced, mild symptoms of hypoxia asserted themselves in varying degrees among the climbers after they passed 14,000 ft.

The physiological studies were begun toward the latter part of the approach march when the effects of the steadily increasing altitude began to take

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effect. However, the scope of the work was limited because of the daily movements of the Expedition, the temporary nature of camps, and persistent respiratory and gastrointestinal infections. Blood smears for reticulocyte and white-blood-cell differential counts were started at 12,000 ft. Pulse rate and blood pressure recovery after a standardized exercise were also measured, and such routine measurements as basal heart rate were recorded daily. Weights were checked every few days.

Base Camp was established on March 22 at the foot of the Khumbu Glacier ice fall, the only practicable route of ascent on Mt. Everest from the south. Along with other base facilities a laboratory tent, 10 by 10 ft, was erected and equipped for the physiological studies. Within a few days the first reconnaissance team had explored the lower half of the ice fall but did not establish a camp because of the hazards presented by the constant movement in the 2,000 ft of shattered ice, crevasses, and hanging ice walls.

A second reconnaissance ended at the midpoint in the ice fall when a huge ice wall collapsed without warning on the roped team of men. Climber John Breitenbach was killed instantly, and his body never recovered from beneath the hundreds of tons of ice. Other members of the six-man team were injured and buried but were able to effect their own rescue. During the months that followed, hundreds of man-days were spent without further accident in traversing the ice fall that, however, now was a mental as well as a physical challenge.

Within a short time Camp I (20,000 ft) was established at the top of the ice fall. Camp II (21,500 ft) and Camp III (23,000 ft) followed in quick succession. Using oxygen above 23,000 ft, the climbers firmly anchored Camp IV in the ice of the steep Lhotse face at 25,000 ft, and on April 16, Jerstad and Pownall climbed to the South Col, the 26,200-ft-high saddle between Mt. Everest and Lhotse where they placed Camp V. This was the route pioneered by the Swiss in 1952 and followed to the summit for the first time, by the British Expedition in 1953.

All climbing by Expedition members beyond Camp III was now brought to a halt partly by persistent storms but more by the need to supply the string of camps with provisions and equipment for the ascent. During this time a climber at Camp II developed thrombophlebitis in the lower right leg. Under the care of Dr. Roberts, the senior physician, no further complications developed and the man was removed permanently to Base Camp.

FIRST SUMMIT ASCENT. In late April all camps up to the South Col had been fully stocked by relay teams of Sherpas, and on the last day of the month James Whittaker and Nawang Gombu, with Norman Dyhrenfurth, leader of the Expedition, and Ang Dawa in support, were in position at Camp VI (27,300 ft) for a

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summit attempt. Despite unusually high winds on May 1st, which drove clouds of fine, dry snow over the faces and ridges, Whittaker elected to make the summit attempt, realizing there would be no other opportunity. He and Gombu reached the summit in the early afternoon after 7 hr of climbing, using oxygen at 4 liters/min. However, the oxygen supply was exhausted at the summit and nearly 4 hr were spent without oxygen descending 500 ft to the point where spare tanks were cached.

Dyhrenfurth and Ang Dawa reached an elevation of 28,200 ft before they were compelled to turn back by a rapidly diminishing oxygen supply. Their feat was outstanding by any standards but in view of Dyhrenfurth's age of 45 years it was especially notable.

A few days preceding the first summit climb, a new, potentially serious medical problem arose when a climber in the support team was striken with primary pulmonary edema at 25,000 ft. Only in recent years has this acute form of pulmonary edema been described and generally recognized as a distinct disease entity. Its etiology is unknown, and specifically, it does not appear due to infection nor to primary cardiac failure, although it is thought by some authorities that ischemic heart disease and viral infection of the respiratory tract may predispose. The onset of the disease is rapid and if left untreated proceeds usually to fatal termination in 12 to 24 hr. Oxygen administration is essential and rapidly effective treatment; the value of digitalis in addition to oxygen is still conjectural.

In this instance the climber was given both oxygen and digitalis and moved permanently to Base Camp (17,800 ft) where he recovered without further treatment, although a tendency to fatigue continued for several weeks.

SECOND SUMMIT ASCENT. The original plans for the Expedition called for an attempt on Lhotse, Mt. Everest's 27,923-ft neighbor, following the ascent of Mt. Everest. Early in the Expedition, however, a great enthusiasm developed for pioneering a new route on Mt. Everest. Consequently, by common agreement, reconnaissance of the unexplored West Ridge of Mt. Everest took precedence over Lhotse as the secondary objective. The final plan called for a "summit meeting," in which a second ascent via the South Col would be made simultaneously with an ascent via the West Ridge. Work on the West Ridge, which began soon after the ice fall was penetrated, continued through April and May in the face of the severest difficulties.

During the weeks following the first ascent, climbers for the second assault rested at Base Camp while other climbers extended the West Ridge route and restocked the high camps leading to the South Col. The West-Ridge team had already suffered setbacks. An avalanche had wiped out a camp, attrition of

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climbers and especially Sherpas severely cut support, and supplies were dwindling rapidly. On May 17, five days before the scheduled summit date, an intense storm struck at night, blowing two tents at the 25,000-ft camp on the West Ridge several hundred feet down the north face of Everest where its occupants were precariously held by climbing ropes. In the morning the remaining tent and equipment were blown into Tibet.

Two days later when the storm had spent itself, the group ascended the West Ridge again to 25,000 ft to rebuild the camp. The following day five climbers and the few remaining Sherpas fit for work carried loads to 27,200 ft, where Dr. Thomas Hornbein and Dr. William Unsoeld were left to carry on what was believed could now be only a high reconnaissance.

On the south side of the mountain, the summit team of Luther Jerstad and Barry Bishop, with Dr. David Dingman and two Sherpas one camp behind in support, ascended the South Col route, reaching the highest camp on May 21st.

May 22nd, the last possible day for the ascent, was windy and cold but tolerable. At six in the morning, Bishop struck a match to light the butane stove and from the violent explosion that followed he and Jerstad were left with smoldering remnants of beards, Jerstad with slightly injured eyes, but otherwise not seriously burned. Because of a late start as a result of the accident and the debilitating effects of weeks at high altitude, they reached the summit at the dangerously late hour of 3:30 PM, exhausting the last of their oxygen supply. At 10:00 PM that night they were still well above 28,000 ft attempting to descend in total darkness.

The same morning Hornbein and Unsoeld left their 27,000-ft camp on the West Ridge and continued to climb throughout the day, using only 2 liters per min oxygen and none at all when one stopped to belay the other. The climbing here was far more difficult, calling for a high order of skill on the smooth rock walls and down sloping slabs covered with fresh snow. By 1:30 PM they had expended all their climbing hardware, and to climb beyond this point meant they would have no alternative but to continue to the summit and then down the technically easier South Col route. After a careful appraisal of their situation they elected to go on, and at 6:30 PM they stood on the summit in rapidly dwindling daylight and with little remaining oxygen.

Late in the evening, descending the unfamiliar South Col route they encountered Jerstad and Bishop who had been halted by the obvious hazards of climbing at 28,000 ft at night without oxygen. Under the circumstances there was no choice but to bivouac until daylight. Bishop's toes and several fingers had already frozen, and by morning Unsoeld's toes were frozen, and Jerstad sustained frostbite in the toes and fingers but less severe than in the other men.

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For reasons not altogether clear, Hornbein was relatively unaffected. In the light of early morning as the four men continued the difficult descent they were met by Dingman and a Sherpa bearing oxygen cylinders and fluids, and the descent to Base Camp continued without further serious incident. The damage from frostbite, however, required subsequent amputation of most of Unsoeld's and all of Bishop's toes.

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AMERICAN MT. EVEREST EXPEDITION MEMBERS

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