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PHYSIOLOGICAL AND MEDICAL ASPECTS OF THE HIMALAYAN SCIENTIFIC AND MOUNTAINEERING EXPEDITION, 1960-61

BY

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The Himalayan Scientific and Mountaineering Expedition of 1960–61, led by Sir Edmund Hillary, set out from Katmandu on September 14, 1960, and returned in June, 1961, having spent eight months above 15,000 ft. (4,570 m.). It was the first expedition to winter in the Himalayas at high altitude and the first to spend more than six weeks at or above 19,000 ft. (5,790 m.). To do this a much higher standard of protection from the weather was necessary than is usual on climbing expeditions, and a prefabricated hut was taken out to the Himalayas in sections. The hut was set up at the head of the Ming Bo glacier at an altitude of 19,000 ft. (5,790 m.) (barometric pressure 380 mm. Hg) and occupied continuously for five and a half months. There was also a staging camp at 18,000 ft. (5,480 m.) and a tented base camp at 15,100 ft. (4,600 m.). In May, 1961, the expedition moved over to Mt. Makalu (27,790 ft.; 8,470 m.) for an attempted ascent of the mountain without oxygen equipment. Oxygen equipment was, however, provided for medical and rescue purposes.

Scientific Objectives

The scientific programme consisted of physiology, zoology, glaciology, and meteorology. The main emphasis was on physiology, as is evident from the composition of the scientific team. The objectives in this field were to fill in certain gaps in our knowledge of man's adaptation to extreme altitude, and to study some of the effects of oxygen lack on men living at or above the limit of altitude at which complete acclimatization is possible.

The composition of the wintering party with whose work this communication is chiefly concerned is given below. Other scientists were with the expedition for shorter periods, mainly in the first three months.

B. C. Bishop, National Geographic Society, Washington, 6, D.C. (glaciologist and meteorologist).

M. B. Gill, Dunedin University, Dunedin, C.2, New Zealand (medical student).

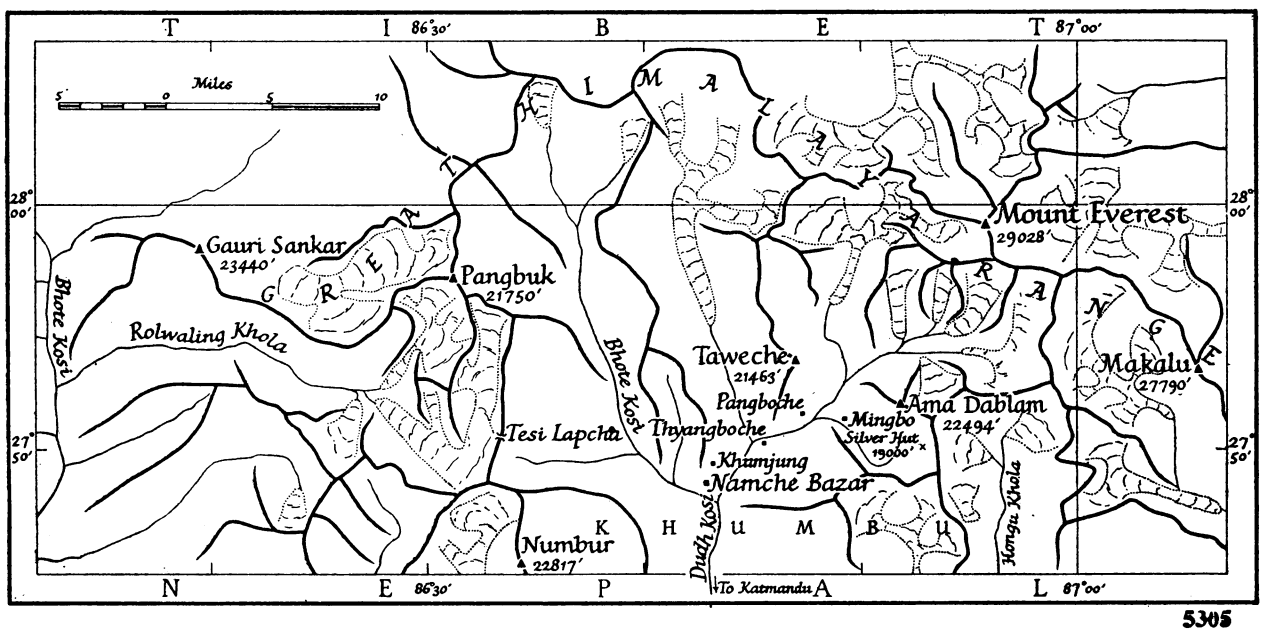
S. Lahiri, Presidency College, Calcutta, 12, India (physiologist).

J. S. Milledge, Birmingham University and Southampton Chest Hospital, Southampton, Hants (physician).

B. Motwani, Defence Science Laboratory, New Delhi, India.

L. G. C. E. Pugh, Division of Human Physiology, National Institute for Medical Research (physiologist).

W. Romanes, Auckland, New Zealand (builder and electrician).



M. P. Ward, Department of Surgery, London Hospital, London E.1 (surgeon).

J. B. West, Department of Medicine, Postgraduate Medical School, Hammersmith Hospital, London W.12 (physiologist).

T. O. Nevison, U.S. Air Force Medical School, was also a member of the physiological team, but was absent during the winter.

Barometric Pressure

It was again confirmed (Pugh, 1957) that barometric pressures in the Himalayas, as in other mountainous regions, are higher than would be expected from the international altimeter calibration used in aviation. The lowest barometric pressures at the hut at 19,000 ft. (5,790 m.) (the Silver Hut) (Fig. 1) occurred in February and the highest from March onwards. The range was from 372 to 384 mm. Hg, which is equivalent to an altitude variation of 750 ft. (228 m.). Extrapolation from the data collected on Mt. Everest in 1953 and Mt. Makalu in 1961 suggests that the pressure on the summit of Mt. Everest (29,028 ft.; 8,845 m.) is about 250 mm. Hg, which is equivalent to 27,500 ft. (8,380 m.) on the altimeter scale. This affords some explanation for the seemingly impossible feat of ascending to within 1,000 ft. (914 m.) of the summit without supplementary oxygen, which has been done altogether by eight men.

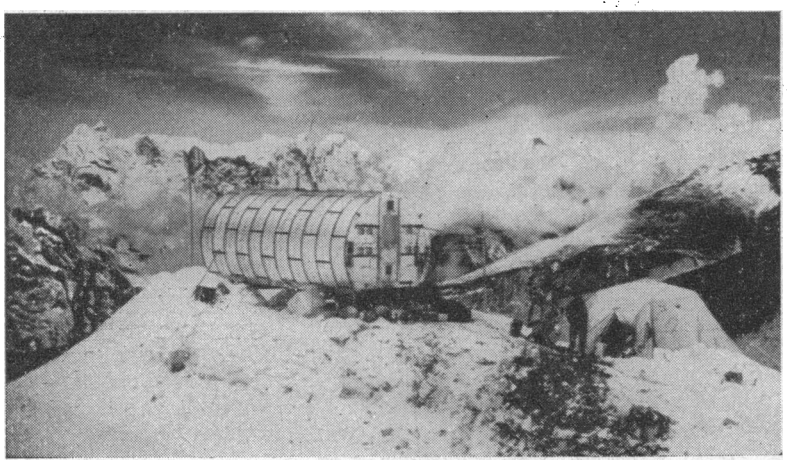


FIG. 1.—The Silver Hut.

Alveolar Gas

Table I shows the composition of alveolar gas at the Silver Hut and at various camps on Mt. Makalu. The samples from the Silver Hut were Otis-Rahn samples, while those from Makalu were Haldane-Priestley end-expiratory samples. The results agree well with previous data from Mt. Everest, which did not, however, extend to such high altitude (Pugh, 1957). The samples from 25,700 ft. (7,830 m.) were taken by M. Gill.

TABLE I.—*Composition of Inspired and Alveolar Gas at Various Altitudes from Sea-level to 25,700 ft. (7,830 m.)*

Altitude ft.	Altitude m.	Barometric Pressure (mm. Hg)	P_{IO_2} (mm. Hg)	P_{AO_2} (mm. Hg)	P_{ACO_2} (mm. Hg)	$P_I - P_{AO_2}$ (mm. Hg)	No. of Samples
Sea-level		750	150	110	38	40	
19,000	5,790	380	69	45	22	24	21
21,000	6,400	344	62	38	21	24	5
24,400	7,430	300	53	34	16	19	8
25,700	7,830	288	50	33	14	17	8

The values for inspired P_{O_2}—that is, the P_{O_2} of atmospheric air warmed to 37° C. and saturated with water vapour—give an idea of the extraordinarily low P_{O_2} which man can tolerate after suitable acclimatization (Fig. 2).

Alveolar P_{O_2} has long been regarded as a measure of arterial hypoxia on high-altitude expeditions because the alveolar-arterial difference in P_{O_2} amounts to only 2–3 mm. Hg at high altitudes (Houston and Riley, 1947). According to Table I, resting arterial P_{O_2} at 25,700 ft. (7,830 m.) must have been about 30 mm. Hg, which corresponds to an oxygen saturation of about 57% if *p*H was normal (7.4), or 64% if elevated, say, to 7.5, as would be expected in the first few days at a new altitude. It seems probable that the arterial oxygen saturation would be about 60% on the summit of Everest.

Basal Metabolism

M. B. Gill measured basal metabolism by the bag method at the Silver Hut in January and again in April. No significant change from sea-level was found, although there was some decline in oxygen consumption in April, associated with loss of weight. The mean of 12 determinations on six subjects was 42 kcal./m.²/hr., with a respiratory quotient of 0.83 and a heart rate of 70 beats/min. The ventilation equivalent for oxygen averaged 15 litres ventilation S.T.P.D. per litre of oxygen absorbed, compared with 20 litres per litre of oxygen in the sitting position during the day, which is the same as at sea-level. This indicates a greater degree of hypoxia under basal conditions and might account for the prevalence of Cheyne–Stokes breathing at night.

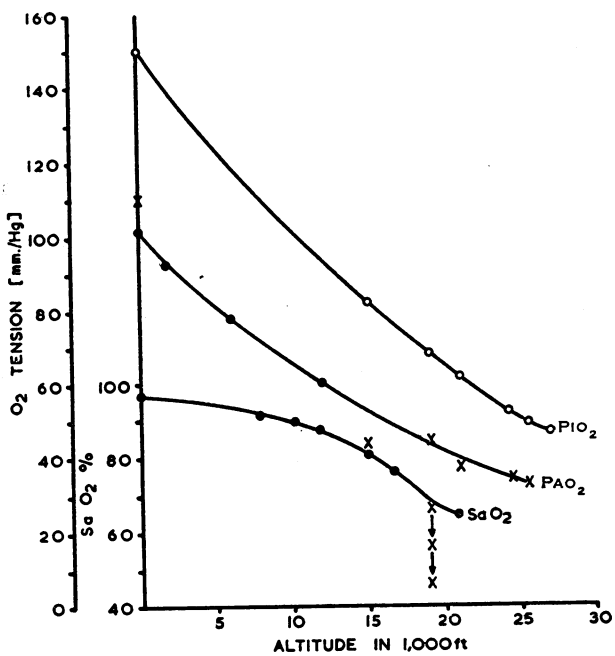


FIG. 2.—The hypoxia of altitude, showing partial pressure of oxygen in inspired air (P_{IO_2}), alveolar oxygen tension (P_{AO_2}), and arterial oxygen saturation (Sa_{O_2}). The crosses represent data collected by the 1960–61 Himalayan expedition. The black circles are published data from other sources. The arrows below the saturation curve show the effect of exercise.

Respiratory Regulation

Studies by Milledge (1962a), extending the work of Lloyd, Jukes, and Cunningham (1958) at sea-level, have greatly added to our understanding of respiratory control at high altitude. He was able to show that even after three months at 19,000 ft. (5,790 m.) the primary respiratory drive was still hypoxia, although the CO_2 mechanism had very nearly caught up with it. There was a major reduction in the response threshold to CO_2—that is, alveolar P_{CO_2} extrapolated to zero ventilation—as well as an increase in CO_2 sensitivity (increase in ventilation for each mm. Hg increase in alveolar P_{CO_2}). The former change took five to seven days for completion; the latter was a slower response, taking three to five weeks. No evidence of any change in O_2 sensitivity was found.

Muscular Exercise

Two bicycle ergometers were taken out and were used at various altitudes ranging from 15,100 to 24,400 ft. (4,600 to 7,430 m.). Control studies were carried out in London before the expedition. As on Mt. Everest (Pugh, 1958), higher levels of work intensity, oxygen intake, and ventilation were observed than in previous studies on non-mountaineers (Christensen, 1937; Houston and Riley, 1947). The data for maximum five-minute exercise are given in Table II, which shows how maximum work, maximum oxygen intake, ventilation S.T.P.D., and maximum heart rate declined with increase of altitude. Maximum work ventilation B.T.P.S., on the other hand, was higher at altitude than at sea-level, except at the highest camp. There was no significant difference between the values obtained at heights between 15,100 and 21,000 ft. (4,600 and 6,400 m.). One factor affecting ventilation at altitude is the reduced work of breathing air of low density—namely, a 20% reduction at 20,000 ft. (6.090 m.) (Cotes, 1954). The low values observed at 24,400 ft. (7,430 m.) are suggestive of respiratory muscle hypoxia, but might have been due to the subjects not pushing themselves quite so hard as at lower altitude. The data at 24,400 ft. (7,430 m.) were obtained by J. B. West and M. P. Ward.

TABLE II.—*Maximum Ventilation, Oxygen Intake, and Heart Rate During Ergometer Exercise at Various Altitudes up to 24,400 ft. (7,430 m.)*

Altitude ft.	Altitude m.	Barometric Pressure (mm. Hg)	No. of Subjects	Weight (kg.)	Ventilation (l./min.) S.T.P.D.	Ventilation (l./min.) B T.P.S.	Oxygen Intake (l./min.) S.T.P.	Oxygen Intake (ml./kg./min.)	Heart Rate (beats/min.)	Work Rate (kg.m min.)
Sea-level		750	6	72·7	97·9±18·4	119·7±22·6	3·40±0·23	46·8±3·2	192±6	1,500–1,800
15,000	4,570	440	5	68	75·0± 7·3	164·8±15·9	2·58±0·12	37·9±1·8	159±17	1,500
19,000	5,790	380	4	65·5	61·4±14·3	159·1±37·2	2·14±0·23	32·7±3·5	144±13	900–1,200
21,000	6,400	340	4	65·25	56·7± 8·6	168·8±25·4	1·95±0·11	29·6±1·7	146±11	900–1,050
24,400	7,430	300	2	67·5	35·2± 2·3	119·8± 7·7	1·40±0·09	20·7±1·3	135±8	600

S.T.P.D. = Standard temperature, pressure, dry. B.T.P.S. = Body temperature, pressure, saturated with water.

Studies of arterial oxygen saturation with the ear-oximeter by J. B. West, and S. Lahiri's direct measurements on arterialized venous blood from the heated hand, showed some remarkable changes in oxygen saturation during severe exercise at 19,000 ft. (5,790 m.) (West, Lahiri, Gill, Milledge, Pugh, and Ward, 1962). Average values were 67% at rest, 63% at a work rate of 300 kg.m./min., and 56% at a work rate of 900 kg.m./min. Values between 44 and 50% were observed on several occasions in subjects working to exhaustion in two to three minutes at 1,200 kg.m./min.

The falling arterial O_2 saturation at high work rate was associated with an increase in alveolar P_{O_2} due to relative hyperventilation, and the alveolar-arterial P_{O_2} gradient became very large—namely, 20–30 mm. Hg at 900 kg.m./min. The fall in arterial O_2 saturation and large alveolar-arterial P_{O_2} gradient were due to the limitation of O_2 absorption imposed by the diffusing capacity of the lungs. West's (1962) diffusion studies with carbon monoxide showed that the membrane component in lung diffusion did not increase with altitude. However, diffusion was assisted by the rise in alveolar P_{O_2} due to the relative hyperventilation, as well as by the increase in blood O_2 capacity, which allowed more oxygen to enter the blood from the lung for a given change in oxygen tension.

Measurements of cardiac output with acetylene showed that the cardiac output for a given work rate was the same as at sea-level, but the maximum output at 19,000 ft. (5,790 m.) was only 16–17 l./min. compared with 22–25 l./min. at sea-level. No evidence of incipient congestive heart failure was found on clinical examination of subjects working to exhaustion, nor were there any E.C.G. changes suggestive of myocardiac hypoxia. The only evidence that cardiac output had reached a maximum and was not just limited by the limitation of work capacity due to other factors was the levelling off of the heart-rate/work-rate curve, showing that the heart rate had reached a maximum (maximum heart rate at this altitude was 144 ± 13 beats/min., compared with 192 ± 6 beats/min. at sea-level).

Evidence was obtained that the oxygen debt in men working to exhaustion decreases with altitude, a finding which agrees with Edwards's (1936) blood lactate studies on the international high-altitude expedition of 1935, and it seems clear that exercise at 20.000 ft. (6,090 m.) and above is halted by other factors than at sea-level. Subjectively, the overwhelming sensation which brings work to a close is breathlessness; and very high ventilation rates of about 200 l./min. B.T.P.S., in fact values approaching the resting 15-second maximum voluntary ventilation (M.V.V. test), were sometimes observed just before the breaking point at 21,000 ft. (6,400 m.) on Mt. Everest (Pugh, 1958) and again on the 1960–61 expedition. Subjects performing the M.V.V. test on Everest complained of respiratory fatigue and could not keep up maximum ventilation much longer than the 15 seconds required by the tests; the conclusion may be drawn that exercise at great altitude is limited primarily by fatigue of the respiratory muscles, and that extreme ventilation is the result of the low arterial oxygen tension—namely, 20–30 mm. Hg at 1,200 kg.m./min.—secondary to the limitation of lung diffusion.

Effect of Oxygen

At 19,000 ft. (5,790 m.) administration of oxygen at sea-level pressure caused ventilation to fall to a level halfway between the sea-level control values and the

values at 19,000 ft. (5,790 m.) on air. Heart rates for a given work load were reduced to the sea-level control or below it, and the maximum heart rate increased. The maximum work load tolerated for five minutes was restored almost to the sea-level value, any discrepancy being attributable to muscular wasting associated with loss of body weight.

From the cardiac-output/work-rate relation and the changes in heart rate it was inferred that oxygen increased the stroke volume of the heart and restored its maximum output virtually to the sea-level control value.

The results contradict the impression created by reports by mountaineers that work capacity of acclimatized men at altitude cannot be restored to the sea-level value by breathing oxygen.

Sherpa Porters

Sherpas were superior to white men in physical performance, and carried loads up to the Silver Hut at about the same speed as climbers without loads. Only one Sherpa learnt to work satisfactorily on the bicycle ergometer at the Silver Hut. For a Sherpa he was unusually large, weighing 140 lb. (63.5 kg.); his maximum oxygen intake was 2.7 l./min., or 3 l./min. for a man of the same weight as the average climber. His maximum heart rate was 186 beats/min., which is evidence of a higher cardiac output than that of the climbers. However, the high oxygen intake cannot be explained unless the diffusing capacity of the lungs as well as the cardiac output was higher than in the climbers.

E.C.G. Studies

Milledge (1962b), extending the work of Jackson and Davis (1960) on Mt. Ama Dablam, made a large series of E.C.G. recordings. Right axis deviation was present in most subjects already at 15,000 ft. (4,570 m.), and all subjects at 19,000 ft. (5,790 m.). The changes were persistent, and were not abolished by breathing oxygen; they increased on ascending to 21,000 ft. (6,400 m.), and again at 24,400 ft. (7,430 m.).

Changes characteristic of right ventricular hypertrophy were present in the V leads, and a chest *x*-ray examination of a climber flown back to Katmandu from Makalu showed enlargement of pulmonary artery and increased convexity of the right border of the heart. Six weeks later the radiological findings were normal. These findings taken together are evidence of right ventricular hypertrophy secondary to pulmonary hypertension and resemble the changes associated with anoxia in certain forms of congenital heart disease and pulmonary disease at sea-level.

Haematology

Erythrocyte values from the work of M. P. Ward are shown in Table III. It is seen that the red cells are larger than normal but are normally filled with haemoglobin; one of the subjects (T.O.N.), who developed sprue (later confirmed by intestinal biopsy) had an M.C.V. of 110 cubic microns.

A tendency towards macrocytosis has been observed before in the 17,500–20,000 ft. (5,330–6,090 m.) range of altitude (Talbott, 1936). Reticulocytosis is a characteristic finding, even after prolonged residence at altitudes above 14,000 ft. (4,260 m.). Nucleated red cells are not present in the blood of men living at altitude, nor does the polycythaemia affect the leucocytes (Hurtado, Merino, and Delgado, 1945).

TABLE III.—*Erythrocyte Values on Men Living at* 19,000 *ft.* (5,790 *m.*)

	No. of Observations at Altitude	Observations at 19,000 ft. (5,790 m.)	Sea-level Controls
Erythrocytes (mil./c.mm.)	18	5·60±0·43	
Haemoglobin (g./100 ml.)	24	19·6 ±0·98	14·1±0·70
Haematocrit (%)	21	55·8 ±3·3	43·2±2·3
M C.V. (cubic microns) ..		102±6·0	
M C.H (γγ)		35±2·8	
M.C.H.C. (%)		35±1·7	

Haemoglobin

Haemoglobin levels on Himalayan expeditions do not as a rule reach the high levels reported on South American miners living at 17,500 ft. (5,330 m.) (Talbott and Dill, 1936; Dill, Talbott, and Consolazio, 1937) —namely, 22.9±2.6 g./100 ml.—although the altitudes visited are much higher. The mean of 51 observations on 40 subjects from five expeditions was 20.53 mg./100 ml., with a standard deviation of 1.35. Mean values reach a steady level in 40–50 days and thereafter are relatively independent of time and altitude so long as the subjects are living mainly above 18,000 ft. (5,480 ml.) (Pugh, 1954b; Pace, Meyer, and Vaughan, 1956). It was formerly taken for granted that the increase in haemoglobin and red-cell concentration on ascending to altitude was mainly due to increased formation of red cells, with some initial reduction in plasma volume accounting for the rise in haemoglobin and red-cell concentration during the first few days (Asmussen and Consolazio, 1941).

Blood-volume studies carried out on the 1960–61 expedition showed a 28% reduction in plasma volume in a group of subjects who had been one to four months at 15,100 ft. (4,600 m.), with recovery to about 18% below the sea-level control values after a further period of one to three months spent at 19,000–22,500 ft. (5,790–6,800 m.) (Table IV). Red-cell mass went on increasing during the whole period above 15,100 ft. (4,600 m.), which was evidence of a persistent increase in the rate of red-cell formation, since the survival time of red cells appears to be unaffected by altitude (Reynafarje, 1957). Blood volumes were reduced by 8.6% in the first period, but later either returned to sea-level values or increased by up to 21% above the sea-level value. These results are similar to the findings of Reynafarje (1957) and his associates at Morococha (14,900 ft.; 4,540 m.). They show that, while increased

TABLE IV.—*Mean Percentage Changes from Sea-level Values in Five Subjects After (I)* 18 *Weeks at* 13,000–19,000 *ft.* (3,960–5,790 *m.*). *(II)* 3–6 *Weeks at* 19,000 *ft.* (5,790) *m.*). *(III)* 9–14 *Weeks at or Above* 19,000 *ft.* (5,790 *m.*)

	Phase I	Phase II	Phase III
Blood volume	−8·6	−0·5	+9·7
Red-cell mass	+17·1	+32·7	+48·1
Plasma volume	−29·4	−20·3	−18·6
Haemoglobin concentration	+31·7	+36·7	+40·9

erythropoiesis lasts many months at Himalayan altitudes, the haemoglobin concentration stabilizes fairly early and is regulated to a large extent by changes in plasma volume.

The steep increase in the apparent viscosity of the blood at haematocrits above 45% (Whittaker and Winton, 1933), especially in the presence of vasoconstriction (Pappenheimer and Maes, 1942), makes it seem likely that special adaptive changes must take place in the vascular system before extreme haemoglobin levels become advantageous. Thus no correlation was observed on Mt. Everest between the haemoglobin level and climbing performance at extreme altitude (the range of haemoglobin values was 17.6–23 g./100 ml.).

The Kidney

It seemed to us that renal impairment, with delayed excretion of bicarbonate, might be a possible factor prolonging the time required for complete acclimatization, but no evidence of this was found. Dr. Ward measured the maximum and minimum *p*H of urine after ingestion of bicarbonate and ammonium chloride, maximum urine specific gravity after 24 hours of water deprivation, and water elimination after a standard water load. The results of all of these tests were normal.

Nutrition

No detailed studies of nutrition were undertaken because the subject had been investigated on two previous expeditions (Pugh, 1954a and 1954c). However, W. Romanes kept dietary records for the party at the Silver Hut over a three-day period, and analysis of these showed an average calorie intake of between 3,000 and 3,200 kcal./man/day.

All members of the wintering party noticed impairment of appetite, particularly for fatty foods, and, as time went on, a marked preference developed for highly seasoned foods and condiments. Some of us observed a tendency towards bulky and greasy stools whenever the fat intake was increased. This suggests a possible disturbance of fat absorption at great altitudes.

All members of the party lost weight at the rate of 1–3 lb. (0.45–1.36 kg.) a week at 19,000 ft. (5,790 m.), but regained some of it on descending to 13,000–15,000 ft. (3,960–4,570 m.). By the end of the expedition weight losses ranged from 14 to 20 lb. (6.4 to 9 kg.).

The diet at the Silver Hut consisted of rice, bread, butter, sugar, freeze-dried fruit, meat, and vegetables, tinned fruit, and other items, supplemented with fresh meat, potatoes, and eggs. In the spring, when a light aircraft was able to land at Ming Bo (15,100 ft.; 4,600 m.), fresh fruit and vegetables were flown in, and all members of the party developed a craving for these. Small supplies of wine and spirits also arrived at intervals and helped to make life convivial at 19,000 ft. (5,790 m.). No harmful effects were observed from taking alcohol in moderate doses at this altitude, although its action was probably greater than at sea-level.

Water Turnover

T. O. Nevison measured water turnover with deuterium oxide. At the Silver Hut, when the party were taking about one hour of physical exercise a day, the water turnover averaged 3.92 l./day, compared with 2.9 l. at sea-level. This figure may be compared with the value of 5 l./day estimated from fluid intake and urine output measurements on Mt. Cho Oyu for men doing five to seven hours' climbing a day (Pugh, 1954a). One reason for the large water requirement at altitude is the increased water loss from the lungs due to the dry air and increased ventilation. Direct measurements at 18,000 ft. (5,480 m.) on Everest showed this to be 2.9 g./100 l. of ventilation B.T.P.S. (Pugh, unpublished observations). Another factor is the liability to sweating while climbing in the sun when there is no wind.

Endocrine Studies

Twenty-four-hour urine samples were collected at 19,000 ft. (5,790 m.) by J. S. Milledge and were analysed by F. C. Grimes at the Royal South Hants Hospital, Southampton, for total neutral 17-ketosteroids, 17-ketogenic steroids, sodium, and potassium. The results showed a significant reduction of 37% and 34% respectively for total neutral 17-ketosteroids and 17-ketogenic steroids, and an 88% increase in Na/K ratio. Twenty-four-hour urine volume increased by 45%, which was also statistically significant ($P<0.05$). These findings suggest a depression of adrenal cortical and gonadal function and possibly also of antidiuretic hormone secretion, but more work is needed for confirmation. A rise in salivary Na/K ratio suggestive of a reduction in electrolyte-active steroids has been previously reported from a Himalayan expedition by Williams (1961).

Ward observed the disappearance of the circulating eosinophils in himself and Gill after a long mountain climb, showing that the adrenal cortex was still capable of responding to stress, even if the functional level in non-stress conditions was depressed.

Acclimatization

Visitors to Ming Bo (15,100 ft.; 4,600 m.) by helicopter or light aircraft who stayed only one to two hours had no altitude symptoms, although they were very cyanosed. Seven out of eight who spent a night or longer suffered from mountain sickness, with malaise, headache, lassitude, irritability, vomiting, effort dyspnoea, and irregular breathing in varying degrees and combinations and lasting one to three days. Three climbers who spent the winter at sea-level and returned by aircraft after an absence of three months found they had lost their tolerance of altitude and were mountain-sick at Ming Bo, but they recovered quickly. A climber who was flown out to Katmandu on account of illness found he had not lost his acclimatization on returning two weeks later.

Those who travelled on foot, taking 18 days to reach Ming Bo (15,100 ft.; 4,600 m.), were singularly free of symptoms there, but could not stay overnight at the Silver Hut without rather severe symptoms until they had been at Ming Bo two weeks or more. At the Silver Hut at 19,000 ft. (5,790 m.) most of the party felt reasonably well and energetic. Card sorting and other tests, devised for us by the Medical Research Council Unit for Applied Psychology at Cambridge and carried out by M. B. Gill, were negative except for a slight increase in mistakes in the last series, conducted in April; nor was there any impairment in visual acuity or restriction of visual fields; hearing, as tested with an audiometer, was also normal. In spite of these negative findings work was more tiring and called for greater effort than at sea-level, even though skill and accuracy

were unaffected. We did about six hours' physiological work a day, sometimes much more, and spent one to two hours ski-ing, and 10 to 12 hours in bed at night. Some of the party remained for periods of up to nine weeks at the Silver Hut without a break ; two were less tolerant and had to go down at intervals for short rests at 15,100 ft. (4,600 m.)—one every four weeks, the other every seven days.

Although there was little objective evidence of deterioration apart from loss of weight, the wintering party were mentally and physically tired when the expedition moved to Makalu, and certainly had no advantage in physical performance over the party of fresh climbers after partial acclimatization.

The impression was that 19,000 ft. (5,790 m.) was too high for complete adjustment and that 17,000 ft. (5,330 m.), or in some cases 15,000 ft. (4,570 m.), would be nearer the limit for plainsmen living at altitude. The native miners studied by the International Physiological Expedition to the Andes in 1935 (Dill, 1938) lived at 17,500 ft. (5,330 m.) and climbed every day to their work at 19,000 ft. (5,790 m.). They refused to occupy a camp built for them at 19,000 ft. (5,790 m.) longer than six weeks, the reasons given being loss of sleep and inability to enjoy their food.

With regard to preliminary acclimatization in preparation for an ascent of one of the 28,000-ft. (8,530-m.) peaks without supplementary oxygen, a winter spent training at heights between 12,000 and 15,000 ft. (3,650 and 4,570 m.), where there is little impairment of exercise tolerance or appetite, would be preferable to a prolonged stay at 18,000–20,000 ft. (5,480–6,090 m.), where the restriction of effort is such that it is not feasible to take more than a few hours' exercise a day over a long period and the intensity of exercise is greatly reduced. This reduction of work capacity combined with loss of weight results in a general impairment of physical condition which more than outweighs any apparent gain in acclimatization. Although there was some evidence from Milledge's work that respiratory adaptation was still going on when we left, it seems certain that our stay at 19,000 ft. (5,790 m.) could not have been prolonged indefinitely, if only for nutritional reasons.

Sickness

The usual gastro-intestinal and upper respiratory infections were seen early in the expedition and broke out sporadically throughout, newcomers being mainly affected. There was one case of sprue, later confirmed by biopsy ; a similar case occurred on Mt. Cho Oyu in 1952. As on other expeditions, piles were a common complaint, and there was one case of ischio-rectal abscess. Two cases of pneumonia and two cases of acute pulmonary oedema occurred, all of which recovered. Hillary, who had been living at lower altitudes most of the time from the end of November to mid-April, and was perhaps insufficiently acclimatized, became ill at Camp 2 (19,000 ft. ; 5,790 m.) on Makalu. The symptoms, which consisted of aphasia and right-sided facial palsy, preceded by headache, cleared up after three days at lower altitude, but he was advised not to go above 15,000 ft. (4,570 m.) again, as it seemed likely that he had had a mild cerebral thrombosis.

The withdrawal of the expedition from Makalu was the result of a chain of accidents set off by P. Mulgrew's illness at 27,400 ft. (8,350 m.). Mulgrew was suddenly stricken with severe right-sided chest pain, dyspnoea, and weakness. With Nevison's assistance, he managed to get down to the camp at 25,800 ft. (7,860 m.), crawling much of the way, and there spent the night. His evacuation from the mountain took five days, and he was eventually flown out by helicopter to Katmandu. At some stage on the mountain he suffered severe frost-bite of his hands and feet. His chest condition, which was diagnosed as a primary pulmonary thrombosis with infarction, went on to abscess formation and empyema. He eventually lost the terminal digits of the fingers of his right hand, and bilateral below-knee amputation was performed after his return to New Zealand, because of intercurrent infection.

Ward, who had taken over the leadership on the mountain when Hillary fell ill, developed pneumonia at the camp at 24,400 ft. (7,430 m.), but recovered sufficiently on oxygen and antibiotics to descend without assistance to the base camp.

Oxygen Equipment

The risks of attempting to climb a 28,000-ft. (8,530-m.) peak without supplementary oxygen were clearly foreseen, and four open-circuit climbing sets and 75,000 l. of oxygen were provided. Oxygen was used for medical and experimental purposes, and the climbing sets were taken to the camp at 24,400 ft. (7,430 m.) on Makalu. Unfortunately, when the accident to Mulgrew occurred not enough oxygen cylinders had been brought up for a rescue operation, and there was scarcely enough for medical treatment.

These happenings dramatically illustrate the extremely dangerous situation which may develop on a 28,000-ft. (8,530-m.) mountain when anything goes wrong, and show that it is not sufficient to have oxygen equipment in reserve unless it is available in adequate quantities at points where it may be needed.

Summary

The expedition spent eight months at heights of over 15,000 ft. (4,570 m.). Physiological investigations were conducted over a period of five months in a prefabricated laboratory situated at 19,000 ft. (5,790 m.) (bar. 380 mm. Hg) during an attempted ascent of Mt. Makalu (27,790 ft. ; 8,470 m.).

Data are reported on basal metabolism, muscular exercise, respiratory regulation, blood volume, haemoglobin and erythrocytes, electrocardiographic changes, nutrition, and endocrine and renal function. The exercise results include data on lung diffusion, cardiac output, and arterial O_2 saturation at 19,000 ft. (5,790 m.) ; and on O_2 intake, ventilation, and heart rate at heights up to 24,400 ft. (7,430 m.) (bar. 300 mm. Hg). At 24,400 ft. (7,430 m.) the maximum O_2 intake was found to be 1.4 l./min., ventilation B.T.P.S. 119 l./min. and heart rate 135 beats/min. Haldane end-expiratory gas samples taken at rest at 25,700 ft. (7,830 m.) (bar. 288 mm. Hg) had an average O_2 tension of 33 mm. Hg and CO_2 tension of 14 mm. Hg. Arterial O_2 saturations of less than 50% were observed during periods of two to three minutes' maximum exercise at 19,000 ft. (5,790 m.), the average resting value being 67%.

The party appeared to acclimatize well to 19,000 ft. (5,790 m.), and card-sorting and other psychological tests revealed no evidence of mental impairment. However, all members of the party continued to lose weight, and this makes it doubtful if they could have stayed there

indefinitely. Newcomers on Mt. Makalu, after four to six weeks' acclimatization, were, if anything, fitter and more active than men who had wintered at 19,000 ft. (5,790 m.).

Medical aspects of the expedition are described. On Mt. Makalu cases occurred of cerebral thrombosis, pulmonary infarction, acute pulmonary oedema, pneumonia, and frostbite. The ascent was made without oxygen equipment, but oxygen was available for medical treatment.

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"As a result of representations made by several members of the public to the Committee for Research on Apparatus for the Disabled a Working Party on Car Modifications was set up. This working party proceeded to examine the problems of the disabled, in the first instance, as far as private cars were concerned. It seemed clear that there was a considerable number of disabled people who were virtually home-bound because they could not, by themselves, get into or out of a car, or who, if they could do this, could not bring their wheelchair in with them, thus rendering themselves immobile at the end of their journey. There were men and women unable to obtain gainful employment because of this as well as housewives unable to shop, to fetch their children to and from school and to lead normal social lives. The Working Party is therefore developing an adapted chassis and body for a motor vehicle which, it is hoped, will enable a wheelchair-user to enter the car and to drive it in his wheelchair. Work on this is proceeding very satisfactorily, although it is still in its early stages." (*Ninth Annual Report*, National Fund for Research into Poliomyelitis and Other Crippling Diseases.)

EFFECT OF VARIOUS MODES OF OXYGEN ADMINISTRATION ON THE ARTERIAL GAS VALUES IN PATIENTS WITH RESPIRATORY ACIDOSIS*

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In subjects with obstructive emphysema and hypercapnia the ventilatory response to carbon dioxide is diminished. This is also true of normal patients made hypercapneic by placing them in an environment rich in carbon dioxide. In both groups hypoxia and a low *p*H remain as the main ventilatory stimuli. The removal of the stimulatory effect of hypoxia by administering oxygen is well known to worsen alveolar ventilation and increase carbon-dioxide retention. In spite of this, the need for oxygen in the treatment of acute respiratory acidosis is accepted by most physicians. However, there is disagreement on the mode of oxygen administration. Thus some (Barach *et al.*, 1952; Wilson *et al.*, 1954; Barach, 1961) feel that continuous administration of oxygen using low flow rates with graded increases will help avoid serious worsening of hypercapnia while maintaining acceptable oxygen tensions. Others (Cohn *et al.*, 1954; Harbord and Woolmer, 1958) suggest that giving oxygen intermittently each hour will lessen the hazards of further alveolar hypoventilation and yet increase the oxygen tension of the blood.

It is the purpose of this paper to report the effects of oxygen administration by various means in patients with obstructive emphysema and respiratory acidosis.

Methods and Materials

A total of 15 adult male patients were studied. Requirements for inclusion in the study were clinical and pulmonary function evidence of obstructive emphysema with carbon-dioxide retention at the time of the study. All patients were in a fasting state, and all procedures were begun in the early part of the day. A Cournand needle was placed in the brachial artery, and two baseline samples were drawn 15 minutes apart while the patient was breathing room air.

The patients were separated into three groups in a random fashion. The groups were comparable by degree of respiratory acidosis. Group A consisted of four patients; these received pure oxygen by mask for 10 minutes. Group B comprised six subjects receiving oxygen by nasal catheter for 10 minutes at flow rates

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