

Maximal exercise at extreme altitudes on Mount Everest

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WEST, J. B., S. J. BOYER, D. J. GRABER, P. H. HACKETT, K. H. MARET, J. S. MILLEDGE, R. M. PETERS JR., C. J. PIZZO, M. SAMAJA, F. H. SARNQUIST, R. B. SCHOENE, AND R. M. WINSLOW. *Maximal exercise at extreme altitudes on Mount Everest*. *J. Appl. Physiol.: Respirat. Environ. Exercise Physiol.* 55(3): 688-698, 1983.—Maximal exercise at extreme altitudes was studied during the course of the American Medical Research Expedition to Everest. Measurements were carried out at sea level [inspired O₂ partial pressure (PO₂) 147 Torr], 6,300 m during air breathing (inspired PO₂ 64 Torr), 6,300 m during 16% O₂ breathing (inspired PO₂ 49 Torr), and 6,300 m during 14% O₂ breathing (inspired PO₂ 43 Torr). The last PO₂ is equivalent to that on the summit of Mt. Everest. All the 6,300 m studies were carried out in a warm well-equipped laboratory on well-acclimatized subjects. Maximal O₂ uptake fell dramatically as the inspired PO₂ was reduced to very low levels. However, two subjects were able to reach an O₂ uptake of 1 l/min at the lowest inspired PO₂. Arterial O₂ saturations fell markedly and alveolar-arterial PO₂ differences increased as the work rate was raised at high altitude, indicating diffusion limitation of O₂ transfer. Maximal exercise ventilations exceeded 200 l/min at 6,300 m during air breathing but fell considerably at the lowest values of inspired PO₂. Alveolar CO₂ partial pressure was reduced to 7-8 Torr in one subject at the lowest inspired PO₂, and the same value was obtained from alveolar gas samples taken by him at rest on the summit. The results help to explain how man can reach the highest point on earth while breathing ambient air.

hypoxemia; diffusion impairment; hyperventilation; respiratory alkalosis; oxygen uptake

THE SEVERE HYPOXIA of extreme altitudes such as the summit of Mt. Everest puts resting man near his limit of tolerance. When O₂ transfer is additionally stressed by exercise, pulmonary gas exchange becomes even more critical. The O₂ partial pressure (PO₂) of inspired air on the summit is only 43 Torr (26), resulting in a resting arterial PO₂ of less than 30 Torr (24). Although climbers have reached the summit breathing ambient air (12, 15), their accounts clearly show how close they are to the limit of human performance.

Available data on maximal O₂ uptake at somewhat lower altitudes (21) suggest that almost all the O₂ that is available on the summit will be required for basal metabolism. Theoretical analyses (8, 27) suggest that the maximal O₂ uptake of a climber on the summit could not exceed about 550-700 ml/min. It is questionable whether man could climb to the summit with such a severe

restriction. Those studies identified several critical gaps in our knowledge which the present investigation was designed to fill.

This paper reports exercise studies carried out during the course of the American Medical Research Expedition to Everest, which took place in the fall of 1981. A sophisticated laboratory was set up at an altitude of 6,300 m, barometric pressure 351 Torr. Subjects were studied both while breathing ambient air and while inhaling low O₂ mixtures containing 16 and 14% O₂. These mixtures gave virtually the same inspired PO₂ as that of climbers at altitudes of 8,050 m (barometric pressure 283 Torr) and at the summit of Mt. Everest, altitude 8,848 m (barometric pressure 253 Torr). The study elucidates how it is possible for man to reach an O₂ uptake of 1 l/min at the highest point on earth.

METHODS

Subjects

There were 15 members of the expedition, and anthropometric data are given in Table 1. Note that two weights are given. The first is for the base-line studies carried out at sea level in La Jolla in May 1981 prior to the expedition, and the second is the weight during the measurements at 6,300 m in October of the same year. Body weight usually falls at these high altitudes due to a combination of loss of appetite, impaired absorption, indifferent food, and possibly some dehydration. The subjects had been at altitudes of 5,400 m (barometric pressure 400 Torr) or higher for about 5 wk before the 6,300-m measurements were made.

Many of the subjects were unusually athletic. For example, of the six subjects (*SB*, *CP*, *RS*, *FS*, *DG*, and *PH*) who took part in the studies at 6,300 m while breathing 16% O₂, the first four were competitive marathon runners. This group also included both of the subjects (*CP* and *RS*) who took part in the studies while breathing 14% O₂. In addition two other subjects shown in Table 1 regularly competed in long-distance running. Two of the subjects (*JM* and *JW*) were not particularly athletic, but they had previously participated in studies of maximal work capacity at altitudes up to 6,400 and 7,440 m during the Himalayan Scientific and Mountaineering Expedition 1960-1961 (21). All subjects gave informed consent for the procedures.

TABLE 1. *Anthropometric data*

Subj	Age, yr	Height, cm	Weight, kg	
			Sea level	6,300 m
FB	47	188	92.8	77.5
SB	34	175	67.6	66.0
JE	42	179	85.5	81.9
DG	30	193	85.8	79.5
PH	33	179	67.8	59.3
DJ	33	179	72.1	65.3
CK	33	179	78.6	74.2
JM	51	175	69.2	64.2
RP	25	183	63.6	63.2
CP	31	183	70.8	65.9
GP	33	171	73.0	56.0
FS	38	169	67.2	65.4
RS	34	179	77.2	74.5
MW	31	175	72.0	71.0
JW	52	183	78.8	70.1

Sea-level measurements were made at La Jolla in May 1981; 6,300-m measurements in October 1981.

Exercise Studies

The exercise studies were carried out in a comfortable laboratory (Weatherport, Gunnison, CO) constructed of fiberglass blankets covering an aluminum frame. The dimensions were 2.4 m wide by 4.6 m long by 2.1 m high, and there was a plywood floor. The laboratory was kept warm (10–20°C) with a propane heater and provided with electrical power and light from a bank of lead acid batteries charged by a gasoline generator and solar panels; 110 V AC were obtained from inverters. A brief description of the instrumentation has been published elsewhere (14).

The subjects exercised on a portable bicycle ergometer identical in design to that used on the 1960–1961 expedition (21, 23). The subjects pedaled at 50 rpm up to a work level of 900 kg·m·min⁻¹ but at 75 rpm at higher work levels. The timing was provided by a metronome which was checked before each run. After an initial warm-up period of at least 10 min at 300 kg·m·min⁻¹ the required work rate was selected by adjusting the load on the bicycle brake band. The load was continually checked by one observer during each run. The required work rate was maintained for 5 min at loads up to 900 kg·m·min⁻¹ at sea level and 3 min at higher work rates. For the studies at 6,300 m during ambient air breathing, the duration was 3 min for work levels above 600 kg·m·min⁻¹ and for the studies in which low O₂ mixtures were inhaled, all the work durations were 3 min.

The valve box (Koegel, San Antonio, TX) had a low resistance and the circuit was made of plastic tubing of 3.8-cm bore. During the last minute (2 min for the 300 kg·m·min⁻¹ load) expired gas was collected in a meteorological balloon and analyzed within a few minutes using O₂ and CO₂ meters (Beckman Instruments, models OM11 and LG2, respectively). The analyzers were calibrated before and after each gas analysis by sampling from tanks of calibrating gases. The outputs of the O₂ and CO₂ analyzers were displayed on a strip-chart recorder (Gould model 222). Following the gas analysis, the remainder of the gas was passed through a dry gas meter (Singer, Warren Collins) for the measurement of

volume. Allowance was made for the small amount of gas which passed through the analyzers.

During exercise the electrocardiogram (Lumiscop) was continuously recorded on one channel of a strip-chart recorder so that heart rate could be determined. The leads were placed in the CM5 configuration. Arterial O₂ saturation was continuously measured by ear oximeter (Hewlett Packard) and displayed on another channel of a strip-chart recorder. The oximeter output was directly calibrated against arterial blood in three subjects (JE, JM, and JW); six samples of blood were taken, three while the subjects breathed a low O₂ concentration, but one of the samples was spilled prior to analysis. O₂ saturation was measured by a new technique in which the PO₂ of the blood was observed when its saturation was altered by adding potassium ferricyanate (Winslow and Samaja; unpublished observations). Figure 1 shows the results of the oximeter calibration done at 6,300 m.

Exercise studies were also carried out with some of the subjects breathing low O₂ concentrations in the laboratory at 6,300 m. In the first series (6 subj), the inspired O₂ concentration was 16.0%. This gave an inspired PO₂ of 48.5 Torr to match that existing at Camp 5 just above the South Col of Mt. Everest (alt 8,050 m, barometric pressure 280 Torr).¹ Two subjects performed maximal exercise breathing 14.0% O₂ which reduced the inspired PO₂ to 42.5 Torr. This was designed to be equivalent to the inspired PO₂ on the summit of Mt. Everest (alt 8,848 m, barometric pressure 250 Torr).² The low O₂ mixtures were prepared by having subjects exercise at a moderate rate on the bicycle ergometer and then collecting the expired gas in large balloons after passing it through a CO₂ absorber. The absence of CO₂ was checked with the CO₂ analyzer. The O₂ concentration was titrated to the correct level by rebreathing some of the gas once more, or diluting it with ambient air.

Barometric pressure was measured with a crystal sensor barometer checked against a mercury barometer kept at Base Camp (26). Venous blood samples were taken in three subjects before and after maximal exercise at 6300 m (air breathing) for measurements of lactate, hemoglobin, and hematocrit. The samples were removed from an arm vein 7 min after the end of exercise.

Ambulatory Measurements

Measurement of exercise ventilation and heart rate were made in two subjects while they were climbing at their maximum rate en route to the summit from the top camp. They breathed ambient air for these studies though for the rest of the climb they were breathing from an O₂ mask (inspired PO₂ about 70 Torr). Ventilation was measured by having the subject breathe through a turbine flowmeter of low resistance (Boehringer Laboratories). Rotation of the turbine interrupted a light beam, and the resulting pulses were recorded on one channel of a slow-running tape recorder (Medilog, Oxford Instruments). A second channel recorded tempera-

¹ We subsequently found that the mean barometric pressure at Camp 5 was slightly higher at 283.6 Torr (26).

² Again, the actual pressure on the summit was a little higher at 253.0 Torr (26).

ture from a thermistor in the flow meter. Electrocardiogram recordings were made continuously from electrodes in the CM5 configuration and recorded on another channel. The fourth channel had timing pulses and also encoded information from an event marker, which the climber operated to indicate what he was doing. He also had a miniature dictating tape recorder to keep a log of his activities.

RESULTS

Table 2 shows the means of the results of all the exercise measurements. The table is divided into four sections. The first shows the studies done at sea level at the University of California, San Diego, in May, 1981.

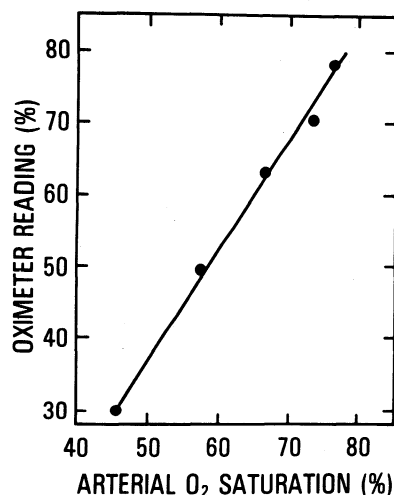


FIG. 1. Calibration of ear oximeter at 6,300 m. Five blood samples were taken from 3 subjects, 2 of whom breathed both air and a low O_2 mixture.

The second study was carried out in the Camp 2 laboratory in October during the expedition. The third and fourth sets of measurements were done in the same laboratory but with the subject inhaling 16 and 14% O_2 mixtures, respectively.

Work Rate

Sea level. Table 2 shows that seven of the subjects (SB, JE, DG, CK, CP, FS, and RS) were able to work for 3 min at the rate of 2,100 $kg \cdot m \cdot min^{-1}$. Actually one other subject (MW) also completed 3 min at this work level, but an error in the gas collection invalidated the results. Five subjects (SB, DG, CK, CP, and RS) were able to work at the rate of 2,400 $kg \cdot m \cdot min^{-1}$, though only one completed the 3 min, the others being exhausted after 2 min.

These work levels are relatively high, particularly as most of the subjects had not trained on a bicycle ergometer. Although the values are not as high as those found in some trained athletes (2, 18), they are substantially higher than those reported by Pugh et al. (21) in a previous study at extreme altitudes with which our study has many similarities. In that study only three subjects could work at sea level at a rate exceeding 1,500 $kg \cdot m \cdot min^{-1}$ (the values were in the range 1,800–2,000 $kg \cdot m \cdot min^{-1}$) and the highest measured O_2 uptake was 3.93 l/min as opposed to 4.63 l/min in the present study.

6,300 m altitude. Eight subjects (SB, JE, DG, CK, CP, RP, RS, and MW) were able to work at a rate of 1,200 $kg \cdot m \cdot min^{-1}$ for 3 min while breathing ambient air. When a gas mixture containing 16% O_2 was inhaled at that altitude, all six subjects in that study (SB, DG, PH, CP, FS, and RS) were able to work at 450 $kg \cdot m \cdot min^{-1}$ and three subjects (SB, DG, and CP) completed 3 min at 600 $kg \cdot m \cdot min^{-1}$. The maximum work rate for 3 min while

TABLE 2. Summary of exercise results

Altitude, PB, Insp O_2	n	Work Rate, $kg \cdot m \cdot min^{-1}$	$\dot{V}O_2$, l/min	Ventilation, l/min		Resp Fre- quency, breaths/ min	FE_{O_2} , %	FE_{CO_2} , %	R	O_2 Saturation*		Heart Rate, beats/min
				STPD	BTPS					Start	End	
Sea level	14	300	0.99	20.0	24.8	17	16.15	4.15	0.85	97	97	95
(750 Torr),	14	600	1.52	29.1	37.2	18	15.78	4.45	0.84	97	97	109
$PI_{O_2} = 147.1$	14	900	2.14	41.5	50.9	23	15.87	4.53	0.88	98	97	125
	14	1,500	3.24	71.2	87.4	31	16.32	4.46	0.98	97	97	155
	11	1,800	3.83	101.7	123.8	41	16.96	4.09	1.06	97	97	172
	7	2,100	4.37	107.7	132.0	38	16.75	4.41	1.07	97	95	174
	5	2,400	4.63	130.7	160.2	52	17.29	3.82	1.06	95	95	178
6,300 m	14	300	0.79	19.1	54.1	30	16.77	3.89	0.92	70	69	108
(351 Torr),	14	600	1.34	32.9	93.2	43	16.83	4.10	1.00	67	66	127
$PI_{O_2} = 63.7$	12	900	1.90	53.3	151.0	49	17.17	4.19	1.15	64	64	134
	8	1,200	2.31	73.1	207.2	62	17.52	4.10	1.27	62	59	146
6,300 m,	6	300	0.84	29.2	83.1	38	12.91	3.61	1.28	58	56	121
$FI_{O_2} = 0.16$,	6	450	1.21	43.1	122.5	51	13.06	3.34	1.18	53	53	127
$PI_{O_2} = 48.5$	6	600	1.45	55.8	158.8	63	13.15	3.53	1.35	55	55	128
6,300 m,	2	150	0.69	27.6	78.4	69	11.40	3.16	1.24	61	58	114
$FI_{O_2} = 0.14$,	2	300	1.07	47.8	135.9	77	11.59	2.98	1.26	52	54	135
$PI_{O_2} = 42.5$	2	450	0.99	57.0	161.9	80	11.59	2.91	1.62	51	46	129

n, No. of subjects. $\dot{V}O_2$, O_2 uptake; FE_{O_2} , expired O_2 fraction; FE_{CO_2} , expired CO_2 fraction; R, respiratory exchange ratio; PI_{O_2} , partial pressure of inspired O_2 ; FI_{O_2} , inspired O_2 fraction. * Measured in the first and last minute of exercise.

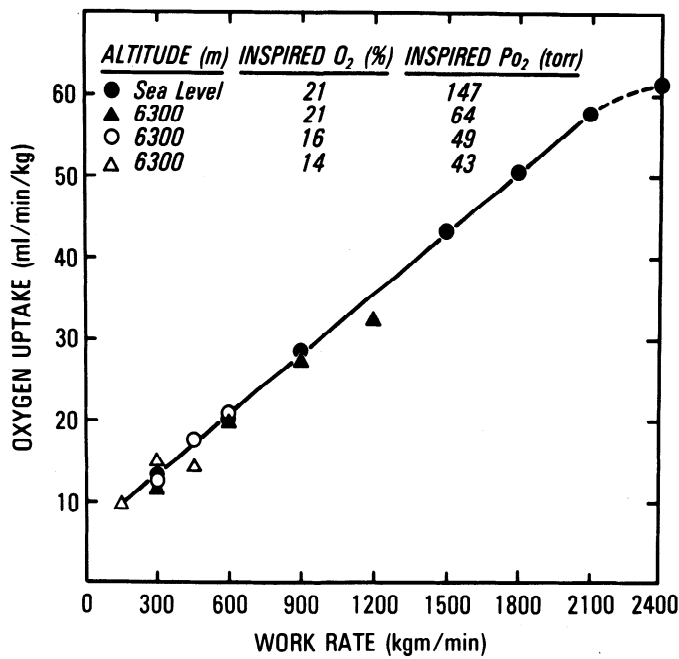


FIG. 2. O₂ uptake (ml·min⁻¹·kg body wt⁻¹) against work rate. Points are mean values. Regression line was drawn through sea-level data. Open triangles (6,300 m, 14% O₂) represent inspired P_{O₂} on summit of Mt. Everest.

breathing 14% O₂ was 450 kg·m·min⁻¹, accomplished by two subjects (CP and RS). One of these subjects (RS) exercised at 600 kg·m for 2 min but the O₂ uptake was less than at 450 kg·m·min⁻¹. Note that Table 7 shows all the data for the two subjects at 6,300 m, breathing 14% O₂.

O₂ Uptake

Sea Level. Table 2 shows that the mean O₂ uptake (STPD) increased from about 1 l/min at a work rate of 300 kg·m·min⁻¹ to 4.63 l/min for the highest work load of 2,400 kg·m·min⁻¹ at sea level. In Fig. 2 the data have been expressed as ml·min⁻¹·kg body wt⁻¹ and show a tight linear correlation between O₂ uptake and work rate up to a work rate of 2,100 kg·m·min⁻¹ where the O₂ uptake was 57.7 l·min⁻¹·kg⁻¹. However, for the highest work rate of 2,400 kg·m·min⁻¹ the O₂ uptake falls below the straight line, indicating increased dependence on anaerobic metabolism. In general the relationship between O₂ uptake and work level was very similar to that observed by Pugh et al. (21) and other previous investigators.

6,300 m altitude, air breathing. It can be seen from Table 2 that at 6,300 m the measured O₂ uptakes (expressed as l/min) fell slightly below the values obtained at sea level for the same work rate. However, as Table 1 shows, body weights at 6,300 m were lower than at sea level. Figure 2 shows that if O₂ consumption is calculated in terms of l·min⁻¹·kg body wt⁻¹, the relationship between O₂ uptake and work rate for sea level and 6,300-m altitude became very close. Note that at the highest work level (1,200 kg·m·min⁻¹), the O₂ uptake departed from linearity.

The physiological significance of the agreement of the

O₂ uptakes expressed as ml·min⁻¹·kg⁻¹ is arguable. Certainly some of the weight loss at high altitude is the result of muscle wasting as evidenced by a marked reduction of girth of arms and legs. However, much of the weight loss is fat. Therefore it is questionable whether it is justifiable to attribute all of the lowering of maximal O₂ uptake to loss of muscle mass.

6,300 m altitude, low O₂ breathing. Figure 2 shows that the two studies in which low O₂ mixtures were inhaled generally showed the same relationship between O₂ uptake (ml·min⁻¹·kg⁻¹) and work rate as was observed at sea level. However, the 300-kg·m·min⁻¹ point for the 14% O₂ study shows an exceptionally high O₂ uptake. A possible reason for this is the very high value of ventilation (136 l/min) and the associated O₂ cost of breathing. This high value could certainly significantly raise O₂ consumption (16, 22).

Note that when the subjects were breathing 14% O₂, the work rate of 450 kg·m·min⁻¹ produced an O₂ uptake no greater than that measured during the work rate of 300 kg·m·min⁻¹. Clearly maximal aerobic power had been reached at the lower work level and the subjects were merely incurring a larger O₂ debt at the higher level. One subject (RS) actually worked at a rate of 600 kg·m·min⁻¹ for 2 min but the measured O₂ uptake was only 0.84 l/min and the respiratory exchange ratio (R) value was 1.84 so this measurement was omitted from Table 2.

Our finding that O₂ consumption for a given work rate is nearly independent of inspired P_{O₂} confirms conclusions reached by earlier workers (6, 10, 13, 21) that the efficiency of muscular exercise is essentially independent of altitude. However, our studies extend the range of observations for maximal exercise to higher equivalent altitudes including the Everest summit.

Ventilation

When ventilation was corrected to STPD the values at 6,300 m were generally higher than those at sea level except at the lowest work rate of 300 kg·m·min⁻¹. Considerable increases were seen in the studies in which low O₂ mixtures were inhaled. Note, however, that the conversion of volumes to STPD at this altitude gives values that are different from those which would be obtained if the subjects were breathing 21% O₂ at the equivalent higher altitude (8,848 m, barometric pressure 250 Torr for 14% O₂ breathing).

Ventilation expressed as BTPS increased dramatically at the same work rate as the inspired P_{O₂} was reduced. Note the extremely high level of 207 l/min recorded for a work rate of 1,200 kg·m·min⁻¹ at 6,300 m when the subjects were breathing ambient air. These enormous levels of ventilations are helped by the fact that maximal voluntary ventilation increases at low barometric pressures because of the reduced work of breathing air of low density (7). The values of maximal exercise ventilation are discussed further below.

Expired Gas Composition

Table 2 shows that the O₂ concentration of mixed expired gas generally increased as the work rate was

raised at any given inspired PO_2 . However, there was no consistent change in the concentration of CO_2 in mixed expired gas as the work level increased at any given inspired PO_2 , though it apparently fell at the highest work level at sea level. On the other hand there was a downward trend for expired CO_2 concentration with increasing work rate for both subjects in the measurements made during 14% O_2 breathing.

Respiratory Exchange Ratio

The values of R generally increased as work rate was raised at a particular inspired PO_2 . In addition, if a given work level is considered, the values of R increased as the inspired PO_2 was reduced.

Note the remarkably high values of R at the low work levels when 16 and 14% O_2 mixtures were inhaled. For example, when the inspired O_2 was 16% and the work rate was only 300 $kg \cdot m \cdot min^{-1}$, the value of R was 1.28 in spite of the fact that the six subjects could achieve double the work rate and almost double the O_2 uptake. In an effort to reduce the level of R to values more comparable with steady-state exercise, we arranged to have the subjects prebreathe low O_2 mixtures for increasing periods of time up to 5 min. However, values of R exceeding 1.0 were always seen. We were limited in the amount of time available for prebreathing low O_2 mixtures because of the very large amounts of gas that had to be prepared. For example, note the extremely high ventilation (BTPS) of 78 l/min for the lowest work level of 150 $kg \cdot m \cdot min$ when 14% O_2 was inspired.

Alveolar Gas Partial Pressures

The O_2 and CO_2 concentrations in mixed expired gas can be used to calculate the alveolar partial pressures if we assume values for the dead space-to-tidal volume ratio. This procedure can be justified during exercise when the dead space-to-tidal volume ratio lies within the range 0.2–0.1 (1), because it can be shown that the resulting values are relatively insensitive to changes of the dead space-to-tidal volume ratio within this range. For example, in the measurements made at an altitude of 6,300 m during air or low O_2 breathing, changing the dead space-to-tidal volume ratio from 0.2 to 0.1 alters the calculated alveolar CO_2 partial pressure (PCO_2) by usually less than 1 Torr.

Table 3 shows the values for alveolar PO_2 and PCO_2 calculated in this way. Only the values for minimal and maximal work rates at any given altitude are shown. In the case of the measurements during 14% O_2 breathing at 6,300 m altitude, only the values obtained on one subject (CP) are given. This is because of the marked differences between the two subjects (see Table 7) and the fact that the resting alveolar gas data on the summit were only obtained on this one subject. Note that the alveolar PO_2 fell from 119 Torr during maximal exercise at sea level to a value of 37 Torr during maximal exercise when the inspired PO_2 was 42.5 Torr. The corresponding values of alveolar PCO_2 were 30 and 8 Torr.

Alveolar gas concentrations were also measured directly at altitudes of 8,050 and 8,848 m during the expedition using a special automatic alveolar gas sampler

TABLE 3. Alveolar PO_2 and PCO_2 during exercise

Site Inspired Gas	Work Rate, $kg \cdot m \cdot min^{-1}$	Tidal Volume, liters	Calculated Alveolar PO_2 , Torr	Calculated Alveolar PCO_2 , Torr	Alveolar Gas at Rest at Equivalent Altitude*	
					PO_2 , Torr	PCO_2 , Torr
Sea level, ambient air	300	1.46	105‡	37		
	2,400	3.08	119§	30		
6,300 m, ambient air	300	1.87	48‡	15		
	1,200	3.34	52§	14		
6,300 m, 16% O_2	300	2.19	38§	12	36	11
	600	2.52	39§	12		
6,300 m, † 14% O_2	150	0.94	35‡	9	38	7.5
	450	1.80	37‡	8		

Alveolar O_2 and CO_2 partial pressures (PO_2 and PCO_2 , respectively) were calculated from mixed expired gas and compared with directly measured values at rest. * From Ref. 24. † Values from subject CP only because of the marked differences between the 2 subjects (see Table 7) and the fact that the directly measured values are also from subject CP only. ‡ Dead space-to-tidal volume ratio assumed 0.2. § Dead space-to-tidal volume ratio assumed 0.1.

(24). The measurements were made during rest and the results are shown in the two right-hand columns of Table 3. Recall that the inspired PO_2 of 48.5 Torr measured during 16% O_2 breathing at 6,300 m was virtually the same inspired PO_2 as at an altitude of 8,050 m. Similarly the measurements made during 14% O_2 breathing at 6,300 m had an inspired PO_2 almost the same as that on the Everest summit, altitude 8,848 m. It can be seen that agreement for the equivalent altitude of 8,050 m was good with the PCO_2 , being within 1 Torr, while the exercise value of PO_2 was 2–3 Torr higher, consistent with the higher value of R. Agreement was also good for the equivalent altitude of 8,848 m (summit), the PCO_2 values being within 1 Torr. The resting alveolar PO_2 was actually slightly higher than the calculated exercise value, but the calculated R from the alveolar gas samples was very high (1.49) for unexplained reasons (24).

Arterial O_2 Saturation

Table 2 and Fig. 3 show the arterial O_2 saturations as measured by ear oximeter. The oximeter was calibrated directly against arterial blood at 6,300 m as shown in Fig. 2, and the values for saturation as read off the calibration line are reported here. Note that at sea level the saturations at the beginning and end of exercise were generally in the range 97–98, though at the highest levels of exercise there was a tendency for the saturation to fall slightly.

At high altitude the O_2 saturations were markedly reduced, the reductions being greater as the altitude increased. Note also that for a given inspired PO_2 , there was a reduction in O_2 saturation (in either the first or last minute of exercise) at the higher work rates. This phenomenon has been shown by several other investigators at high altitude (3, 5, 13, 25). Note particularly that the fall in arterial O_2 saturation occurred in the face of a rise in alveolar PO_2 as shown in Table 3. The fall in

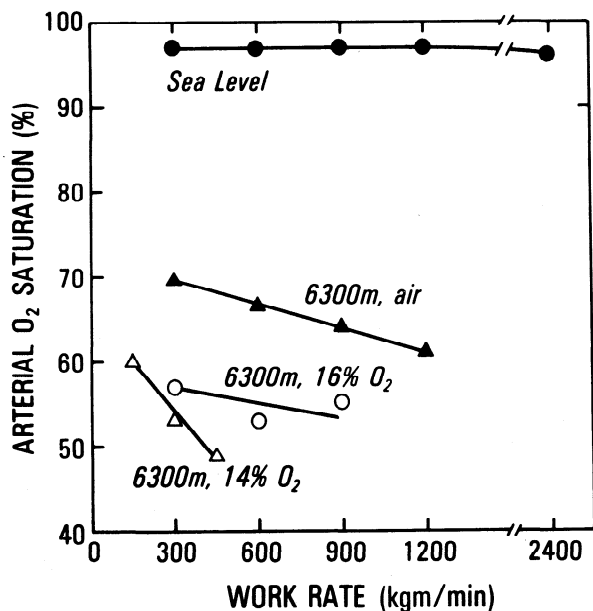


FIG. 3. Arterial O₂ saturation against work rate for 4 conditions studied. Note steeply falling O₂ saturation as work rate was increased when the inspired PO₂ was very low. This can be explained by diffusion limitation of O₂ transfer across blood gas barrier.

O₂ saturation can be explained by diffusion limitation of O₂ transfer under these conditions (17, 25, 27). Indeed theoretical analyses show that even in a resting subject on the summit of Mt. Everest, marked diffusion limitation of O₂ transfer must exist (24, 27).

Table 2 also shows that during the period of exercise at any particular work rate or inspired PO₂ at high altitude there was a tendency for the O₂ saturation to fall, especially at the highest work rates at the lowest values of inspired PO₂. This can presumably be explained by depletion of O₂ stores, resulting in a continued fall in the PO₂ of mixed venous blood and therefore the O₂ saturation of arterial blood.

It is possible to calculate alveolar-arterial PO₂ differences from the available data. To derive the arterial PO₂ from the measured arterial O₂ saturation, we have used measurements of base excess and the PO₂ at which hemoglobin is 50% saturated (P₅₀) made on samples of venous blood taken at the 6,300 m camp (Winslow, Samaja, and West; unpublished observations). The mean value for base excess was -8.7 meq/l and that for P₅₀ at pH 7.4 and PCO₂ = 22.6 was 29.8 Torr. Knowing the alveolar and therefore arterial PCO₂, the arterial O₂ saturation could then be calculated using the Kelman sub-routines (see Ref. 28 for summary). We were unable to take into account the effect of any lactic acidosis; insofar as this reduced the pH, it would reduce the alveolar-arterial PO₂ differences. On the other hand the increases in lactate were small as shown below.

Table 4 shows that the alveolar-arterial PO₂ differences were large at high altitude, being in the range of 8-15 Torr. These are very high in view of the fact that the values of PO₂ are low on the O₂ dissociation curve. These results are consistent with the known marked diffusion limitation of O₂ transfer at high altitude, particularly during exercise. The values are generally consistent with the large alveolar-arterial PO₂ differences

calculated during maximal exercise at an altitude of 5,800 m (barometric pressure 380 Torr) on the 1960-1961 expedition (25). However, they are greater than those reported by Houston and Riley (13) in their low-pressure chamber experiment where the work levels were generally much lower. Note that any alveolar-arterial PO₂ difference caused by ventilation-perfusion inequality is essentially negligible under these extremely hypoxic conditions (27).

Blood Lactate Concentrations

Table 5 shows the results in three subjects after maximal exercise at 6,300 m during air breathing. The maximal work rate of these subjects was 900 kg·m·min⁻¹ and the mean O₂ uptake was 1.75 l/min. The mean value after exercise was 27.1 mg/dl, the mean increase being 11.7 mg/dl. These small increases in lactate concentration in spite of the severe hypoxemia shown in Table 4 are consistent with the results of Edwards (11). The reason for the low lactate levels is not understood but may be related to the relatively low work levels or perhaps the low buffering capacity of the bicarbonate-depleted blood.

Heart Rate

Table 2 shows that, as expected, heart rate increased with work rate at any inspired PO₂ and that, for a given work rate, the heart rate was higher the lower the inspired PO₂. Pugh (19) showed that at an altitude of 5,800 m (barometric pressure 380 Torr) acclimatized subjects had the same cardiac output-O₂ uptake relationship as they do at sea level. If the same holds for the lower values of inspired PO₂ studied here, the high heart rates imply substantial reductions in stroke volume.

TABLE 4. Calculated alveolar-arterial PO₂ differences at high altitudes

Site, Inspired Gas	Work Rate, kg·m·min	Alveolar PO ₂ , Torr	Arterial PO ₂ ,* Torr	Alveolar-Arterial PO ₂ Diff, Torr
6,300 m, ambient air	300	48	36	12
	1,200	52	31	21
6,300 m, 16% O ₂	300	38	28	10
	600	39	27	12
6,300 m, 14% O ₂	150	35	27	8
	450	37	22	15

PO₂, O₂ partial pressure; PCO₂, CO₂ partial pressure. * Calculated from the arterial O₂ saturation, pH, PCO₂, and P₅₀.

TABLE 5. Blood lactate concentrations before and after maximal exercise at 6,300 m

Subj	Hemoglobin, g/dl	Hemato-crit, %	Work Level, kg·m·min ⁻¹	Lactate, mg/dl	
				Before	After
FS	18.7	54	900	21.5	28.6
PH	20.2	54	900	11.0	23.1
JM	17.4	50	900	13.8	29.7

TABLE 6. Summary of results of maximal exercise at each value of inspired PO_2

Site, Inspired Gas	PB, Torr	Equivalent PB for Air Breathing, Torr	Equivalent Altitude for Air Breathing, m	Max Work Rate, $kg \cdot m \cdot min^{-1}$	$\dot{V}O_{2max}$, l/min	$\dot{V}O_{2max}$, $ml \cdot min^{-1} \cdot kg^{-1}$	$\dot{V}E_{max}$, BTPS l/min	Max Heart Rate, $beats \cdot min^{-1}$
Sea level, air	750	750	SL	2,400	4.63	61.3	160	170
6,300 m, air	351.3	351	6,300	1,200	2.31	32.4	207	146
6,300 m, 16% O_2	350.0	280	8,050	600	1.45	20.6	159	128
6,300 m, 14% O_2	350.5	250	8,848	300-450*	1.07	15.3	162	135

PO_2 , O_2 partial pressure; PB, barometric pressure; $\dot{V}O_{2max}$, maximal O_2 uptake; $\dot{V}E_{max}$, maximal exercise ventilation; SL, sea level. * Although both subjects were able to work at $450 kg \cdot m \cdot min^{-1}$ for 3 min (one subject at $600 kg \cdot m \cdot min^{-1}$ for 2 min), the values of the $\dot{V}O_{2max}$ were slightly lower than for $300 kg \cdot m \cdot min$ (see Fig. 3 and Table 2).

Maximal Exercise in Relation to Inspired PO_2

Table 6 summarizes the data.

O_2 uptake. It can be seen that maximal O_2 uptake declined dramatically at the lowest values of inspired PO_2 , i.e., at the highest equivalent altitudes. At sea level the mean value in five subjects was $4.63 l/min$ or $61.3 ml \cdot min^{-1} \cdot kg^{-1}$. When the inspired PO_2 was reduced to 42.5 Torr, equivalent to an altitude of 8,848 m, the mean maximal O_2 uptake in two subjects was $1.07 l/min$ or $15.3 ml \cdot min^{-1} \cdot kg^{-1}$.

Figure 4 shows a plot of maximal O_2 uptake ($ml \cdot min^{-1} \cdot kg^{-1}$) against inspired PO_2 . The figure also shows data reported by Pugh et al. (21) based on measurements made during the Himalayan Scientific and Mountaineering Expedition of 1960-1961 and additional measurements at lower altitudes on Mt. Everest (see Fig. 2, Ref. 21).

Our data differ from those of Pugh et al. (21) in several ways. First note that the maximal O_2 uptake at sea level was substantially higher (24%) than in the subjects of the previous study. As indicated earlier under METHODS, many of our subjects were marathon runners, and in fact all the five subjects whose results gave the point at the top of Fig. 4 fell into this category. Consistent with (though not necessarily caused by) this, the maximal O_2 uptakes at values of inspired PO_2 of 63.7, 48.5, and 42.5 Torr were all above those of the previous study. It might be added that the regression line (least-squares fit) of the previous study is considerably influenced by the lowest point, which was measured under demanding conditions in a tent at an altitude of 7,440 m on the Makalu Col. In addition, one of the two subjects studied developed frank pneumonia 2 days later and was probably not very fit at the time of the exercise study.

Figure 4 shows that the two lines are not widely separated in the lower region, but because the slope is so steep, a small leftward displacement of the curve results in a large increase in maximal O_2 uptake in the present study. These measurements clearly help to explain how climbers can reach the summit of Mt. Everest without supplementary O_2 . Such a feat was very difficult to understand on the basis of the previous measurements of maximal O_2 uptake at extreme altitudes (27). Nevertheless with a maximal O_2 uptake of only about 1 l/min, the climbers clearly have very little in reserve.

Ventilation. Table 6 and Fig. 5 show that maximal total ventilation, expressed as BTPS, increased with alti-

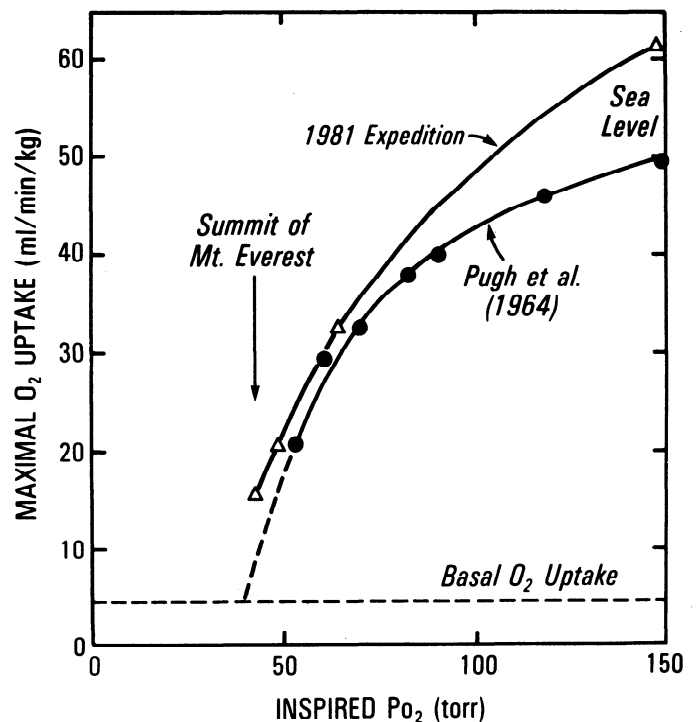


FIG. 4. Maximal O_2 uptake against inspired PO_2 . The present data (triangles) are contrasted with measurements (circles) made previously by Pugh et al. (21). Note that although curve derived from data in present study is only slightly shifted to left, because of steepness of slope, gain in maximal O_2 uptake at extreme altitudes is substantial.

tude up to a certain point and then decreased again. At first sight this is paradoxical, since, as Table 2 shows, for any given work rate the ventilation continued to increase as inspired PO_2 was reduced. The reason for the maximum is that at the highest altitudes the work rates are so low that although ventilation in relation to work rate is high, the absolute value of ventilation is reduced. Note that the broken line showing maximal ventilation in Fig. 5 is based on only four points and is therefore very approximate. It is not possible to incorporate the measurements of maximal ventilation reported by Pugh et al. (21) because the ventilations in that study were generally considerably lower, consistent with the lower maximal work rates. Note that although total ventilation decreases at the lowest PO_2 values, respiratory rate continues to increase resulting in very shallow breathing.

Heart rate. Table 6 and Fig. 5 show that maximal heart rate also decreased with inspired PO_2 . Again there is

something of a paradox here because for a given work rate heart rate increases as inspired PO_2 is reduced. The decline in maximal heart rate can be explained by the fall in maximal work rate.

The finding that maximal ventilation and heart rate were reduced at great altitudes confirms earlier observations of Pugh et al. (21). They noted that maximal ventilation fell from a peak of about 170 l/min BTPS at an altitude of 6,400 m (barometric pressure 340 Torr) to about 120 l/min at 7,440 (barometric pressure 300 Torr). Maximal heart rate fell consistently with increase in altitude as in the present study as also observed by others (5, 6, 21).

Studies during climbing. Ventilation and heart rate were also measured on two subjects (CP and PH) when they were climbing at their maximal rate above Camp 5 en route to the summit on October 24. For these studies the subjects took off their oxygen masks and breathed

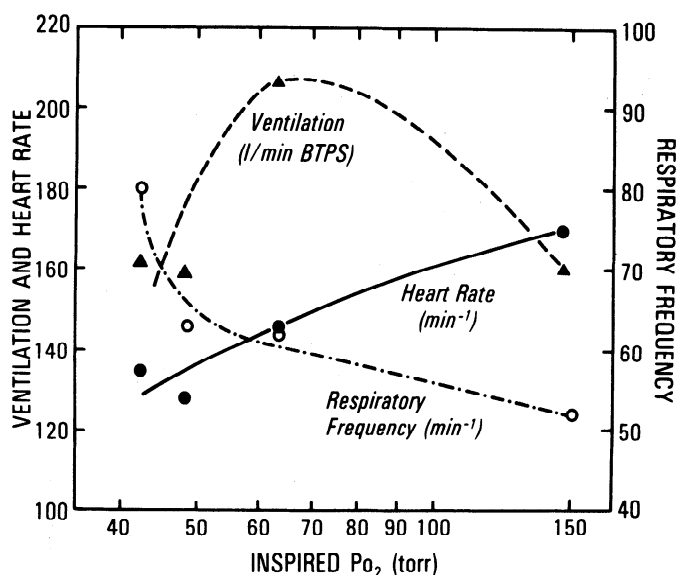


FIG. 5. Maximal ventilation (BTPS), maximal respiratory frequency, and maximal heart rate against inspired PO_2 on a log scale. This scale was chosen because otherwise the high-altitude points fall very close together. Note that both maximal ventilation and heart rate fall at extreme altitudes because work levels become so restricted. However, respiratory frequency continues to increase.

air. The best data were obtained on *subject CP*, who was at an altitude of 8,300 m, as judged from the barometric pressure of 271.4 Torr which he read from his portable barometer. He climbed for 6 min 45 s, while measuring his ventilation with the turbine flow meter. During the middle 4 min his mean ventilation was 107.0 ± 6.3 (SD) l/min BTPS, though it was slightly less for the first and last minutes. The mean respiratory frequency for the middle 4 min was 86 ± 2.8 (SD) breaths/min, giving a mean tidal volume of 1.26 liters. He was therefore breathing rapidly and shallowly.

These values are considerably lower than those obtained during his 3-min measurement of maximal O_2 uptake during 14% O_2 breathing at 6,300 m (Table 7). On that occasion the ventilation was 166 l/min BTPS, frequency 100 and tidal volume 1.66 liters (note the rapid shallow pattern again). The lower values are consistent with the fact that climbers in the field do not reach the highest work levels that can be obtained in the laboratory. In fact Pugh (20) showed that climbers typically select an O_2 uptake of one-half to three-fourths of their maximum for normal climbing at altitudes up to 6,000 m.

The mean heart rate during the last 5 min of free climbing was 134 ± 2.7 (SD) beats/min, which was virtually the same as that measured during maximal exercise during 14% O_2 breathing at 6,300 m, where the value was 132/min.

The data obtained on the other subject (PH) were less satisfactory. He did not have a barometer but estimated his altitude to be 8,500 m, where the barometric pressure is 265 Torr (26). The duration of exercise was only 1 min 36 s, which is too short for steady-state data. Over that period the average values for breathing frequency and heart rate were 50 breaths/min and 144 beats/min, respectively. A satisfactory measurement of ventilation volume was not obtained.

Differences Between Subjects

The pooled data of Table 2 do not show the differences between subjects at any given inspired PO_2 . In some instances these were considerable. For example, some

TABLE 7. Comparison of two subjects exercising at 6,300 m while breathing 14% O_2

Variable	Subj CP			Subj RS			
Barometer pressure, Torr	349.9			351			
Work rate, $kg \cdot m \cdot min^{-1}$	150	300	450	150	300	450	600
Duration, min	5	3	3	3	3	3	2
Ventilation, l/min BTPS	78.8	165.9	179.5	78.0	106.0	144.2	136.6
Respiration rate, breaths/min	84	100	100	54	54	52	60
Heart rate, beats/min	96	132	120	132	138	138	144
Expired O_2 , %	11.89	12.10	12.45	10.90	11.07	10.72	11.07
Expired CO_2 , %	2.32	2.09	2.09	4.00	3.86	3.73	3.18
O_2 uptake, l/min	0.57	1.09	0.92	0.81	1.04	1.05	0.84
Resp exchange ratio	1.12	1.12	1.43	1.35	1.39	1.80	1.82
Oximeter reading, % for each min	1	68	54	55	39	25	20
	2	64	60	37	37	25	17
	3	64	62	45	33	23	17
	4	62					
	5	60					
Alveolar PCO_2 , Torr*	8-9	7-8	7-8	14-15	13-15	13-14	11-12

Comparison between 2 subjects was made at a partial pressure of inspired O_2 value of 42.5, equivalent to the Everest summit. * Calculated for a range of dead space-to-tidal volume ratio from 0.1 to 0.2.

subjects developed a much higher exercise ventilation than others, with the result that they had higher values for expired O_2 concentration and arterial O_2 saturation and lower values of expired CO_2 concentration. Table 7 shows an example of marked differences in ventilatory responses in the two subjects (*CP* and *RS*) studied while inhaling 14% O_2 at 6,300 m. The data are given for all work levels but note particularly the results for the rate of 300 $kg \cdot m \cdot min^{-1}$. This gave the highest O_2 uptakes. Although both subjects were able to sustain 450 $kg \cdot m \cdot min^{-1}$ for 3 min, the measured O_2 uptakes did not increase.

Table 7 shows that both subjects were able to consume just over 1 l/min of O_2 at a work rate of 300 $kg \cdot m \cdot min^{-1}$. However, note the striking differences in ventilation; the value for *subject CP* was 166 l/min, whereas that for *subject RS* was only 106 l/min. Consistent with this, the alveolar PCO_2 calculated assuming a dead space-to-tidal volume ratio in the range 0.1–0.2 was 7–8 Torr for *subject CP* but 13–15 Torr for *subject RS*. As pointed out earlier this value of PCO_2 was the same as that found for *subject CP* on the summit of Mt. Everest when he collected alveolar gas samples using the automatic sampler (24).

Figure 6 shows the ventilations and ear oximeter readings for all the work rates at 6,300 m during 14% O_2 breathing. In this figure the oximeter readings have not been converted to O_2 saturation because the calibration data (Fig. 1) do not extend low enough. Note that the lower ventilations of *subject RS* were associated with lower oximeter readings. These were remarkably low at the high work levels; indeed during the 600 $kg \cdot m \cdot min^{-1}$ run, the reading fell to less than 10% at one point.

However, the low O_2 saturations in *subject RS* cannot be wholly accounted for by his relatively low ventilation. This is clearly seen at the work rate of 150 $kg \cdot m \cdot min$, where although the ventilations were the same, the O_2 saturation of *subject RS* was much lower. It is true that his O_2 uptake was slightly higher (0.81 compared with 0.57 l/min for *subj CP*) and his alveolar PO_2 (calculated from and assumed dead space-to-tidal volume ratio) was 26 as opposed to 29 Torr for *subject CP*. However, this small difference cannot explain the much lower oximeter reading. Presumably the explanation lies in differences in the diffusing capacity for O_2 though we have no data on this point.

The magnitudes of the exercise ventilations correlated strongly with the hypoxic ventilatory responses measured both at sea level and in the Base Camp laboratory (alt 5,400 m, barometric pressure 400 Torr). These studies, which clearly have an important bearing on exercise tolerance at extreme altitudes, will be reported separately.

Comparison of Two Subjects Studied 20 Years Apart

Two subjects of the present expedition (*JM* and *JW*) were members of the Himalayan Scientific and Mountaineering Expedition 1960–1961, and it is of interest to compare their exercise responses both at sea level and high altitude 20 yr later. Table 8 shows that the altitudes at which exercise was studied were slightly different; in 1961 the barometric pressure was 344 Torr (alt 6,400 m),

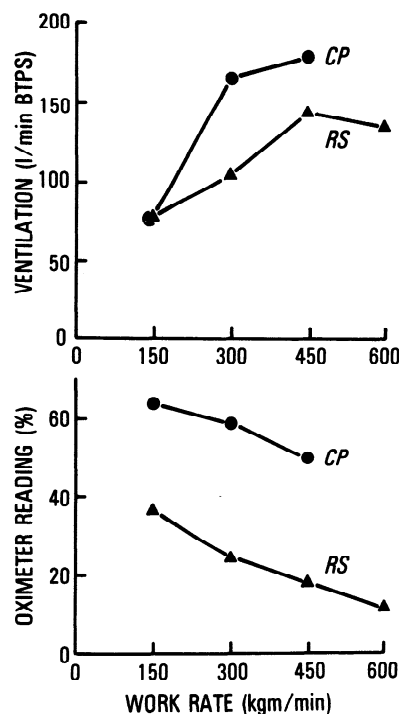


FIG. 6. Ventilations and ear oximeter readings for 2 subjects who exercised at 6,300 m while breathing 14% O_2 . Inspired PO_2 of 42.5 Torr was equivalent to that at summit of Mt. Everest. Note much higher ventilations and oximeter readings for *subject CP*.

whereas on the present expedition the barometric pressure was 351 Torr (alt 6,300 m). *Subject JW* actually performed a measurement of maximal exercise at an altitude of 7,440 m (barometric pressure 300 Torr), in 1961 but there was no comparable study on the present expedition.

It can be seen that there was only a moderate deterioration of performance over the 20 yr period. The O_2 uptakes (l/min) fell by only 6 and 7% at sea level and by only 5% at high altitude for *subject JW*, though the figure was 20% for *subject JM*. When the O_2 uptakes were expressed as $ml \cdot min^{-1} \cdot kg^{-1}$, the decrements were slightly greater, being 7 and 12% at sea level and 25 and 13% at high altitude for *subjects JM* and *JW*, respectively. Maximal heart rate showed a consistent downward trend, but maximal ventilation was variable. The results are in line with the studies by Dill et al. (9), who found that the effects of aging on human performance at high altitude show great variability between individuals but that changes up to the age of about 50 yr may be very small.

DISCUSSION

Limitations of the Low O_2 Inhalation Studies

The experiments in which subjects inhaled O_2 concentrations of 16 and 14% while performing maximal exercise at an altitude of 6,300 m allowed us to obtain information on factors limiting performance at extreme altitudes up to the summit of Mt. Everest. These subjects were well acclimatized to 6,300 m with a partially compensated respiratory alkalosis, the mean base excess being -9.4 meq/l (Winslow, Samaja, and West; unpub-

TABLE 8. Comparison of two subjects studied 20 yr apart

Variable	Subj JM						Subj JW					
	Sea level		% Fall	High altitude		% Fall	Sea level		% Fall	High altitude		% Fall
	1961	1981		1961	1981		1961	1981		1961	1981	
Barometric pressure, Torr	750	750		344	351		750	750		344	351	
Age, yr	30	50		30	51		32	52		32	52	
Weight, kg	68.5	69.2		59.9	64.2		73.9	78.8		64.0	70.1	
Max work rate, kg·m·min ⁻¹	1,860	1,500		900	900		1,950	1,800		1,050	900	
O ₂ uptake, l/min	3.65	3.43	6	1.90	1.52	20	3.93	3.67	7	2.11	2.01	5
O ₂ uptake, ml·min ⁻¹ ·kg ⁻¹	53.3	49.6	7	31.7	23.7	25	53.2	46.2	12	33.0	28.7	13
Ventilation, l/	146.5	105.8		149.4	176.9		153.9	140.1		202.3	178.1	
Heart rate, min	197	182			120		177	162		133	120	

lished observations). The subjects had been at altitudes of 5,400 m or higher for about 5 wk before the studies were made.

However, it should not necessarily be assumed that a subject who is well acclimatized to an altitude of 6,300 m and who breathes 14% O₂ at that altitude is in the same situation as a climber who reaches the summit of Mt. Everest. We might expect additional acclimatization changes to occur during the 3–5 days that a climber would normally take to go from Camp 2 (alt 6,300 m) to the summit. On the other hand there is some evidence that these additional changes were small and that therefore the low O₂ inhalation experiments did nearly reproduce the conditions at extreme altitudes. For example, it was pointed out during the discussion of Tables 3 and 7 that the alveolar PCO₂ of 7–8 Torr of *subject CP* when he achieved his maximal O₂ uptake breathing 14% O₂ at 6,300 m was the same as that measured from alveolar gas samples taken by him on the summit 6 days later (24). This suggests that his exercise ventilation was not inhibited by a lack of acclimatization to altitudes above 6,300 m.

Another line of evidence also indicates that *subject CP* was as well acclimatized to extreme altitudes when the low O₂ mixture experiment was performed at 6,300 m as he was on the summit 6 days later. On the morning following his successful summit climb, venous blood was taken and brought down to Camp 2 (6,300 m) for tonometry and analysis. This sample showed a base excess of -5.9 meq/l (24). This was higher (less negative) than the average base excess of -8.7 meq/l of the expedition members living at 6,300 m (Winslow, Samaja, and West; unpublished observations). Indeed a sample of blood taken from *subject CP* at 6,300 m 16 days prior to the experiment of Table 7 gave a base excess of -9.8 meq/l. Thus these data certainly do not suggest that *subject CP* reduced his base excess more as a result of his excursion to the summit than was the case at the time of the experiment at 6,300 m with 14% O₂ inhalation. Naturally it is possible that other features of acclimatization such as the cerebrospinal fluid pH were not comparable on the two occasions, but we have no data in this area.

During the experiments with low inspired O₂ mixtures, some concern was expressed about possible dangers of the very severe hypoxemia. As an example, the oximeter

reading on *subject RS* during 2 min of exercise at a work rate of 600 kg·m·min⁻¹ while breathing 14% O₂ fell to less than 10%. Since calibration points were not obtained as low as this the actual arterial O₂ saturation is not known but it was certainly extremely low. However, none of the subjects who exercised while breathing low O₂ mixtures (or ambient air) complained of dizziness; the overwhelming sensations were fatigue and breathlessness. Our justification for carrying out these studies was that all the subjects planned to go to the South Col (equivalent to 16% O₂ breathing) and hoped to go to the summit (equivalent to 14% O₂ breathing). Thus the conditions that we simulated were no different from those that they planned to encounter in the next few days in much more hazardous circumstances.

Maximal O₂ Uptake on the Summit of Mt. Everest

The data presented here together with measurements of alveolar gas concentrations taken on the summit (24) improve our understanding of how it is possible for man to climb to the summit of Mt. Everest without supplementary O₂. Previous data (21) as shown in Fig. 4 suggested that the available O₂ on the summit was barely sufficient to satisfy metabolic demands. An extensive theoretical analysis (27) suggested that reaching the summit was probably feasible but that the maximal O₂ uptake there would be less than 700 ml/min.

The present study showed that it was possible for two well-acclimatized subjects to manage an O₂ consumption of over 1 l/min when the inspired PO₂ was 43 Torr, as it is on the summit. Although this is clearly very limiting, it is apparently sufficient to explain how Messner and Habeler reached the summit of Mt. Everest without supplementary O₂. Messner (15) stated that the last 100 m of height took more than an hour to climb. If we assume that they climbed at a rate of 2 m/min and that the weight of a climber plus equipment was 100 kg, the average work rate was 200 kg·m·min⁻¹. This is therefore appreciably less than the 300 kg·m·min⁻¹ corresponding to the maximal O₂ uptake of 1.07 l/min measured on the two subjects inspiring a PO₂ of 42.5 Torr. In fact both subjects were able to sustain a work rate of 450 kg·m·min⁻¹ for 3 min, though there was no increase in O₂ uptake, indicating that they were merely incurring a

larger O₂ debt.

The disparity between our results and those of the earlier experimental study (21) is partly explained by the fact that our subjects were considerably fitter and had a considerably higher maximal O₂ uptake at sea level. In addition, the improved performance of our subjects over that predicted from the theoretical analysis (27) probably stems from three main factors. First, the barometric pressure on the summit is slightly higher than we had predicted (26). Second, the base excess of a subject on the summit is substantially higher (less negative) than we had predicted. It can be shown that the resulting alkalosis enhances the loading of O₂ in the pulmonary capillaries under these very hypoxic conditions and ultimately results in a higher tissue PO₂ (4). Third, the very low PCO₂ brought about by extreme hyperventilation helps to maintain the alveolar PO₂ in the face of a

greatly reduced inspired value. This is the most important of the three factors. Whereas we had previously assumed a PCO₂ of 10 Torr on the summit, *subject CP* reduced his to 7.5 Torr with striking gains in arterial and therefore tissue PO₂. It is the combination of these three factors that just allows human beings to reach the highest point on earth.

We are indebted to all members of the expedition, especially John P. Evans, Deputy Leader—Climbing, and F. Duane Blume, PhD., Deputy Leader—Logistics and Finance.

The expedition was supported by Public Health Service grant RO1-HL-24335 and contract NO1-HR-2915 and grants from the American Alpine Club, American Lung Association, National Geographic Society, National Science Foundation, Servier Laboratories (Paris), The Explorers Club, and the US Army Research and Development Command.

Received 1 November 1982; accepted in final form 22 March 1983.

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