BIBLIOGRAPHICAL STUDY ON THE EFFECTS OF HIGH ALTITUDE ON AEROBIC WORK CAPACITY—MAINLY, MAXIMAL O_2 UPTAKE AND HEART RATE

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Introduction

The climbing of high mountains has fascinated man. It is becoming increasingly popular to spend summer and winter vacation in high mountain areas for recreations such as camping and skiing. Besides this, around 25 million people manage to live and work in the high Andes of South America and the Himalayan ranges of Asia. Even in the state of Colorado, U.S.A. alone, 15 railroad passes are found between 10,285 (3,100 m) and 12,098 feet (3,700 m). More than 10 million people live at altitudes above 3,600 m, and there are mount in dwellers in Peru who daily go to work in a mine at any elevation of 5,700m. They rebelled against living in a camp complaining that they had no appetite, lost weight, and could not sleep. It seems, therefore, that 5,600 m altitude that was built for their settlement is the highest altitude at which even acclimatized man can live permanently.

The 1968 Olympic Games in Mexico City at an altitude of 2,300m created a special interest in the problems concerning the effects of altitude on physical performance.

Let us first examine the usual reactions of an unacclimatized person to hypoxia. Decrease in O_2 tension, reducing the transfer of oxygen from inspired air to the blood in the lungs, calls forth several immediate reactions by the body. The breathing rate increase, in order to bring more air into the lungs. The body steps up its production of red blood cells and of hemoglobin to improve the blood's oxygen carrying capacity. In a person who remains at high altitude these acclimatizing changes take place over a period of time. Investigators, Alexander (1), Bynum (12), Dill (19), Edwards (22), Gill and Pugh (27), Hock (32), Hurtado (34), Kellogg et al. (36), Klausen (37), Lahiri et al. (40), Milledge and Pugh (43), Mayess (42), and Pugh (44), who have measured expeditions to the Himalayan and Andes mountains found that the hemoglobin content of the blood continued to increase for two or three months and then level off.

It has been found by Houston (33), that at 6,000m, a man's capacity for performing exercise without incurring an O_2 debt is only about 50 per cent of that at sea level. The tolerance of such a debt, and of the accumulation of lactic acid in the muscles also is reduced (7, 22, 18). This accounts for the fact that mountain climbers at extreme altitudes can take only a few tortured steps at a time and must rest for a considerable period before going on. The limits on the capacity for work are set, of course, by the limits of the body's possible physiological adjustments to the high altitude conditions. These limits affect the rate of ventilation of the lungs, the heart rate, the cardiac output, and the blood flow to the exercising muscles.

The arterial O_2 saturation which expresses the relationship between the O_2 carrying capacity and the actual content in the blood, drops at high altitude. A drop of the

arterial O_2 saturation to 80 per cent of its normal value will cause severe symptoms of hypoxia known as mountain sickness. Christensen and Forbes (14) reported that 60 per cent of this value, corresponding an arterial Po_2 of approximately 27–28 mmHg, and to 7,000 m high, made nonacclimatized individuals fall unconscious. This reduction in arterial O_2 content at high altitude ought to be a compensatory increase of the blood flow. Since cardiac output consists of two factors; stroke volume and heart rate, it can be considered which of these factors—increase in heart rate or increase in stroke volume—may be of greater relative magnitude when cardiac output becomes higher in acute hypoxia. The maximal O_2 uptake of individuals is depending on the cardiac output and alveoravenous O_2 difference. It is the purpose of this paper, therefore, to study mainly the O_2 uptake, and heart rate at high altitude to assess man's aerobic work capacity in low Po_2 condition.

Acute Hypoxia at High Altitude

At High Altitude. Pugh (48) has reported observations on mountaineers of the 1960-1961 Everest expedition. The experiments were held at four different barometric pressures, 750, 440, 380 and 300 mmHg. The minute volumes of ventilation during heavy exercise were 120, 165, 159 and 120 liters respectively. As compared to the maximum O_2 uptake at the 750mmHg altitude, those at other three altitudes were 76, 63 and 41 per cent of that respectively. Pugh remarks that the several months spent at a barometric pressure of 380 mmHg resulted in some deterioration. Experiences of Chilean sulphur miners support this observation. Evidently the critical barometric pressure above which the long-range net effect is deterioration even for rugged mountaineers is between 400 and 380 mmHg. This reduction of maximum O_2 uptake in acute hypoxia had been reported by Margaria (41), Astrand (5) too. This was due to an effect of the reduced partial pressure of O_2 at altitude and the consequently lower saturation of the arterial blood with oxygen.

Dill et al. (19) tested the work capacity on men at sea level and high altitude (4,300m, Pb 484). The $\dot{V}_{O_2\,max}$ at the altitude was decreased from sea level. In reaching maximum performance at altitude it is noteworthy that the volume of air breathed is as great or greater than in maximum performance at sea level, at least in the more fit subjects. Sometimes the volume decreases at the greatest altitude, eg., Christensen (14) but not always, eg., Pugh's (44) mountaineers. Generally the decline attainable O_2 uptake is relatively greater than the decline in heart rate.

The decrease in maximal oxygen uptake at high altitude was reported by Blomqvist and Stenberg (10), too. During maximal work at hypoxia (4,000m) the O_2 uptake was an average 27 per cent, while cardiac output was 100 per cent of the values attained at sea level. They stated that the maximal O_2 uptake was slightly correlated with the volume of O_2 offered to the tissue.

Dejours et al. (18), and Asmussen (3) found that the exercise heart rate was consistently higher in acute hypoxia than in chronic normoxia. Astrand and Astrand (6) also found that muscular work during acute exposure to high altitude (4,300m, 452-459mmHg) gave a heart rate 15-30 beats higher per minute than under sea level conditions. But at the heavier work loads (1,200 and 1,350 kpm, O_2 intake about 2.5 liters per minute) the heart rate was even lower than in experiments at lower altitude. According to the

study by Dejours et al., the resting heart rate also increases in proportion with the severity of acute hypoxia. The all three subjects in the study elevated their resting heart rate in acute hypoxia (3,100m) and tended to rise progressively rather than fall throughout the 3 weeks of chronic hypoxia. This progressive rise in steady-state resting level during acclimatization differs from some studies (28,3).

Regarding the heart rate at high altitude, Lahiri et al. (40) compared those between Sherpa Highlanders and lowlanders. Resting heart rates of the Sherpa subjects were generally lower than those of the lowlanders at a given altitude. The average heart rates permit at around 3,000m were 63 and 83 for Sherpas and the lowlanders respectively. The corresponding values at 4,800m were 65 and 90, respectively. On the other hand, the rate of increase of heart rate with work rate of bicycle ergometer was greater in the Sherpas at altitude. Some of Sherpas achieved on the maximum heart rate which was the normal maximum for lowlanders at sea level. However, the corresponding work rate or O_2 uptake was smaller (ca. 70%) than at sea level. Heart rate in acclimatized lowlanders was limited to 146–165 at the O_2 uptake levels of 1.70–2.00 liters per minute and the maximal rate at high altitude was considerably lower than at sea level which confirms earlier observations (3,48). Contrary to expectation, there was no significant difference between the Sherpas and the lowlanders in the rate of decrease of heart rate during recovery from a given work rate. As sea level, the heart rate of one Sherpa subject was considerably lower than at high altitude and lower than the rate at sea level of one lowlander. The rate of this heart rate with work rate at sea level was also slower.

Work capacity is determined by O_2 transport and its utilization. The superior work capacity of Sherpas can be attributed to their greater O_2 conductance: i.e. their greater lung diffusing capacity for O_2, possibly better perfusion and lesser Po_2 gradient in working muscles, better control of acid-base homeostasis, and greater ability to tolerate hypoxia.

In the collaborated study by Grover and Reeves (29,30), and Reeves et al. (49, 50) five track athletes from Lexington, Kentucky (300m, 740mmHg) were studied at 3,100m altitude, 530mmHg, Leadville, Colorado. The inspired O_2 pressure is decreased one third. This reduction in the pressure head of O_2 significantly reduced the capacity of the body to take up O_2: i.e. maximal O_2 uptake was reduced an average of 25 per cent with day after arrival. During maximal exertion heart rate approached 200 beats per minute in Lexington. At the 3,100 m altitude the maximal heart rate was still close to 200. In other word, this altitude was insufficient to produce the reduction in maximum heart rates observed at higher altitudes by Pugh et al. (48), Pugh (45, 46, 47), and Houston (33). The recovery heart rate following exertion was affected little by change in altitude. Furthermore, for any given work load, heart rate was faster at the higher altitude, and it follows that maximum heart rate was reached at a lesser work load in Leadville.

As their maximal O_2 uptake decreases, it is natural to decrease in their aerobic work performance. Subjects pedalled bicycle ergometer with a load corresponding five liters of $\dot{V}_{O_2}$ following 5 minute-warm-up with a power load which required 2 liters of $\dot{V}_{O_2}$. The duration of effort and maximal heart rate at sea level were 1.39 minutes: that of the test at 2,800m shortly after their arrival was 1.36 minutes. Of course, in this study by Balke et al. (8) too, all subjects showed a reduced maximal O_2 uptake capacity at the altitude, averaging approximately 6 per cent less than sea level. This decrement occured

despite a substantial rise of pulmonary ventilation as other studies reported.

In the field tests, the 400m run (test of anaerobic performance capacity) and the mile run (test of aerobic work capacity) were in complete agreement with results of tests performed in the laboratory. They found that times for the 400m confirmed laboratory findings that sprint-type efforts remain practically unaffected by a change of altitude from 400–2,300m. However, the mile runs at the altitude test were considerably slower than at sea level.

Consolazio (15) and Consolazio et al. (16, 17) reported the data on two studies conducted at 3,500m and 4,300m to evaluate aerobic work performance on the bicycle ergometer. O_2 uptake during rest and submaximal work were unchanged from sea level up to elevation of 4,300m. Pulse rates and pulmonary ventilation were increased with an increase in high a altitudes. However, there was a significant decrease in maximal bicycle riding time, pulse rate and O_2 uptake with an increase in high altitude. The decrease in performance from sea level values averaged 20 per cent at 4,300m, 17 per cent at 3,500 m, and 7 per cent at 1,500m.

No significant differences or beneficial effects were observed in maximal work performance in men who had physical conditioning or those who did not. This was also true for the men who ascended gradually or abruptly to high altitude.

Under the condition of reduced O_2 pressure in the inspired air, the lower O_2 saturation must be compensated with something else during exercise. For this document, Asmussen and Neilsen (4), and Stenberg et al. (55) found an increased cardiac output during submaximal work. At Pikes Peak, Grollman (28) found increased cardiac output for the lst several days at 3,800m. According to the study by Vogel et al. (58), cardiac output increased from sea level to an 4,300m altitude by 12 per cent at rest, 16–18 per cent during exercise, and 20 per cent during recovery. The largest absolute change with altitude during exercise occurred at the moderate work level.

It is clear that the increase in the cardiac output is brought about by an increase in the heart rate. There are conflicting results whether stroke volume increases or not. There is a disagreement regarding the behavior of the stroke volume during submaximal work at high altitude. Pugh (46), Klausen (37), and Alexander et al. (1) found a decrease. Alexander et al. stated that the reduced stroke volume could be with a decreased myocardial efficiency. A few possible mechanism are: pulmonary hypertension and right ventricular overload: depretion of myocardial norepinephrine stores: diminished activity of the sympathetic nervous system: smaller blood volume and lower ventricular filling pressures: impairment of myocardial oxygenation: and shift in pH.

As contrast of those finding, Vogel et al. (58) found that during the first week at 4,300m, stroke volume was unchanged from sea level values at rest, mild exercise, and recovery, but was significantly elevated at moderate and maximal exercise.

At Moderate Altitude. It is generally considered that the altitude below 2,300–2,500m is moderate. Highly conditioned college subjects were studied at an altitude of 2,300 m (24). Compared to sea level values, the maximum O_2 uptake determined on the bicycle ergometer decreased 2–3 per cent during first two weeks while the pulmonary ventilation at maximum O_2 uptake increased 16 per cent. These findings show the similar trend as found at high altitude.

Saltin (51,52,53), working with Scandinavian athletes, reported that at the acute

exposure in a low pressure chamber to 580mmHg, which is corresponding to the altitude in Mexico City, there was no difference in the maximum values for heart rate, blood lactate and the pulmonary ventilation compared sea level. The maximum O_2 uptake was, however, markedly reduced. The average difference was 16 per cent from sea level. This difference is much bigger than expected from studies on non-athletes. Balke et al. (8) and Dill et al. (20) studying non-athletes or not top athletes have presented below ten per cent difference with about the same altitude as Mexico City or even slightly higher altitudes. One possible explanation for the fact that those with very high aerobic work capacities are more affected than non-athletes may be that the pulmonary diffusing capacity limits the athletes more at high altitude.

Similar result was reported by Ikeda et al. (35) working on Japanese athletes. The maximum O_2 uptake at 2,300m in Mexico City showed a drop of the maximum O_2 uptake by 20–30 per cent of sea level after arrival. The record of electrocardiogram after exercise suggested increased load on the right atrium and a slight coronary hypoxia in the first week of this stay.

Prolonged Exposure to High Altitude.

The effects of a prolonged stay at high altitude is discussed here. The effects are called acclimatization to reduced oxygen pressure in the inspired air both in short-term adaptation (weeks or a few months) and long-term adaptation (years). In the Astrand and Astrand's study (6) there was a gradual decrease in heart rate at a given O_2 intake when the hypoxia (4,300m) was prolonged. In the later states of acclimatization the heart rates attained during lower level conditions.

Schilling et al. (54) found a tendency to a lower maximal heart rate in man and in dog later acclimatization to altitudes of 4,500 to 6,000m. Cerretelli and Margaria (13) also found the same tendency. In their study of 11 members of a Himalayan expedition, an average reduction of maximum heart rate was approximately 0.80 after 60 days acclimatization period at 5,000m as compared with the sea level values. Hurtado (34) observed lower heart rates when residents at an altitude of 4,200m ran at a fixed speed to exhaustion than when similar experiments were conducted at sea level with subjects resident there. The interpretation of these findings of a depressed maximal heart rate after acclimatization to altitude is not clear. However, one of the lowlanders in Lahiri's study (40) showed gradual increase in his maximum heart rate in the increasing duration of resistance at 4,800m.

In the previously mentioned study (51, 52, 53), although they showed a slightly delayed slowing recovery heart rate only on the first few days after arrival at 3,100 m, the high school athletes regained their sea level rate within 2 weeks.

The aerobic capacity of lowlanders is markedly reduced at high altitudes even during more than one month acclimatization period. Cerretelli and Margaria (13) found that average reduction of maximum O_2 uptake to 0.44 of the sea level was observed after 60 days acclimatization at 5,000m. Buskirk et al. (11) reported the similar finding from their investigation at a 4,000m altitude on conditioned runners. All runners showed a marked reduction of maximum O_2 uptake. On the average $\dot{V}_{O_2\,max}$ was reduced 29 per cent on days 3 and 21, and 26 per cent on day 48 at the altitude. On return from altitude $\dot{V}_{O_2\,max}$ of the runners were similar to prealtitude values. Bicycle-riding time on

day 3 at 4,000m was reduced by 12 per cent compared to that at 3,000m: but it approached prealtitude values after the 20th day at altitude. Average running times for the 400m, 800m, 1,600m, and 3,200m distances were 91, 82, 77, and 81 per cent of sea level times. Post altitude performance times of the runners were 96–100 per cent of their prealtitude times. Thus, there was no change in either the $\dot{V}_{O_2\,max}$ or performance on the track pre-as compared to post-altitude. The studies by Consolazio (15) and Consolazio et al. (16, 17) confirmed with the above mentioned findings. No superior maximal work performance on the bicycle ergometer was observed on return to sea level after 28 days of exposure to high altitudes of 4,200m.

Those reports contradicted with the results of living at high altitude by Balke et al. (9) and Klausen et al. (38) which reported that the prealtitude marks were surpassed after returning to low altitude. Klausen et al. found an average 14 per cent increase in the maximal O_2 uptake after return to low altitude as compared with the control values observed before ascending the mountain although a 13 per cent reduction of the maximal O_2 uptake of control values was found at a 2.5 week-acclimatization at altitude. They found an increase of only slightly 4 per cent $\dot{V}_{O_2\,max}$ during the period of acclimatization. Klausen et al. also reported a trend downward in maximal heart rate during the 5 weeks stay at an altitude of 3,800m and an increase again upon descent to lower altitude.

The similar observations were reported from the studies at moderate altitudes too. Faulkner et al. (24) found normal capacities for maximum O_2 uptake by the third week at a 2,300meters acclimatization. On return to sea level, the $\dot{V}_{O_2\,max}$ averaged 8–9 per cent higher than in the prealtitude control tests. Maximum work capacity on the bicycle ergometer decreased initially at altitude also increased equal to sea level values during the second and third week, and increased significantly over prealtitude control values on return to sea level. The study by Balke et al. (8) at a 2,800m altitude also demonstrated clearly that acclimatization of ten days duration had restored maximum O_2 uptake capacity of nearly all subjects to normal.

There are the studies which reported non-significant effects of acclimatization at the altitudes. In the second study by Faulkner et al. (25), no change in $\dot{V}_{O_2max}$ was observed at a 2,300m altitude during 6 weeks of training. Time trials of 1–3 miles at this elevation were 2–13 per cent slower than at sea level. During the post-altitude control period this study could not find significant improvements of time trial performance and $\dot{V}_{O_2\,max}$ from the prealtitude control values either. Let's see the studies in Mexico City, 2,300m. An seven day acclimatization showed only 1–2 per cent of improvement in maximal O_2 uptake from that of acute exposure to altitude. Fifteen per cent of improvement was found by 15 day-acclimatization (51, 52, 53). Although $\dot{V}_{O_2\,max}$ of Japanese athletes reached its highest value after 12–14 days of acclimatization in Mexico City, it was reported that the value still remained 10–20 per cent below sea level (35).

A sharp increase in cardiac output has been consistently noted following ascent to high altitude in many studies. A marked reduced maximum $\dot{Q}$ during prolonged duration of acclimatization (4–7 months) was reported by Pugh et al. (48) although the cardiac output at rest and at a given work intensity was the same as sea level. The values were 16–17 liters per minute compared with 22 to 25 liters per minute at sea level. They stated that this reduction of cardiac output was a combined effect of a lowered stroke volume and maximal heart rate (192 to 135). The same finding had earlier been report-

ed by Christensen and Forbes (14) and Alexander et al.(1). Alexander et al. also stated that the reduced cardiac output was chiefly due to a decrease in stroke volume. On the contrary, Hartley et al. (31) measured somewhat 8 per cent of increased cardiac output of 3,100m residents after 10 days at sea level, and 15 per cent of increased stroke volume. Saltin (53) also found that sea level residents studies after two weeks at 4,300m had a 20 per cent reduction in maximal cardiac output due to a smaller stroke volume. Furthermore, Vogel et al. (58) studied 16 soldiers, at sea level and during 15–18 days at 4,300m. Their values for maximal cardiac output were significantly greater initially at high altitude than at sea level and fell back toward sea level values during 2 weeks' sojourn. For the decrease of cardiac output at high altitude after acclimatization, Anderson and Gray (2) pointed out that it may be the increased hematocrit.

There are the studies that investigated the aerobic capacities on acclimatized high landers native. The $\dot{V}_{O_2\,max}$ of high school residents at 3,100m increased 27 per cent of the value at a 300m altitude (30). Five highly trained athletes at University of New Mexico (1,550m) were studied at sea level conditions. The mean treadmill run time at their native altitude of 231 seconds was considerably less than the 326 seconds recorded at sea level. Since total work performance at sea level was greater than that at altitude, and this additional work was accomplished with relatively small change in terminal pulse and terminal ventilation rate, it would seem that this would indicate an increase in aerobic work capacity at a higher barometric pressure (12).

Hurtado (34) assessed the aerobic capacity of the Peruvians dwelling at Morococha (445mmHg, 4,540m) in terms of the performance of running on the treadmill at sea level. High altitude residents proved superior despite the fact that their conterparts were athletes. The O_2 debt was greater in the athletes: evidently they had to rely more on anaerobic reserves than did the residents of Morococha.

Another study (39) also investigated the aerobic capacity of the chronically acclimatized Peruvian Indians native to 4,000m altitude. Maximum aerobic capacities of the Indians were similar to those observed in trained runners who were newcomers to altitude and remained there between 50 and 64 days. The average $\dot{V}_{O_2\,max}$ of the indians and newcomer athletes was 53 ml/kg per min. and 49 ml/kg per min. respectively. The values exceed those reported by Elsner (23) for natives at 4,500m and by Velasquez (57) for newcomers to 4,500m who lived at this altitude for 12 months. However, $\dot{V}_{O_2max}$ of the newcomer non-athletes was 37 ml/kg min.. Despite high aerobic capacities, the indians achived the lowest maximum bicycle riding times and peak work loads. This lower gross efficiency reported for natives during treadmill running by Hurtado (34). Kollias et al. (39) concluded that the newcomer athletes to high altitude appearently has an O_2 transport system relatively equivalent to the Indian natives to high altitude. But the indian native to high altitude has an O_2 transport system superior to the unconditioned newcomer to altitude.

Frisancho et al. (26) studied the influence of developmental adaptation on aerobic capacity at high altitude. Again, the Peruvian Indians were investigated. The aerobic capacities of the Peruvian highlanders native, Peruvian lowland migrants (they were born below an altitude of 1,000m and migranted to high altitudes between the ages of 2 and 16 years), and Peruvian lowland and U.S. newcomers were tested at an altitude of 3,400m. The duration of residence at the altitude was 3–21 yrs., 1–4 yrs. and 0.3–2.4 yrs. in the migrants, Peruvian newcomers and U.S. newcomers respectively. The average

$\dot{V}_{O_2\,max}$ of the lowland migrants did not differ from the highland permanent dwellers (46 ml/kg min.). Those of Peruvian and U.S. newcomers were significantly lower than that attained by the highland controls. It was also reported that the average maximal heart rate for the lowland migrants and lowland newcomers was 193/min compared to 196 of highland controls. The maximal heart rate, 187 of U.S. newcomers was significantly lower than that of highland controls. The investigators found that among those who were acclimatized to chronic high-altitude hypoxia, during the developmental period, the age at migration and length of residencc at high altitude are significantly correlated with the attainment of $\dot{V}_{O_2max}$. In contrast, among the Peruvian and U.S. newcomers who were acclimatized to high altitude as adults, the aerobic capacity was not related to age at migration or length of residence at high altitude. It appears, therefore, that acclimatization to high altitude during the developmental period permits a sea-level men to acquire an aerobic capacity equal to that attained by the high-altitude native.

Little information exists about the performance of women at altitude except that obtained in Mexico City during the competition in the Pan American and Olympic games.

Summary

The paper has reviewed the physiological effects on the aerobic capacity at high altitudes. The most incisive feature of the altitude climate is the attenuation of barometric pressure with a proportionately leads to a disparity between the O_2 requirements of aerobic metabolism and the O_2 available from the atmosphere. Therefore, as man acutely ascends from sea level to high altitudes, maximal O_2 uptake decreases owing to the fall in O_2 tension of inspired air and the consequent decrease in O_2 content of arterial blood. The factors limiting an aerobic capacity at high altitude, however, are not limited to the O_2 tension in the air, but other factors enter into picture affecting the resistance to hypoxia: these factors are responsible for acclimatization.

With hypoxic condition pulmonary ventilation at a given O_2 uptake is markedly elevated. This finding is common to all investigators at altitudes. Such hyper ventilation constitutes a definite advantage in that it yields a proportional increase of the alveolar O_2 pressure and arterial O_2 pressure, and hence a proportionally larger O_2 diffusion gradient between blood and tissues is maintained. This obviously facilitates the diffusion of O_2 to the blood in the pulmonary capillaries.

The heart rate increases in proportion with the severity of acute hypoxia. Most of the studies report an increase in resting heart rate. During physical exercise at fixed work loads, the heart rate is always higher in acute exposure to high elevation than it is at low altitudes. This increase in the heart rate plays important role to increase in the cardiac output which is inevitable to compensate reduced O_2 pressure in the inspired air, the lower O_2 saturation as mentioned previously. The maximal heart rate, therefore, is attained at work intensity levels below those attainable at sea level. In general, the maximal heart rate during a few days or even a month at altitude, may be less than that at sea level. It is well established by many studies that the greater the altitude the lower is maximal heart rate.

In general, exposure to elevations above 2,300m have invariably resulted in impairment in maximum aerobic capacity and in performance distance running. The effect

of the reduced oxygen pressure on the physical work capacity is different in different individuals. Dill et al. (20) summarized the aerobic work capacity in acute hypoxia as followed:

Table 1

Altitude (m) Pb (mmHg)	3,100 535	3,800 485	4,300 455	
Work capacity	95	91	86	% of sea level
$\dot{V}_{O_2\ max}$	90	86	81	
Lactate	93	96	89	
$\dot{V}_{E\ max}$	102	106	99	
HR_{max}	98	97	96	
O_2 pulse	92	88	84	

According to Table 1, there is less loss in work capacity than in $\dot{V}_{O_2\ max}$. This may depend in part on a smaller decrease in anaerobic work capacity rather than in aerobic work capacity. Success in attaining the same limiting $\dot{V}_{E\ max}$ may indicate that the bellows function of the respiratory system is unimpared. Maximal heart rate is nearly but not quite as high as at sea level.

With prolonged exposure to reduced O_2 pressure in the inspired air, a further increase in pulmonary ventilation at a given work load is observed. This hyperventilation will further increase Po_2 of the alveolar air as a conpensatory device. The maximal heart rate is reduced compared with sea level values at high altitude. During both, submaximal and maximal work the stroke volume is reduced. A decreased myocardial efficiency at high altitude is a possible rationale for the reduction. Therefore, the initially observed increase in cardiac output during exercise is replaced by a gradual decline to or even below the values observed at sea level.

Table 2 Reduction (%) of $\dot{V}_{O_2\ max}$ from low altitude controls

Investigator(s)	2,300	3,100 535	508	3,800 485	4,000 462	4,340 455	4650 440	5,000 410	5800 380	6400 344	7440m 300mmHg	staying
Saltin (1967)	85											acute
Dill et al (1966)		90		86		81						acute
Stenberg (1966)	85				72							acute
Astrand (1954)		91			72							acute
Dill et al (1967)				76								7-day
Buskirk (1967)					71							3-21
Klausen (1966)				88								17
Saltin (1967)	93											14
Dill et al (1967)				83		78						21
Consolazio et al (1966)			82			75						21
Grover et al (1966)		75										21
Buskirk (1967)					79							48
Balke (1964)				94								56
Cerretelli (1961)								44				60
Christensen (1937)								50				
Pugh et al (1964)							70		61	51	33	

In short term acclimatization, the reduced $\dot{V}_{O_2\,max}$ may not regain the value at sea level. The disparity in the reported reduction in $\dot{V}_{O_2\,max}$ at altitudes is shown in the Table 2. The disparity may depend on fitness, on athletic status, of the subjects, and on the end of the test. There is also lack of agreement as to where improvement begins.

There seems to be some disagreement about the effect of prolonged acclimatization to high altitude on cardiac output. However, it is the fact that during maximal work the cardiac output is reduced. A reduced stroke volume appears to be the primary reason for the reduced cardiac output. The lowering of the heart rate may cause the reduction but it inconsistent.

At a high altitude hemoglobin concentration is markedly higher in the natives: and also their arterial O_2 content is greater than that in acclimatized sojourners. It can be stated, therefore, that an increased hemoglobin concentration in the blood is also a conpensatory device in acclimatization at high altitude. This increase in the hemoglobin concentration in sojourners constitutes a significant adaptive mechanism to hypoxia because of the resulting increase in O_2 carrying capacity of the blood. Since Torrance reported that cardiac output, O_2 consumption and arteriovenous O_2 content differences are identical in the highland natives and the acclimatized sojourners at altitude, it can be understood that the natives have a smaller O_2 utilization and thus a greater O_2 reserve

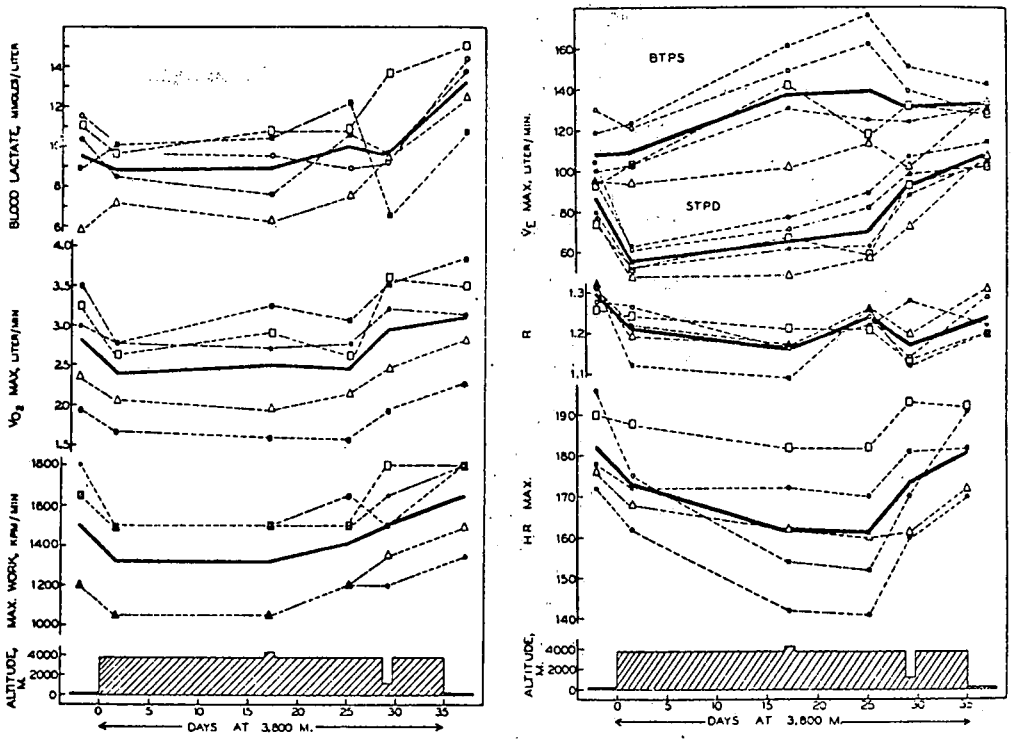

Fig. 1. Maximum values of work rate (max work), oxygen uptake ($\dot{V}_{O_2\,max}$), blood lactate, heart rate (HR max), respiratory exchange ratio (R), and ventilation ($\dot{V}_{E\,max}$)from the six serises of experiments. Thick lines=mean values. From left to right on the abscissa the values represent date from experiments at: 1) 264m before ascent to 3,800m, 2) 3,800m 1-2 days after ascent, 3) 4,343m, 4) 3,800m 3-4 weeks after ascent, 5) 1,220m, and 6) 264m after descent from 3,800m. (by Klausen et al., 1966)

than the acclimatized sojourners. Consequently the natives have better tolerance to exercise at high altitude.

An exposure with physical training to hypoxia increases vascularization in the skeletal muscles and may change the oxidative transport system in the mitocondoria. However, the findings reported do not show an agreement in the improvement of aerobic performance after return to sea level.

Figure 1 shows the six serises of experiments, 2 pre- and post-altitude tests, and 4 tests at altitudes during 35 days of sojourn.

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