ALTITUDE TRAINING CAMPS

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Rationale of Altitude Camps

The idea that athletes might benefit from a period of several weeks training at a high altitude camp first gained credence in the quadrennium preceding the Mexico Olympics. The classical studies of Barcroft (7) and Haldane (23) had shown much earlier that mountain climbers underwent a progressive physiological adaptation to the stresses of high altitude, while the natives and permanent residents of mountainous regions showed adaptive features similar in type but of even greater magnitude. It was thus reasoned that a period of altitude training might minimize the disastrous early effects that the altitude of Mexico City was reputed to cause in coaches and athletes alike.

In fact, the problems associated with competition at an altitude of 7,350 ft (2,230m) were greatly exaggerated. From the medical point of view, there were no cases of pulmonary oedema and only one or two mild episodes of apparent lack of cerebral oxygen. Cardiac arrhythmias were perhaps more frequent (28,38) than at sea level, but no permanent myocardial damage was sustained. In terms of physical performance, results were even more remarkable; 29% of successful competitors exceeded the best world marks, and the average of winning scores was only 0.9% poorer than world records — compared with 2.9% poorer in previous Olympic contests (1).

If performance is expressed as a percentage of the world record, and is plotted against the duration of the event, it becomes possible to distinguish brief events (where performance was aided by the diminished air resistance), and longer events (where there was a progressive loss of performance: 3% at 4 minutes, increasing to 8% at one hour) (1). The performances of Keino, Burton and Hohne far exceeded that of the other contestants, and in the case of Keino it was logical to postulate a causal relationship between residence at altitude and outstanding performance. From this point, it seemed but a small leap of faith to suggest that if living at altitude was good for competition at altitude, it was even better as a preparation for sea level competition.

Definitions

Altitude. High altitude has different meanings for different people. The classical physiologists were

concerned with balloon exploration, mountain conquest, high altitude flying, and problems of mining in the peaks of the Andes. The trajectory of Tissandier's balloon, reaching almost 9,000m (2,800 ft) is typical of this period of investigation.

Athletic training camps, in contrast, have been set at quite moderate altitudes (2,000-3,000m — 6,500-9,800 ft — comparable with Mexico City and other large cities of South America). It is thus dangerous to assume that the classical physiological work can be applied to adaptation at these altitudes. The fall in partial pressure of oxygen with altitude is logarithmic, and in the altitude camp we are dealing with a barometric pressure of 550-600 mm Hg corresponds to an oxygen pressure of 115-125 mm Hg.

Class of athlete. It is equally necessary to distinguish the impact of the training camps upon several different classes of athlete; the performance of air-resisted sports is modified in a different way to that of water-resisted sports. Equally, the impact of altitude differs for short term (anaerobic), medium term (aerobic), and long term (glycogen, heat, and water limited) sports.

The Ultimate Response

The physiological characteristics of the fully-adapted high-altitude resident are well shown by Hurtado's classical comparison of the residents of Lima (close to sea level) and Morococha (14,900 ft — 4,550m). The most obvious feature of the Morocochans is a substantial increase of red cell count, with an almost parallel increase in the haematocrit. There is also an increase in the colour index (the quantity of haemoglobin in the individual red cell) and an increase in the total blood volume. More recent work (17,29) has shown there is also an increase in 2:3 diphosphoglycerate levels that leads to a shift to the right of the oxygen dissociation curve. While this would facilitate the release of oxygen from haemoglobin in the tissues, in practice the gains are at least partially offset by the respiratory alkalosis of altitude.

There are also ventilatory changes. Hurtado's study showed that consciousness is maintained at a very low alveolar oxygen pressure, and a modest increase of resting ventilation is sustained despite very low alveolar carbon dioxide pressures. Natives resident at high altitudes are also said to have rather barrel-shaped chests,

and in his study he found a small increase of vital capacity. The literature is somewhat confused regarding changes of static and dynamic lung volumes; in general though, the BTPS vital capacity is said to remain unchanged, the BTPS dynamic volumes to increase, but the STPD dynamic volumes to remain unchanged.

Data on the ventilatory response to exercise are likewise confused by problems of units. Generally, ventilation has been expressed as l/min BTPS, which is what the body *feels*. However, in terms of oxygen transport, it is the STPD volume that is important. The BTPS volume is increased on first reaching altitude, but not to the extent that it is in the permanent resident and the native; in the study of Reeves et al (31), the STPD ventilation of the lowlander was also increased at 10,000 ft (3,050m). Natives of high altitude may show a blunting of peripheral chemoreceptor sensitivity to oxygen lack, but this is generally regarded as either a genetic characteristic, or at least the response to a stimulus present from birth.

Other recent work has shown an increased capillarity of the skeletal muscles (10,45), elevation of myoglobin levels (25,38), enhanced lactic acid utilization through development of the DPNH-oxidase system (30,37), and various ultrastructural changes (8).

Value to the Athlete at Sea Level

Assuming the various changes I have discussed could be induced by a short period of altitude training, and that they could also be preserved on return to sea level, how useful would they be to the athlete?

Haemoglobin

An increase in haemoglobin level is of obvious advantage to the competitor over medium distances. About 50% of the energy requirements for an event of one minute's duration are met from oxygen transport, and in a five minute event 80% of effort is dependent upon the maximum oxygen intake.

I have made an analysis of oxygen transport between the atmosphere and the active muscles (3). The overall conductance for oxygen ($\dot{G}$) is best described by a complex four term equation:

$$\frac{1}{\dot{G}} = \frac{1}{\dot{V}_A} + \frac{B}{1-B} \frac{(1)}{(\lambda \dot{Q})} + \frac{1}{\lambda \dot{Q}} + \frac{K}{1-K} \frac{(1)}{(\lambda \dot{Q})}$$

where $\dot{V}_A$ is the alveolar ventilation, λ is the effective solubility of oxygen in blood, $\dot{Q}$ is the cardiac output, and B and K are constants. At sea level, the second and

fourth terms are very small, and the equation thus simplifies to

$$\frac{1}{\dot{G}} = \frac{1}{\dot{V}_A} + \frac{1}{\lambda \dot{Q}}$$

with $\lambda \dot{Q}$ having a larger impact upon oxygen transport than $\dot{V}_A$.

Oxygen transport could be augmented by an increase in either λ or $\dot{Q}$. The normal response to endurance training is an increase of $\dot{Q}$ (max). The value of λ is determined by the slope of the oxygen dissociation curve between arterial and mixed venous points, and is thus increased in direct proportion to any gains of haemoglobin concentration, whether produced by training or altitude exposure. However, the increase of oxygen transport is a little less than indicated by the increase of λ , since the maximum cardiac output may be diminished by the associated increase of blood viscosity, and at altitude the increase of 2:3 DPG produces a shift to the right of the oxygen dissociation curve.

Several strands of experimental evidence document the importance of haemoglobin level to oxygen transport. A recent study of carbon monoxide poisoning (19) has shown a close parallel between blood carboxyhaemoglobin levels and the associated diminution of maximum oxygen intake. In anaemic East African children, maximal oxygen intake is less than would be predicted from leg volume (15). And, more directly, the physical working capacity of athletes has been increased by autotransfusion of refrigerated blood (36; Åstrand — personal communication).

An increase of haemoglobin level may be of particular value to the athlete, since many endurance competitors have low initial readings, the reasons for which remain uncertain. In some competitors, it may reflect an expansion of plasma volume without a proportionate increase of red cell mass. In others, a fad of feeding or poverty may lead to an inadequate diet. If prolonged exercise is performed repeatedly in a hot climate, a substantial quantity of iron may be lost in the sweat. With some forms of exercise, particularly track work, increased haemolysis is signalled by a decrease of serum haptoglobins, while in other instances there are decreases in red cell formation (Kiiskinen — personal communication). But irrespective of mechanism, it is plain that many athletes have less than an optimum haemoglobin level for sedentary adults, and their performance in endurance type events could probably be improved by an increase in their haemoglobin readings.

Ventilatory changes

The control of respiration during exercise is still

something of a mystery (16). It is perhaps convenient to distinguish the early phases of effort (when neural factors such as conditioned reflexes, proprioceptive drive, and radiation of impulses from the motor cortex are dominant), more sustained activity (where acid metabolites assume increasing importance), and prolonged work (where a rising core temperature may make some further contribution). As we have noted above, the reduced blood and tissue bicarbonate levels of the altitude-adapted athlete are associated with an enhancement of the second phase in the ventilatory adaptation to a given level of effort — a given increment of blood lactate leads to a larger increase of respiratory minute volume.

This may have adaptive value at altitudes where STPD ventilation is initially reduced, with substantially unsaturated arterial blood, but it does not help sea-level competition, where arterial blood is normally close to full oxygenation. Indeed, it reduces the potential for performance, for while the maximum oxygen intake is not materially changed by hyperventilation, an increased proportion of the total aerobic power is diverted to the respiratory muscles (41).

The diminution of tissue bicarbonate also affects the working muscles, reducing their buffering capacity. This limits their potential for anaerobic work, restricting performance in all events where lactate accumulates — sprint and medium distance events lasting from 10 to 60 seconds, and longer events that involve substantial isometric effort.

Tissue changes

Tissue diffusion normally imposes little limitation upon oxygen transport, and the gains of performance from increases in muscle capillarity and other ultrastructural changes are thus slight. The increase of myoglobin level is particularly useful in increasing the alactic anaerobic power of the sprinter, while the increased ability to metabolize lactic acid influences performance over the range 10 to 60 seconds of activity. Unfortunately, there seems no quantitative information on the likely extent of changes at training camp altitudes.

Short-Term Effects of Altitude

Few athletes have the good fortune to be born at altitude, and constraints of practicality and international rules limit the usual altitude camp to no more than six weeks. It is thus necessary to consider both the time course of the changes seen in permanent residents at altitude and certain shorter-term disturbances that appear soon after arrival at camp.

Haemoglobin

The classical study of Pugh (33,35) showed the red cell

count increasing over a month of altitude exposure and hard physical work, as the mountaineers climbed over successive ridges of the Himalayas. Haemoglobin increased even more slowly, reaching a peak after about two months. The red cell count returned to normal during a three week descent, although the haemoglobin concentration remained increased. However, the altitude of these experiments was 15,000-20,000 ft, (4,600-6,100m) and it could be argued that the privations of the journey, including limited rations and progressive dehydration contributed to the observed changes.

A number of studies were made in preparation for the Mexico Olympics. We studied three subjects who spent a week in Mexico City; at the end of this time, the red cell count was uniformly increased, but there was a small decrease of haemoglobin level, possibly attributable to the release of immature red cells into the circulation. Faulkner (1) found a 4% increase in the haematocrit and a 10% increase in haemoglobin after two to three weeks at altitudes comparable to those of Mexico City, but these changes were almost completely reversed within four days of return to sea level. Asahina et al (4) found increases in red cell count, haemoglobin level, and haematocrit developing over the first week in Mexico City; the haemoglobin level rapidly reverted to the sea-level figure in Japan, although surprisingly the red cell count remained elevated. The Swedish team studied by Saltin (39) showed a comparable pattern: rise in haemoglobin within a few days, and loss of haemoglobin beginning immediately on return to Stockholm. Two studies at higher altitudes (around 14,000 ft — 4,300m) have yielded a similar type of response, although the absolute gains in haemoglobin were larger. Buskirk et al (9) found the major part of their gain of haemoglobin occurred within six days, with complete reversion between three and eight days after descent to sea level. Haematocrit changes were well established at six days, but developed further at 11 days, returning to normal again between three and eight days at sea level. Finally, there is a study by Hannon, showing that in women athletes at least, the adaptive changes can be speeded by administration of iron.

The usually accepted rate of red cell formation is 1% per day. Thus in order to produce an 8-10% increase of haemoglobin level within one to two weeks, it would be necessary almost to double the normal rate of red cell formation. It is hard to believe that this occurs in response to such a mild stimulus as provided by the usual altitude training camp, and from the plasma volume data to be discussed below it seems more reasonable to postulate an initial haemoconcentration, with the true increase of body haemoglobin content developing more slowly over several months. The other factor clearly brought out by these various studies is the transient nature of the increase in haemoglobin concentration. On return to sea level, normal values

reappear within a week or less. While decreases of red cell production and increased haemolysis may play some role, a restoration to normal of the plasma volume seems largely responsible. However, Pugh's study suggests that if a true increase of haemoglobin is induced by longer residence at altitude, this may persist for several weeks.

Ventilatory changes

Our data for the first week in Mexico City showed no change of vital capacity and a small gain of the BTPS one second forced expiratory volume. Cotes (13) has shown that the gain of dynamic capacity is purely a mechanical effect of the reduced gas density, and it is thus reversed immediately on return to sea level.

The time course of buffer changes has been studied by Severinghaus and his associates (40). The blood pH remains elevated throughout the first week at an altitude of 12,470 ft (3,800m). However, cerebrospinal fluid pH may return to sea-level values within as little as 12-24 hours, implying a quite rapid adjustment of tissue buffer systems. On the other hand, the adjustment of plasma bicarbonate continues slowly over the course of exposure. Our studies in Mexico City confirmed that the changes in standard bicarbonate level of the blood are slow, and the resting arterial pH remains elevated over the first week at the new altitude. Buskirk's data illustrate adaptations after a month at 13,000 ft (3,950m). At rest, a very low pCO_2 is sustained without appreciable rise of pH, since plasma bicarbonate is markedly reduced. However, the low bicarbonate gives impaired buffering, so that in exercise pH falls much more than at sea level. On return to an altitude of 900 ft (275m), the tissue changes are fairly rapidly reversed, but blood bicarbonate is restored relatively slowly. Thus, exercise is again associated with an abnormal depression of blood pH.

The long-term effect of high altitude exposure is a decreased production of lactic acid on maximum effort, as exemplified by the data of Edwards (18). However, this is less clearly established for shorter periods of residence. Buskirk (9) found an increased blood lactate following maximal exercise on the 42nd day at altitude; his initial sea level readings were quite low, and it is possible subsequent production of lactate was boosted by training and/or increased lactate production in sub-maximal stages of the test procedure. Hermansen (26) has shown that while at altitude, lactate production begins at a smaller absolute oxygen intake, although at a similar percentage of aerobic power.

The practical impact of the diminished tissue buffering seems reflected in the subjective comments of athletes. Pulse recovery rates are slowed, and longer intervals are needed between heats or training sessions; further, if attempts are made to maintain a training

schedule, unusual muscle stiffness may be noted (43).

Tissue adaptations

Adaptations at the tissue level are apparently completed within one to two weeks, and although there are little data on this point, reversion to sea level values might be expected over a similar time span. The tissue oxygen conductance is almost thirty times that of the cardio-respiratory system (3), so that the sea-level competitor would gain little advantage from further development in this part of his oxygen transport system.

Short-term disturbances

A number of short-term disturbances may interrupt the pattern of training while at altitude. The average citizen is not particularly conscious of mountain sickness at altitudes below 12,000 ft (3,660m). However, the athlete who is seeking to maintain a high training schedule may complain of headache, insomnia, irritability, and a variety of gastrointestinal disturbances in the first three days at altitude. His training plan may be further disrupted by a long journey, the psychological problems of life away from home, and gastrointestinal infections such as the famous "turista", so that he approaches competition in far from peak condition.

To these non-specific problems must be added a very dramatic fluid loss. Jungmann, for instance, found a weight loss of almost 2 kg (4.4lb) in the first two weeks at 2,000m (6,550ft) (28), and Hannon (24) found an even greater change with several weeks of residence at 14,000ft (4,270m) on Pike's Peak. Hannon suspected that part of the weight loss was fat, since parallel changes could be demonstrated in various body skin folds; however, it is important to remember that the state of body hydration can also influence skinfold thickness. Their data (for women athletes) showed a 20% diminution of blood volume, maximal at about the 30th day of exposure. Buskirk's data (9) showed an equal change in men by the 15th day of exposure, with full reversal 15-20 days after return to sea level. As might be expected, the changes of plasma volume lead in turn to a reduction of cardiac stroke volume. In a study by Vogel (46), the stroke volume at a given work load was reduced by 10% in the third week at altitude, but normal values were restored within five days of return to sea level. Cardiac output data reflect not only the diminished stroke volume, but also a compensatory tachycardia in sub-maximum effort; for the present purpose, the most significant finding is a normal cardiac output response to work within five days of return to sea level. In view of the dominant influence of cardiovascular conductance (λQ) upon oxygen transport, it would seem imprudent for an endurance athlete trained at a mountain camp to compete at sea level before normal plasma and stroke volumes are restored.

Aerobic power

The responses to standard sub-maximal and maximal exercise tests provide a convenient physiological measure of the responses to altitude training, favourable and unfavourable.

The immediate responses to altitude, well shown in our step test data are increases in the pulse rate and the cardiac output during sub-maximal effort (42); these in themselves can be sufficient to compensate for the 7-8% decrease in the oxygen content of arterial blood. As we have seen, in the study of Vogel (46), the cardiac response to sub-maximal work is normal within five days of return to sea level. The maximum rate of working is generally reduced, due to the reduction in both stroke volume and heart rate and the diminished oxygen saturation of arterial blood. Pugh (33,35) has demonstrated that the reduction of maximum pulse rate and oxygen transport develop progressively as the altitude is increased; however, some authors (9) have argued that there is a threshold of 5,000-6,000 ft (1,500-1,800m), and that no changes of maximum oxygen intake develop below this ceiling.

The ultimate effect of altitude training is still in dispute. With fairly high altitudes it seems reasonably well established that maximum oxygen intake is depressed not only during exposure, but also to a lesser extent on return to sea level. In Buskirk's experiments (9) at 14,000 ft (4,270m), $\dot{V}O_2$ (max) was no more than 75% of normal after 48 days at altitude, and only 96% of normal on the seventh and 15th days after the return to sea level. Grover (22) exposed his athletes to 3,100m (10,000 ft); again, aerobic power was about 75% of the sea level value, with a persistent deficit of about 12% on return to sea level. Consolazio (12) took his subjects to 11,400 ft (3,475m); again there was a 25% loss of aerobic power, with recovery but not improvement of aerobic power after return to sea level.

Some of those who have worked in Mexico City, such as the Swedish team (39) have had similar experiences. Saltin compared his maximum oxygen intake data obtained in Mexico City with that previously determined in a low pressure chamber in Stockholm; the loss of aerobic power was about 18% in Stockholm, 16% after eight days in Mexico City, and 11% after 16 days. Saltin commented on the difficulty top athletes found in maintaining training in Mexico City, and on return to sea level small decrements of maximum oxygen intake were registered.

In contrast to this work, Faulkner (1) found that his athletes were able to develop 100% of their sea level maximum oxygen intake after 21 days at an altitude of 2,300m (6,550 ft), and that there was an 8-10% advantage of aerobic power from three to 21 days after return to sea level. Faulkner conceded that his athletes

were not in peak condition before going to altitude, and the gain of aerobic power could represent a response to training rather than to residence at altitude. However he pointed out that it would be unusual to achieve a gain of this order in a partially conditioned athlete attending a sea level training camp. Faulkner measured his maximum oxygen intake data not only on the treadmill, but also during tethered swimming; with the latter technique, there was an 8% loss of aerobic power after 14 days of altitude residence, and a 3% advantage in free style and butterfly strokes on return to sea level. Asahina et al (4) also noted significant gains of maximum oxygen intake and treadmill performance on return to sea level, these gains disappearing over two weeks.

Several factors probably contribute to the discordant results. One is altitude — is this great enough to cause a reduction of plasma volume and stroke volume? A second important variable is the level of competition. Saltin (39) found a greater loss of aerobic power in top-level athletes than in physical education students during the period at altitude. Linked to this is the body's potential for training and the extent to which normal training schedules have been maintained at altitude. Certainly, any gain of maximum oxygen intake after brief residence at altitude is small, and the top athlete may actually find his condition has deteriorated.

Physical performance

The ultimate proof of any training regimen is performance on the track; in general, data for endurance performers coincide with the maximum oxygen intake figures previously discussed. Our figures for a Canadian swimmer in Mexico City showed a deterioration of performance that increased with increase in the competitive distance (42); the somewhat smaller losses found by others in track athletes was due to the influence of diminished air resistance. Thus, at 13,100 ft (4,000m) Buskirk (9) found a 20-24% loss of performance compared with a 25% loss of maximum oxygen intake; on return to sea level, track performance was only 94-96% of normal on the third day and 92-97% of normal after 15 days.

On the other hand, Faulkner (1) found only a 6% increase in the time to swim 500 yards (457m) on the first day at altitude; the impairment of performance had dropped to 4% on the third and subsequent days. On return to sea level, swimming times were 1-2% better than before exposure to altitude. Likewise, track times at 2,300m (6,550 ft) were 5% poorer on the first day, but by the 14th day at altitude the increase in times was a mere 1%. On return to sea level, track times were 7% better than before exposure both on the third and 21st days.

Thus, the physical performance data confirm the physiological evidence. Brief exposure to very high

altitudes has a detrimental effect on performance. The response to more moderate altitudes apparently depends on initial fitness, the level of competition, and ability to maintain training schedules. It may be beneficial, but this is by no means always the case. Much may depend on the ability to make the necessary translations of technique quickly; thus, when swimming at altitude, shorter periods are spent underwater, and more strokes are made per length, with corresponding alterations of breathing frequency — particularly in distance events.

Recommendations

To conclude, the advantages of altitude camps are far from proven. If reliance is to be placed on this technique, the best recommendation would seem to choose a moderate altitude where derangements of training by mountain sickness, gastrointestinal infections, and loss of plasma volume are minimal. Further, the period of residence should be long, so that there is a true polycythaemia rather than a haemoconcentration. Competition should then be arranged within about three

days of return to sea level. This will allow recovery from the journey, psychological and technical (pace) adjustment to the sea-level milieu, and reversion of plasma volume and tissue buffers to normal levels, with preservation of a fair part of any altitude induced polycythaemia.

However, even if the exercise is successful, it may be questioned whether it is worthwhile. Is it sporting to attempt to manipulate blood haemoglobin levels? And if so, are there not more effective methods of producing such changes — autotransfusion, the administration of various erythropoietic agents, or even periodic exposure to low oxygen pressures at sea level? Where does one draw the line between the legitimate treatment of anaemia in an athlete and the deliberate perversion of human performance? Given adequate funds for research, there is no question that physiologists could devise techniques that would further extend endurance performance, but sports physicians must urgently face the question of whether it is ethical or desirable to do so.

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DISCUSSION

CHAIRMAN: Faulkner's runners were extremely slow, were they not?

PROFESSOR SHEPHARD: Of course, this is one of the criticisms which has been made of Faulkner's study relative to the others. You will remember that his was the one that showed benefit from altitude training, and several people have commented that his subjects were not in anything like the same category as those studied by Saltin for instance.

CHAIRMAN: Another point of interest is this question of what I consider not an anaemia, but hypoaemia (if that means anything) — the physiological hypoaemia of the man who is capable of using extremely large quantities of oxygen per minute.

When I gave up physiology three or four years ago, the record figure, expressed properly in ml oxygen/kg, was slightly over 6 litres of oxygen per minute. This was published in a paper of Hermansen. There was an enormous cardiac output too — 40 litres per minute, I think — but the blood was relatively low in haemoglobin. Probably a great deal of haemoglobin was being pushed round by a magnificent pump; but pushed round extremely fast because the blood was relatively dilute and its viscosity low.

There are all sorts of speculations in which one can indulge in this connection. I am not convinced that polycythaemia at sea level would be of much use. Looking on it as a total increase in haemoglobin, one would probably need an increase in the circulating plasma volume too — in other words, more haemoglobin more dilute. If that is achieved, the body weight is raised and more energy is needed to get over the ground — so perhaps we are back where we started.