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# Adaptation to High Altitude

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**Abstract.** Hypoxia is inconsequential for physiologically fit persons below an effective altitude of 2640 metres. At higher altitudes, the adaptation is brought about by four main factors, viz., hyperventilation, increased diffusion of oxygen across alveolar membrane, erythrocythemia and maintenance of body hydration. Carbon dioxide sensitivity is markedly elevated at high altitude, both in sojourners and acclimatized low-landers. The greater pulmonary diffusing capacity observed in high altitude natives is well documented. RBC count, haemoglobin and haematocrit increase whereas arterial oxyhaemoglobin saturation percentage decreases at high altitude. Diuretics (Furosemide) have no role in adaptation to high altitude and adequate body hydration must be maintained. The ultimate adaptive mechanisms occur at tissue level which facilitate the diffusion of oxygen from blood to tissue and its utilization. The work capacity decreases at high altitude and a relationship between load carried and speed of marching has been determined at various altitudes. Although altitude has an adverse effect on process of cold acclimatization, yet it is possible to induce cold acclimatization by exposing subjects to a temperature of 0° to −5°C for a period of three hours daily for three weeks. The caloric requirements increase at high altitudes and are 4,286 K Cal and 4,380 K Cal at 13000 feet (3950 m) and 17000 feet (5170 m), respectively.

## 1. Introduction

No defining limits have been set for the term high altitude. Practically, however, hypoxia is inconsequential for physiologically fit persons below an effective altitude[1,2] of 2640 metres, but increases at a progressively greater rate from minimal to severe as elevation increases. The adaptation to high altitude in temporary residents is brought about by four main factors: hyperventilation, increased diffusion of oxygen across alveolar membrane, erythrocythemia and maintenance of body hydration.

## 2. Hyperventilation

Nayar[1-3] studied in detail about hyperventilation from sea level to 4560 metres altitude (Table 1). The increase in pulmonary ventilation was directly proportional to high altitudes. At 4560 metres altitude, the temporary residents showed an increase of about 45% in ventilation as compared to sea level values. At all the altitudes, the basal metabolic rate ($O_2$ consumption) was almost same but the ventilatory quotient increased with increase in altitude. Hurtado[4] got lower figures because his subjects were natives of 4540 metres altitude. High altitude hyperventilation was an important adaptive mechanism because it kept the alveolar $PO_2$ high and the fall from atmospheric to alveolar $PO_2$ was decreased. Inspite of hyperventilation, the alveolar $PO_2$ fell from 101.2 mm *Hg* at sea level to 46.2 mm *Hg* at 4560 metres altitude. However, the factors responsible for the high altitude hyperventilation are not fully understood. *Mathew*[5] *et al.* studied the chemoreceptor sensitivity in adaptation to high altitude. The results showed no significant alteration in the hypoxic sensitivity of the lowlanders (LL); but $CO_2$ sensitivity was markedly elevated at high altitude, both in sojourners and acclimatized low-landers (AL). The high altitude natives (HAN) showed less sensitivity to hypoxia, whereas the $CO_2$ sensitivity remained normal. Subba & Nayar[6] found that inhalation of 100% oxygen through BLB mask increased arterial oxygen saturation at 3350 metres altitude (mean 85.18%) far below expectation, while at Delhi the rise was satisfactory (mean 98.88%). It was further observed that inhalation of pure oxygen by acclimatized subjects at 4560 metres altitude under resting conditions led to decrease in respiratory minute volume which became halfway between sea level value and that observed when subjects were breathing atmospheric air at 4560 metres altitude[1-7].

## 3. Increased diffusion of Oxygen Across Alveolar Membrane

Diffusion of oxygen through alveolar membrane between alveolar air and circulating blood is the second important factor in adaptation to high altitude since higher the diffusion greater will be arterial $PO_2$ and oxygen saturation of blood. The diffusion through the alveolar membrane of the lungs was studied by Selvamurthy[8] *et al.* They concluded that there was an initial significant decrease in pulmonary diffusing capacity for carbon monoxide ($D_L$ CO) in LL on arrival at high altitude (3500 m). During ten days of stay at high altitude the $D_L$ CO showed improvement, returning to initial sea level value and showed a trend to increase further. One year of altitude acclimatization improved $D_L$ CO of the LL to the level of $D_L$ CO observed in high altitude natives (HAN). West[9] observed no consistant change in $D_L$ CO at 15,300 feet altitude (subjects breathing ambient air) compared with sea level, but $D_L$ CO was significantly increased after 7-10 weeks stay at 19,000 feet (mean changes of 15 and 19% for work levels of 300 and 900 kg-m/min, respectively). West[9] accounted this small change in $D_L$ CO due to increased rate of reaction of carbon monoxide with haemoglobin as a result of hypoxia and increased blood haemoglobin concentration. The greater pulmonary diffusing

**Table 1.** Resting respiratory value in sea level and temporary residents at various altitudes. Mean values with standard error

| Altitude (metre) | Sea level | 1530 | 2640 | 3350 | 3950 | 4650 |
|---|---|---|---|---|---|---|
| Duration of stay at altitude (month) | Residents | 6 | 8 | 8 | 11 | 11 |
| Number of subjects | 59 | 17 | 12 | 7 | 16 | 7 |
| Barometric Pressure (mm Hg) | 738 | 632 | 550 | 501 | 462 | 435 |
| Body Weight (Kg) | 58.9±1.01 | 58.9±0.98 | 58.2±1.03 | 57.4±0.81 | 57.8±0.85 | 58.9±0.93 |
| Body Height (cm) | 167.1±1.21 | 167.8±1.23 | 167.2±1.09 | 166.7±1.07 | 168.6±1.05 | 170.4±1.04 |
| Body Surface Area ($M^3$) | 1.64±0.03 | 1.65±0.04 | 1.64±0.03 | 1.66±0.02 | 1.68±0.03 | 1.69±0.02 |
| Ventilation (l/min BTPS) | 6.79±0.21 | 7.46±0.28 | 8.42±0.39 | 9.11±0.99 | 9.24±0.61 | 9.89±0.61 |
| Ventilation (l/min/$M^2$. BTPS) | 4.14±0.09 | 4.52±0.13 | 5.16±0.18 | 5.46±0.26 | 5.50±0.29 | 5.85±0.31 |
| Ventilation (l/min/Kg. BTPS) | 0.12±0.001 | 0.13±0.002 | 0.14±0.003 | 0.15±0.02 | 0.16±0.003 | 0.17±0.004 |
| Respiratory Rate/min | 13.9±1.0 | 14.5±1.2 | 16.9±0.8 | 17.2±0.9 | 17.9±0.7 | 18.7±1.1 |
| Tidal volume (l/BTPS) | 0.49±0.02 | 0.51±0.02 | 0.50±0.01 | 0.53±0.02 | 0.52±0.03 | 0.53±0.02 |
| $O_2$ Consumption (l/min STPD) | 0.196±0.029 | 0.210±0.009 | 0.194±0.008 | 0.192±0.010 | 0.190±0.011 | 0.191±0.005 |
| Ventilatory Quotient (litres Vent. BTPS/litres $O_2$ Consumed STPD) | 34.6±0.61 | 37.1±0.82 | 43.4±1.00 | 47.4±1.25 | 48.6±1.31 | 51.7±1.66 |
| Alveolar ($PO_2$ mm Hg) | 101.2±3.6 | 83.4±2.5 | 68.1±2.7 | 59.0±2.2 | 48.1±2.0 | 46.2±2.1 |

capacity observed in high altitude natives is well documented[10,11&12]. This enhanced diffusion in high altitude natives is due to the persence of a greater surface area of the alveo-capillary membrane[13], the high altitude hyperventilation and the greater pulmonary blood volume in them[11,13&14] all these factors leading to a greater ventilation perfusion ratio[13].

## 4. Erythrocythemia

It is the third main factor for adaptation to high altitude. The peripheral haematological values in sea level and temporary residents at various altitudes is shown in table 2. It will be seen that RBC count, haemoglobin and haematocrit increased at high altitude, whereas arterial oxyhaemoglobin saturation percentage decreased progressively from sea level to 4560 metres altitude. All the same, arterial blood oxygen content volume percentage remained almost the same at sea level and at various altitudes. This is brought about by absolute increase in RBC count (erythrocythemia). Thus at high altitudes sufficient oxygen is available in the arterial blood but at a lower tension[1]. These findings were confirmed by Jain[15] *et al* who found that haemoglobin, haematocrit, erythrocyte and reticulocyte counts increased first rapidly on induction to high altitude and then attained maximum values by 2nd or 3rd week of exposure. Plasma volume decreased in LL but remained at the same level in HAN on re-induction to high altitude. This resulted in relatively less haemoconcentration in HAN which was beneficial from haemodynamic viewpoint. Erythrocytes formed at high altitude were macrocytic and remained in circulation for some days even after deacclimatization. Various haematological parameters studied returned to initial sea level values on deacclimatization except a little rise

**Table 2.** Peripheral haematological values in sea level and temporary residents at various altitudes. Mean values with standard error.

| Altitude (metre) | Sea level | 1530 | 2640 | 3350 | 3950 | 4560 |
|---|---|---|---|---|---|---|
| Duration of stay at Altitude (month) | Residents | 6 | 8 | 8 | 11 | 11 |
| Number of Subjects | 59 | 17 | 12 | 7 | 16 | 7 |
| Red blood cells (million/cu mm) | 4.85±0.32 | 5.01±01.18 | 5.64±0.18 | 5.81±0.41 | 5.92±0.31 | 6.08±0.29 |
| Haemoglobin (G/100ml) | 15.2±0.6 | 15.8±0.4 | 17.6±0.6 | 18.4±0.7 | 19.2±0.3 | 20.1±0.9 |
| Haemotocrit (% RBC) | 43.5±1.8 | 45.1±0.9 | 49.2±1.8 | 50.6±1.8 | 51.7±3.0 | 53.4±2.5 |
| Arterial oxyhaemoglobin saturation (%) | 97.2+2.4 | 96.2±1.0 | 92.5±1.1 | 85.9±1.8 | 83.9±1.5 | 81.4±0.7 |
| Arterial blood oxygen content volume (%) | 19.5±1.1 | 19.2±0.9 | 19.5±0.7 | 18.9±0.8 | 19.9±0.9 | 20.5±0.7 |

in serum bilirubin which persisted for a few more days. Erythrocyte life span did not change significantly in man upto an altitude of 3500 metres. In animal studies erythrocyte life span decreased on acclimatization and re-induction to simulated altitude of 6100 metres. This was further supported by the observed decrease in erythrocyte glutamic oxaloacetic transaminase (EGOT). The re-induction to hypoxia imposed a more severe stress than acclimatization as the erythropoiesis was more rapid. The observed deaths in rabbits during re-induction phase were possibly due to haemoconcentration and increased blood viscosity as blood volume did not increase with a decrease in plasma volume. The rabbits became fully adapted after three generations at high altitude as exhibited by normocythaemic hypervolemia.

## 5. Maintenance of Body Hydration

Jain[16] *et al* studied body fluid compartments in sea level residents during 12 days of acute exposure to an altitude of 3500 metres. Measurements of total body water and extracellular water were done on the third and 12th days of acute exposure, while plasma volume was measured on 12th day only. The intracellular water, blood volume and red cell mass were computed from the above parameters. Total body water and extracellular water decreased progressively, the decrease being 4.7% ($p < 0.001$) and 6.0% ($p < 0.05$) respectively on the 12th day. Plasma volume and blood volume decreased significantly with a slight increase in red cell mass. Intracellular water, decressed by 4.3% on 12th day. This study suggested hypohydration on acute altitude exposure. Jain[17] *et al* studied organ fluid compartments in rats exposed to 6100 metres altitude for 5 to 24 hours. Total water and extracellular water content of various organs, i.e., lungs, liver, spleen, heart, kidney, muscle, brain, testis and subcutaneous tissue were determined by the difference of dry and wet weights and using radiobromide-82 respectively. Lungs and liver were found to be significantly hydrated with lower water contents in subcutaneous tissue, spleen and muscle on prolonged exposure. The data indicated a shift of fluids from extracellular to intracellular compartments. These findings are in agreement with those of Zink[18] *et al* and Oelz[19]. In fact, the latter recommended to estimate daily urine output and to drink enough beverages and soups so as to achieve a daily urine volume of at least 1.5 litres. The high rate of fluid loss from the lungs associated with increased ventilation in the dry cold air made it necessary to consume 4-7 litres of fluid every 24 hours. However, this is contrary to the recommendations made by Singh[19,20] *et al.* They did large scale controlled trials in men air lifted from sea level to 11500 feet and found that furosemide 80 mg every 12 hours for two days, begun immediately on arrival, had a definite place in the prevention of acute mountain sickness and high altitude pulmonary oedema in fresh entrants as well as former residents. They claimed that the prevention was nearly complete in acute mountain sickness and complete in high altitude pulmonary oedema. However, Kwatra & Viswanathan[21] failed to confirm the usefulness of prophylactic dehydration procedures in animals. They used different batches of animals of different species (rat, mice, guinea-pigs)

for experiments and furosemide was given by injection 10 mg/kg body weight 2 hours before exposure to simulated altitude of 10,000 metres for 6 hours. The number of survivors was noted. The results showed that the animals altitude tolerance was definitely reduced by giving the drug before exposure. Significantly more animals died in the group given furosemide than among the controls. Zink[18] *et al* recommended haemodilution as a clinical routine procedure of proved safety, effective and feasible before induction to high altitude. They summed up that high altitude climbing was always associated with moderate-to-severe dehydration unless special precautions were taken. With much water lost by humidification of inspired air (upto 7 litres/day), the resulting haemoconcentration might quickly become hazardous, since it caused increased blood viscosity, hypercoagulability of the blood, decreased oxygen transport capacity and disturbed microcirculation with an impaired heat supply, especially to the acral tissues. Bhardwaj[22] *et al* measured total body water (TBW) in the HAN after they had stayed at sea level for one month. TBW was experimentally determined by oral administration of 200 $\mu$Ci of tritiated water. These men were found hyperhydrated. Thus hyperhydration of the lean body mass could be an adaptive response of HAN to the new environment.

### 5.1 *Adaptation at Tissue Level*

Inspite of the major four adaptive mechanisms, there was still fall in arterial $PO_2$ e.g. : at 4560 metres altitude the arterial $PO_2$ fell from 95.2 to 45.5 mm Hg (Table 3). Thus the ultimate adaptive mechanisms occurred at tissue level which facilitated the diffusion of oxygen from blood to tissues and its utilisation. These included increased capillary bed[23] higher myoglobin content in muscles [24,25] and change in enzymatic pathways in cells which allowed increased utilisation of oxygen at low tensions[25,26]. Ahuja & Nayar[27] assayed plasma membrane bound ATP ases of erythrocyte ghosts of rats bred at approximately sea level and at high altitude

**Table 3.** $PO_2$ in mm Hg gradients from inspired air to venous blood in sea level and temporary residents at various altitude. Mean values with standard error.

| Altitude (metre) | Sea level | 1530 | 2640 | 3350 | 3950 | 4560 |
|---|---|---|---|---|---|---|
| Duration of stay at altitude (month) | Residents | 6 | 8 | 8 | 11 | 11 |
| Number of subjects | 59 | 17 | 12 | 7 | 16 | 7 |
| Inspird air ($PO_2$ mm Hg) | 145.3±1.5 | 123.8±1.9 | 105.7±2.1 | 95.2±1.1 | 87.8±2.5 | 81.9±1.5 |
| Alveolar air ($PO_2$ mm Hg) | 101.2±3.6 | 83.4±2.6 | 68.1±1.7 | 59.0±2.2 | 48.1±2.0 | 46.2±2.1 |
| Arterial blood ($PO_2$ mm Hg) | 95.2±2.4 | 80.2±1.0 | 66.5±1.1 | 57.9±1.8 | 47.0±1.5 | 45.5±0.9 |
| Venous blood ($PO_2$ mm Hg) | 40.9±9.8 | 39.2±10.5 | 39.1±9.8 | 38.4±5.9 | 32.5±4.5 | 28.8±3.6 |

(3500 metres). The $Mg^{2+}-AT$ Pase showed a threefold increase while $Na+$, $K-AT$ Pase showed a small decrease in ghosts from high altitude bred rats. The results indicated that better conditions existed for energy dependent transmembrane transport of ions at high altitude while sodium pump activity was reduced.

### 5.2 *Work Capacity*

Immediately upon arrival at high altitude, only moderate physical work can be performed because of extreme breathlessness and subsequently headache, nausea, weakness and dizziness. Nayar[28] found that on acute induction to 3350 metres altitude, maximum work rate fell to 50% of the sea level value. Table 4 shows maximum rates of oxygen consumption and work along with percentage work tolerance of subjects at sea level and high altitudes. Thus, the expectation that freshly deployed, unacclimatized troops can go immediately into action (combat or other) is most likely to be frustrated and might end in disaster if such a unit was confronted with an acclimatised enemy. Even after several months of residence at 4560 metres the maximum rate of work (measured ergometrically) remained at 64% of the sea level value[29]. Table 5 shows physiological responses to maximum exercise at sea level and at various altitudes. It will be seen that the limiting factors to work capacity at high altitude are multiple. Firstly, fall in alveolar $PO_2$ leads to decreased saturation of arterial oxyhaemoglobin which in turn decreases the release of oxygen at tissue level. Secondly, decrease in maximum heart rate due to inadequate oxygen supply limits cardiac output. Thirdly, diminished capacity to incur oxygen debt due to lowered ability to accumulate blood lactic acid. Lastly, attainment of maximum breathing copacity at lower work rate. Nayar[30] determined the maximum economical load which could be carried by a healthy male adult while marching at a fixed speed at sea level, 13,000 and 16,500 feet above sea level-respectively. The maximum economic load was taken as that load which could be carried with a relatively low oxygen consumption and beyond which any increase in load, caused the oxygen consumption to increase disproportionately and more steeply. It was found that the maximum economic load which could be carried while marching 120 paces

**Table 4.** Maximum rates of oxygen consumption and work alongwith percentage work tolerance of subjects at sea level and rapid induction to high altitudes. Mean values with standard error.

| Altitude (metre) | Sea level | 3350 | 3950 | 3950 |
|---|---|---|---|---|
| Duration of stay at altitude | Residents | 24 hours | 3 weeks | 8 weeks |
| Number of subjects | 7 | 7 | 7 | 7 |
| Maximum Oxygen Consumption (l/min STPD) | 2.99±0.7 | 1.97±0.09 | 2.31±0.11 | 2.39±0.12 |
| Maximum work rate (Kg meters per minite) | 1062±13 | 528±8 | 718±7 | 798±8 |
| Work tolerance (%) | 100 | 49.8 | 67.6 | 75.1 |

**Table 5.** Physiological responses to maximum exercise at sea level and various altitudes. Mean values with standard error.

| Altitude (metre) | Sea level | 1530 | 2640 | 3350 | 3950 | 4560 |
|---|---|---|---|---|---|---|
| Duration of stay at altitude (month) | Residents | 6 | 8 | 8 | 11 | 11 |
| Numher of subjects | 59 | 17 | 12 | 7 | 16 | 7 |
| Work rate (Kg metres per minute) | 1102±22 | 1074±10 | 1091±25 | 921±15 | 790±15 | 711±9 |
| Oxygen consumption (l/min STPD) | 2.80±0.71 | 2.99±0.18 | 2.76±0.72 | 2.67±0.26 | 2.32±0.69 | 2.14±0.22 |
| Alveolar $PO_2$ mm Hg | 109.0±4.8 | 88.5±3.8 | 79.4±3.9 | 69.7±3.4 | 62.1±5.1 | 55.9±2.7 |
| Heart rate/min | 190±14 | 186±16 | 188±12 | 162±8 | 157±8 | 152±9 |
| Blood lactic acid (mg%) | 63.7±6.7 | 61.9±3.8 | 52.8±6.7 | 38.8±5.4 | 38.7±4.8 | 35.8±3.2 |
| Minute ventilation (l/min BTPS) | 107.2±10.4 | 112.9±9.2 | 112.4±12.4 | 116.4±15.3 | 121.7±15.9 | 121.9±9.3 |
| Rate of respiration (Breaths/min) | 42±5 | 45±4 | 44±5 | 46±5 | 45±5 | 44±6 |
| Maximum breathing capacity (l/min BTPS) | 134.4±18.1 | 132.0±16.9 | 138.1±13.4 | 130.2±18.6 | 135.7±14.9 | 134.6±14.8 |

per minute at sea level, 13000 and 16500 feet was 40%, 30% and 25% of the body weight respectively. This was against the actual requirements. Although at high altitudes one should carry less load yet per force he had to carry more because of the weight of winter clothing etc, required by him. Thus the only other factor which could be reduced was the speed of walking so that the rate of work done decreased at high altitudes. Nayar[31,32] studied load carriage at various altitudes on the basis of optimum rate of oxygen consumption. With the help of arterial oxygen saturation blood lactic acid, oxygen extraction percentage from inspired air and mechanical efficiency, it was possible to determine the optimum rate of oxygen consumption at different altitudes. From sea level to 2640 metres altitude, the optimum rate of oxygen consumption remained about one litre per minute (STPD). At 3350, 3950 and 4560 metre altitudes, the optimum rate of oxygen consumption decreased to 0.89, 0.84 and 0.74 litre per minute respectively. Table 6 shows the relationship between load carried and speed of marching at the cost of optimum oxygen consumption determined at various altitudes. Thus an infantry commander can select the necessary load-march relationship required for a particular military operation.

**Table 6.** Relationship between load carried and speed of marching at the cost of optimum oxygen consumption determined at various altitudes.

| Load Carried (Kg) | Speed of Marching at various altitudes : metres per minute | | | | | |
|---|---|---|---|---|---|---|
| | Sea level m | 1530 m | 2640 m | 3350 m | 3950 m | 4560 m |
| 16 | 98 | 98 | 97 | 89 | 85 | 67 |
| 22 | 95 | 93.5 | 94.5 | 87.5 | 83 | 59 |
| 28 | 91 | 90 | 87 | 83 | 80 | 54.5 |
| 34 | 88 | 87 | 84.5 | 72.5 | 68.5 | 50 |
| 40 | 85 | 84 | 81.5 | 67 | 63 | 45 |

## 5.3 *Cold and Altitude*

In a joint study Davis[33] *et al* measured the responses, of six Jat soldiers who had never been exposed to altitude and cold, five Jat soldiers who had eleven months residence at 12,000—17,000 feet altitude and five Tibetans who were born and raised at an altitude, against a standard cold exposure of 2°C for 60 min. both at high and low altitude. The Tibetans were not measured at low altitude. Both the Jat groups completed the requirements of the standard exposure test at low altitude without difficulty. At 13000 feet only two of the six low altitude acclimatised subjects completed the standard exposure test while four of the five high altitude accalimatised subjects completed the exposure test at high altitude. All the Tibetans completed the high altitude exposure test. The oxygen requirements of the Jat groups during exposure was higher at high altitude than at low altitude. Both high and low

altitude oxygen consumption of the high altitude acclimatised Jats was lower than those of the low altitude aeclimatised Jatss. The oxygen consumption of Tibetans was considerably lower than either of those of the Jat groups. As with the oxygen consumption, a similar pattern was demonstrated for shivering. During cold exposure rectal temperature of both Jat groups was higher at high altitude than at low altitude. Under the conditions of this study it was concluded that altitude induced an augmentation of the acute responses to cold. This was probably due to an impairment of non-shivering thermogenesis resulting in a dependence upon shivering thermogenesis and a decrease in cold tolerance. In a joint study, Gupta[34] *et al* studied the process of cold acclimatisation at 13000 feet above sea level. In these trials, unacclimatised subjects were exposed partly dressed to a temperature varying from 0° to—5°C for three hours every day. Positive evidence had been found that acclimatisation could occur if subjects did not do any physical activity during exposure and there was stimulus of shivering present throughout. Oxygen consumption, fall in skin and rectal temperature became static within three weeks. As it was intended that subjects shiver, this parameter although decreasing with progress of acclimatisation never reached to a static level upto the end of the trial. Although altitude had an adverse effect on process of cold acclimatisation, yet is was possible to induce cold acclimatisation by exposing subjects to a temperature of 0° to-5°C for period of three hours daily for three weeks.

### 5.4 *Caloric Requirements at High Altitudes*

Nishith[35] *et al* determined the caloric cost of 24 hours activities of a male healthy adult by indirect calorimetry and found it 3,253 K. Cal. at sea level and 4,286 K. Cal. at 13,000 feet above sea level. The caloric cost of each activity per minute had been found to be more at high altitude. The increase might be on account of more than one of the following reasons; which would modify the effect of each other as well, e. g. : lower temperature, high altitude (hypoxia and terrain), heavy clothes and heavy respiratory and circulatory effects. Inanother joint study, Nishith[36] *et al* worked out the caloric cost of 24 hours activities of a male healthy adult at 17000 feet above sea level and found it 4,380 K. Cal. The effects of cold and heavy clothes were not acting during this period of trial.

### 5.5 *Acclimatisation*

In recent years, acclimatisation to high altitudes has been the subject of many studies, inspite of this many aspects continue to be poorly understood. It consisted of many complex physiological compensatory adjustments which progressed with duration of exposure. These include hyperventilation, alveolar membrane diffusion, erythrocythemia and transient body fluid shifts. A person is said to be acclimatised to high altitude when he can sleep, eat and work well. The process of accli matisation begins upon arrival at high altitude. If the ascent to altitude is rapid and high, a proportion of the exposed population will suffer from symptoms of acute mountain sickness. The greater the change in altitude, the greater will be the proportion of

the group succumbing thereto, and intensity of symptoms. Disappearance of symptoms which occur in four to seven days, does not indicate complete acclimatisation. This process continues for months or years as evidenced by progressive improvement in physical performance. Selvamurthy[37] *et al* recorded EEG in healthy male adults during acclimatisation to high altitude. The EEG was recorded by the 10-20 international system of electrode placement in LL at sea level during rest and hyperventilation (HV). Then they were air-lifted to an altitude of 3500 metres where periodical recordings of EEG were made for four weeks. For comparison at high altitude, the recordings were also made in AL and HAN. The quantitative analysis of EEG (occipital) was done to determine alpha index (AI) and average amplitude. The mean AI was 25.5% at SL in LL which changed to 45.7%, 15.8%, 28.0%, 30.3% and 33.2% on days 2, 7, 14, 21 and 28 at high altitude respectively. The average amplitude was 17.3 $\mu V$ at SL which changed to 23.3 $\mu V$, 11.8$\mu V$, 16.2$\mu V$, 17.3$\mu V$ and 19.8$\mu V$ on days 2, 7, 14, 21 and 28 at high altitude respectively. In two subjects spiking was seen along with the desynchronised pattern of EEG on days 7 and 14. The AI and average amplitude of AL and HAN were significantly more than those of LL. The magnitude of build-up during HV was more at altitude. These results indicated that there was cerebral cortical depression in the initial phase of induction which changed to cortical desynchronisation in the later part of the first week of induction. During acclimatisation there was gradual build-up of EEG waves as observed in AL and HAN. Since there exists no means of shortening the processes of acclimatisation and since the absence of acclimatisation in new arrivals at high altitudes precludes the execution of jobs involving heavy physical performance, the deployment of troops at high altitude poses a serious problem. Troops rapidly transported to high altitudes from much lower ones cannot be committed immediately to patrolling, entrenchment, combat or other physically taxing duties. Present state of knowledge therefore dictates a deployment which makes for some degree of acclimatisation prior to heavy physical duties. Two possibilities appear practical:

5.51 (a) *Slow ascent*

Staged movements of sea level troops with 2-3 days acclimatisation stops at a series of intermediate altitudes e. g. : 2500 metres, 3500 metres and then 4500 metres.

5.52 (b) *Rapid ascent*

Alternately, troops might be moved directly to 3500 metres with allowance for their illness and relative inactivity for the first week of residence.

5.6 *Maladaptation*

There are three main diseases which are due to maladaptation to high altitude, viz., acute mountain sickness, high altitude pulmonary oedema and chronic mountain sickness. The exact etiology of these disorders is unknown, but the basic cause is fall in atmospheric $PO_2$ since all the three are cured when the person is brought at the sea level atmosphere. A moderate decrease in the oxygen content of inspired air

caused an increase in the pulmonary arterial pressure both in animals and human beings[38,39,40]. Kabins[41] suggested that 5-HT could be the initiating factor in the neurohumoral mechanisms involved in pulmonary arteriolar and venous constriction. Sackner *et al*[42] showed that alveolar hypoxia and 5-hydroxy-tryptamine (5-HT) produced a form of pulmonary vasoconstriction in which there was increased smooth muscle tone of a major portion of the pulmonary arterial tree. Nayar *et al*[43]. undertook estimation of 5-HT concentration in right ventricular (RV) and carotid arterial (CA) blood in eleven mongrel dogs prior to and during 45 minutes of breathing 10 per cent oxygen in nitrogen. Acute hypoxia was not associated with any significant increase in the levels of circulating 5-HT. During ambient air breathing in a group of another 8 dogs, the level of circulating 5-HT had to be increased at least 15 times above control levels in order to duplicate the rise in RV pressure due to acute hypoxia. Depletion of the pulmonary vascular nerve endings of 5-HT by reserpine in a set of another 6 dogs, did not prevent the usual rise in RV pressure due to acute hypoxia. Thus the study provided no evidence of a role of 5-HT in the pulmonary arterial pressor response to acute hypoxia of moderate degree. Hegde *et al*[44] estimated alveolar macrophages and pulmonary surfactant in rats raised at high altitude (3500 metres) and compared with control rats. Macrophages were obtained by washing the lungs with isotonic saline and counted in haemocytometer to obtain the total number present. Proportions of two types of macrophages (precursor and mature) were evaluated. Different fractions of phospholipids in lung lavage and lung tissue were separated by thin layer chromatography and estimated calorimetrically. The results showed a reduction in the number of macrophages/g of lung with a corresponding decrease in surfactant. The percentage of immature (precursor) macrophages were lower in high altitude raised rats. These changes observed in alveolar macrophages and lung surfactant could be due to an altered metabolism at high altitude. Selvamurthy *et al*[45] conducted experiments on 200 adult rats of either sex, body weight ranging from 160 to 370 g to evaluate the role of hypothalamus in the modulation of hypoxic tolerance and in the development of high altitude pulmonary oedema (HAPO). The lesioning, electrical stimulation and electrophysiological recording techniques were used in the study with the aid of stereotoxic apparatus. It was observed that hypothalamus through its control on autonomic functions played a significant role in the modulation of hypoxic tolerance. The anterior hypothalamic lesion decreased hypoxic tolerance while the lesion in the posterior hypothalamus resulted in enhancement of hypoxic tolerance. These effects were associated with the shift of autonomic equilibrium either towards sympathetic dominance or parasympathetic preponderance as in the case of lesions in anterior or posterior hypothalamus respectively. The 'Edemagenic and Anti-edemagenic' areas located respectively in posterior and anterior hypothalamus were associated with the development of high altitude pulmonary oedema (HAPO) in rats. These hypothalamic areas bring about their pulmonary effects through autonomic nervous system. Selvamurthy *et al*[46] conducted experiments on 20 high altitude natives (HAN-1) to evaluate the changes in autonomic responses during their 2-month sojourn in the plains, and on return to high altitude. The autonomic indices measured were

heart rate (HR), blood pressure, oral temperature, mean skin temperature, respiratory rate, cold pressor response, HR-response to tilt of 70° and alpha index (AI) of EEG occipetal). These indices were recorded periodically during the 2nd month of their stay in the plains and thereafter at altitude for one month after their return. For comparison, the same responses were studied on 10 LL in the plains and on induction to HA alongwith HAN-1. The study was repeated at altitude, once on 10-HAN who had never been to the plains (HAN-II) and on 10 AL. The results suggested that the relative parasympathetic dominance observed in HAN-1 showed a gradual decrease during their sojourn in the plains, probably due to the elevation in sympathetic activity. On return to high altitude, HAN-1 showed further increase in sympathatic excitation, as abserved in LL on acute induction, but the magnitude of this response was less in HAN-1. However, they showed a trend to a faster return of autonomic responses, towards those of HAN-II whereas LL lagged behind in this recovery response as observed in AL who had some of autonomic responses elevated even after one year of stay at altitude. Mathew *et al*[47,48] attempted to find out the role of chemoreceptor sensitivity in the causation of maladaptation syndromes on acute exposure to high altitude. The experiments were done in two phases. In phase 1, the response in chemoreceptor sensitivity was studied in subjects who had acclimatised well at high altitude and compared with those who suffered from either HAPO or AMS. In Phase II, a similar comparison was done in two groups of subjects, one representing normal sojourners at 3500 metres and the other being subjects who had just recovered from HAPO. The first phase was done at Delhi, and the second at an altitude of 3500 metres. The results showed significantly lower sensitivity to both hypoxia and $CO_2$ in maladapted subjects, as compared to those who were well acclimatised in both the categories suggesting thereby that reduced chemorecptors sensitivity might be an initiating factor in the causation of maladaptation syndromes at high altitudes.

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