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PROBLEMS OF HIGH ALTITUDE

(जुलाई, 1997 में जारी)

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(Case No. 12256/DGAFMS/DG-3C)

INTRODUCTION

1. High altitude (HA), with its attendant hypobaric hypoxia and cold, results in certain stresses to the human body. To overcome these adverse effects of the environment, certain physiological changes occur which, if successful, leads to acclimatization of the individual. Failure, on the other hand, leads to the various maladies of high altitude. Apart from hypoxia and cold, other factors at HA which could affect performance and lead or contribute to illness include low humidity, solar and ultraviolet radiation. However, hypoxia remains the single most important challenge to the system and is responsible for the various life-threatening problems that may occur. With the induction of troops to extreme altitudes, these problems have become compounded and new problems have arisen.

Definitions

2. *High Altitude.* Altitudes above 2700m (9000'). However, HA-related problems may start at lower altitudes and may be seen as low as 2500m (approx. 8000').

3. *Extreme Altitude.* Altitudes beyond 5500—5800. (i.e. 18000—19000'). Though man can survive at such altitudes for short periods, successful and permanent acclimatization cannot occur.

4. *Acclimatization.* This involves reversible changes in the anatomy or physiology of the body which enables the organism to survive in an alien environment (HA).

PHYSIOLOGY OF ACCLIMATIZATION

5. Oxygen, which is vital for the survival of the cell, travels from the atmosphere to the tissues down its pressure gradient. This involves ventilation whereby oxygen from the atmosphere reaches the alveoli, diffusion from the alveoli to the pulmonary capillary blood, oxygen transport in the blood from the lungs to the periphery and, finally, diffusion of the gas across the cell membrane to reach the mitochondria. At each of the above steps, oxygen transport is dependant on its partial pressure (PO.).

6. At sea level (SL), the barometric pressure is 760 mm Hg; since the oxygen content of the atmosphere is 20.9% (irrespective of the altitude), the partial pressure exerted by atmospheric oxygen at SL is 160 mm Hg (20.9% of 760 mm Hg. In the airways the inspired air is humidified; since the partial pressure of water vapour is 47 mm Hg, the PO_2 of inspired air is approximately 150 mm Hg i.e. 20.9% of (760 - 47). As the inspired air reaches the alveoli, it gets diluted and the PO_2 falls to 100 mm Hg. The pulmonary arterial PO_2 is 5—15 mm Hg (depending on age) less than alveolar PO_2 and the PO_2 of mixed venous blood is 40 mm Hg. Thus, at SL, Oxygen flows down its pressure gradient from 160 mm Hg in the atmosphere to 100 mm Hg in the alveoli and finally to 40 mm Hg in mixed venous blood. This is also known as the "oxygen cascade".

7. As opposed to SL, at HA this slope of the oxygen cascade is less steep. For example, at a height of 3000 m, the barometric pressure is 510 mm Hg. Therefore, at this altitude, the atmospheric or ambient PO_2 will be 106 mm Hg. Similarly, at 5500 m (extreme altitude), the barometric pressure is half that of SL and the PO_2 here would be 20.9% of 380 or around 80 mm Hg. With this there would be a corresponding fall in the alveolar and arterial PO_2s. However, the PO_2 of mixed venous blood does not fall much and this is responsible for the fall in the steepness of the slope with increasing altitude (Fig. 1). The minimal fall in mixed venous PO_2 despite a considerable difference in atmospheric PO_2 suggests that a HA there is increased extraction of oxygen by the tissues.

8. To overcome this fall in PO_2 with fall in barometric pressure at HA (termed 'hypobaric hypoxia'), changes occur at various sites along the chain (lungs to the tissues) in order to reduce the impact of this hypoxia at the cellular level and these changes constitute the process of acclimatization.

Hyperventilation

9. The earliest change that occurs on ascent to HA is hyperventilation. The hypobaric hypoxia of HA leads to a fall in the arterial PO_2 which stimulates the chemoreceptors in the carotid bodies and aortic arch, this, in turn, stimulates the respiratory centre resulting in hyperventilation. At mild to moderate HA, this hyperventilation is caused by an increase in tidal volume without much change in the respiratory rate while at extreme altitudes there is probably also an increase in respiratory rate. The increased ventilation washes out the CO_2 from the alveoli and increases the alveolar PO_2 thus maintaining a relatively high PO_2 in the alveoli in the face of a low atmospheric PO_2 at HA. This hyperventilation caused by hypoxia is known as the 'Hypoxic Ventilatory Response' (HVR), and a good HVR is a requirement for proper acclimatization. The hyperventilation also leads to hypocapnia (due to CO_2 washout) and respiratory alkalosis and this is compensated for over the next days by renal excretion of bicarbonate.

Pulmonary Vasoconstriction

10. The alveolar hypoxia at HA also leads to pulmonary vasoconstriction. The exact beneficial role of this response is uncertain apart from opening up closed vessels and making the perfusion of the lungs more uniform. On the other hand, this pulmonary vasoconstriction has been implicated in the genesis of high altitude pulmonary oedema.

Polycythaemia

11. This develops more slowly. The resultant rise in RBC mass and haemoglobin concentration increases the oxygen carrying capacity of the blood at HA. At extreme altitudes, this change occurs more rapidly. Although this polycythaemia is valuable, it also leads to an increase in viscosity of blood, which, if excessive, can be deleterious.

Shift in the O_2 Dissociation Curve (Fig. 2)

12. This curve is 'S' shaped, which has several advantages. At SL, alveolar PO_2 is high and falls in the upper flat portion of the curve so that a fall in alveolar PO_2 will not cause a significant fall in the O_2 saturation. The steep portion of the curve suggests that in the periphery (capillaries) where the PO_2 is lower, a small fall in the PO_2 will lead to a relatively greater drop in the O_2 saturation. This means that at the tissue level, oxygen can be unloaded from the blood for only a small decrease in PO_2.

13. At HA, the increased haemoglobin concentration leads to an increase in the oxygen carrying capacity of the blood (see above). However, this would be useful only if the same oxygen can also be readily delivered to the tissues (unloaded). The latter can be achieved by a shift of the dissociation curve to the right (curve 'B' in Fig. 2). This would mean that at a given PO_2 the oxygen saturation is less i.e. it has been unloaded or delivered. This change does occur at HA and the rightward shift of the curve is caused by a rise in red cell 2, 3, diphosphoglycerate (in turn caused by the hypoxia and alkalosis). However, this increased unloading at the periphery is at the cost of decreased loading of oxygen in the lungs and may be harmful at extreme altitudes where there is severe hypoxia which would necessitate increased loading. This means that at extreme altitude a leftward shift would be more beneficial. Such a shift of the curve to the left does occur in animals indigenous to HA.

Other Changes

14. These include an increase in sympathetic activity leading to a rise in heart rate, a redistribution of blood from the periphery to the vital organs, increase in the number of capillaries in the periphery and changes in intracellular oxidative enzymes.

TEMPERATURE REGULATION

15. Cold is a feature of HA and there is, on an average, a drop of 1°C for an ascent of 150 m. Heat loss at HA occurs in three ways :—

(a) by convection in which there is transfer of body heat to the cold environment, a process aggravated by wind

(the 'wind-chill factor') since wind blows away the protective layer of warm air surrounding and insulating the skin;

(b) by conduction where heat is lost by contact with cold objects such as cold, bare metal, ice and cold stones on the mountainside;

(c) by evaporation—there is a significant amount of heat lost from the respiratory tract and skin by evaporation at HA due to the low humidity which is common.

16. On exposure to cold, the primary aim of the system is to preserve the core body temperature. This is achieved by increasing heat production by shivering and involuntary motor activity as well as by decreasing heat loss by peripheral vasoconstriction. When the body temperature regulating mechanisms fail, there is lowering of the core body temperature leading to hypothermia.

CLASSIFICATION

17. There is as yet no universally accepted classification for the medical problems of HA. These problems include the well known HA illnesses as well as certain problems seen at SL but which, in our experience, are common at HA. Not much study has been carried out in the latter group. A tentative classification for the medical problems of HA is given below :—

I. High Altitude Illnesses :

A. Due to abnormal response to the process of acclimatization :—

(i) *Acute*. Acute Mountain Sickness, High Altitude Pulmonary Oedema and High Altitude Cerebral Oedema.

(ii) *Sub-Acute*. Pulmonary Arterial Hypertension of HA (at extreme altitudes).

(iii) *Chronic*. Chronic Mountain Sickness and Pulmonary Arterial Hypertension of HA (at mild to moderate altitudes).

B. Unrelated to Acclimatization :

(i) High Altitude Retinopathy, Thrombotic episodes.

II. Effects of Cold :

Hypothermia, Local cold injury.

III. Other Problems at HA :

(i) Hypertension at HA.

(ii) Gastrointestinal Problems.

(iii) Infections at HA.

(iv) Cutaneous Reactions.

(v) Others.

PROBLEMS OF HIGH ALTITUDE

HA Illnesses Related to Acclimatization

18. These can be further classified into acute (onset within a few hours to few days of induction), subacute (onset within few weeks to months) and chronic (onset in months to years). The acute illnesses show an individual predisposition and is related to the speed of induction, being less common among inductees by road than air inductees. These illnesses (acute) are considered to be at different points in the same spectrum of disease.

19. On ascent to HA, certain symptoms occur which are due directly to hypobaric hypoxia and the attendant physiological changes of acclimatization. These include awareness of ones breathing, palpitations due to increased heart rate and breathlessness on exertion. Such symptoms are more common in the timid and are of no significance and should not be confused with the syndrome of acute mountain sickness.

Acute Mountain Sickness (AMS)

20. This is the commonest manifestation of HA illness and lies at one end of the spectrum which extends at the other end to the life-threatening conditions of high altitude pulmonary oedema and high altitude cereberal oedema. It develops usually after a lag period of 6—96 hours but may occur immediately on induction to HA and the onset is most often during the first day. The pathogenesis of this disorder is unclear but seems to be due to retention of fluids or to a shift of fluid from the intracellular to the extracellular compartments especially in the brain and lungs. It has been observed that patients of AMS give a history of oliguria prior to the onset of disease as opposed to the diuresis which occurs in healthy inductees. Individuals susceptible to AMS have been shown to have a blunted hypoxic ventilatory response (see above) and so have lower arterial PO_2s; this could affect the endocrine system (particularly ADH and the renin-aldosterone systems) leading to fluid retention. In addition, hypoxia causes cerebral vasodilatation and increased cerebral blood flow, which cou'd also explain the symptoms of AMS.

21. *Clinical Features.* Dickinson (1982) has classified AMS into benign and malignant forms, the latter including the pulmonary and cerebral oedemas of HA. This has the advantage of providing a rational basis for the management of the illness by lay personnel. The exact incidence of AMS is not known, but it is fairly common; the milder cases do not report sick and hospital admissions for AMS probably form the tip of the iceberg. An incidence as high as 53% has been quoted. All ages are susceptible and a susceptible individual often develops symptoms during each induction. The largest series to date on the clinical aspects of AMS is that of Inder Singh et al in 1969 on 1925 Indian soldiers.

22. In the mildest form of AMS, there is headache. This is usually bilateral, frontal, throbbing, aggravated by exertion and more in the mornings on waking. As the disease progresses, the headache becomes more severe and no longer responds to analgesics. Other symptoms include malaise, lassitude, disinclination to work, loss of appetite, nausea, vomiting, shortness of breath on exertion and disturbed sleep. Untreated, this may resolve or may progress to high altitude cerebral oedema or high altitude pulmonary oedema (see below). In the majority the symptoms of AMS resolve by the end of the first week, though occasionally they may persist for longer (several weeks).

23. *Treatment*. In mild cases treatment consists of reassurance, rest and analgesics. In more severe cases or those with persistent symptoms, acetazolamide (diamox) has been found to be useful in a dose of 250 mg 6 to 8 hourly for 2 days. Apart from producing diuresis, ingestion of this drug leads to a state of metabolic acidosis which stimulates respiration and so causing respiratory alkalosis—a state of acid base balance seen in acclimatized individuals. Similarly, dexamethasone (8 mg stat followed by 4 mg every 6 hours for 3 to 4 doses) is effective in ameliorating the symptoms of AMS. The use of steroids is based on the premise that cerebral oedema is responsible for the symptoms of AMS. These drugs may be given with or without oxygen supplementation. If pulmonary or cerebral oedema develops, the treatment will change accordingly.

24. Mild cases do not require hospitalization but merit frequent examination by the RMO to detect early signs of the malignant pulmonary or cerebral complications. On recovery from AMS, the individual is fit to continue serving in HA.

High Altitude Pulmonary Oedema (HAPO)

25. This is the commonest cause for admission among the HA illnesses and is potentially life-threatening but, if treated on time, can recover almost completely. It has been recognized as a distinct entity by the western world only in the last 30

years, prior to which cases were being diagnosed and managed as pneumonias.

26. The predisposing factors for HAPO include rapid ascent, cold, re-entry and exertion. It is more common among air inductees than road inductees, probably because road induction, which entails a travel of a few days, partially acclimatizes the individual. Re-entry to HA is an important factor as the majority of cases are seen amongst reinductees; it usually occurs after a minimum stay of 10 days in the plains though cases have been recorded after just one day's absence from HA. There is a definite individual predisposition to the illness. While exertion can precipitate HAPO in a susceptible individual, it is not essential and the majority of patients do not give a history of having engaged in strenuous exertion prior to the onset of illness. The role of preceding upper respiratory infection is not clear.

27. *Pathophysiology*. Invasive studies during HAPO have shown high pulmonary arterial pressures with normal pulmonary wedge and left atrial pressures; this suggests that the oedema is not due to left heart failure. Analysis of the oedema fluid after bronchial lavage has shown it to have a high protein content as well as red blood cells and macrophages, which point towards a permeability oedema i.e. oedema caused by increased vascular permeability. The cause of this remains obscure. Susceptible individuals show a heightened responsiveness of the pulmonary vasculature to hypoxic challenge. It is postulated that the high pulmonary artery pressure caused by hypoxic pulmonary vasoconstrition accompanied by the increased pulmonary blood flow due to redistribution of blood from the periphery is responsible for HAPO. The vasoconstriction is non-uniform leading to overperfusion in some areas of the lungs and underperfusion in others, which explains the patchy distribution of oedema seen radiologically. The increase in pulmonary artery pressure in these individuals at HA is further accentuated by exercise and, therefore, exertion can precipitate HAPO in susceptible persons. After prolonged stay at HA, there is an increase in blood volume due to increased RBC mass; on deinduction, there is a fall in the RBC mass and a compensatory increase in plasma volume.

When such an individual is reinducted to HA, the increased plasma volume further increased pulmonary blood flow and could explain the higher incidence of HAPO amongst reinductees.

28. *Pathology*. The pathology of the lungs in patients dying of HAPO shows grossly oedematous lungs which do not float in water. Histologically, there are dilated pulmonary capillaries and alveolar oedema. Western workers have also reported the presence of hyaline membrane lining the alveoli and thrombi in the pulmonary arteries with pulmonary infarcts. Superadded bronchopneumonia may be present but has been rare in our series. The majority of fatal cases show the presence of cerebral oedema in addition.

29. *Clinical Features*. Like in AMS, the largest series of HAPO described in literature have been from the Indian Armed Forces by Inder Singh et al (1965) and Menon (1965). The incidence of HAPO has been estimated to be between 0.4—6.1%. The majority of patients in the armed forces are in the third decade of life (i.e. between 21—30 years) though no age is immune. Even children, females and native highlanders are susceptible.

30. The vast majority of cases occur between 2700—3700 m Apart from induction to HA, HAPO can occur in an acclimatized individual who ascends further by as little as 300 m. It is less common at extreme altitudes probably because an individual gets acclimatized as he proceeds to such altitude by the land route. The onset of illness is within seven days of arrival at HA in most with the majority occurring within the first three days; however, this may not be necessarily true at extreme altitudes where this condition has been seen occurring many days after induction to that height. In such cases, pheumonias and pulmonary embolism need to be considered in the differential diagnosis.

31. The clinical picture usually starts with symptoms suggestive of AMS. Thus there is headache soon after arrival at HA and this is followed by bodyache, cough, breathlessness on exertion which is progressive, non-anginal chest pain, anorexia, disturbed sleep, vomiting and giddiness. Cough is usually accompanied by mucoid expectoration and there is often haemoptysis. At times, fever may be the presenting symptom. In severe cases there may be associated symptoms of cerebral oedema.

32. On examination, the individual appears ill and is tachypnoeic with respiratory rates going upto 60 per minute in severe cases. Tachycardia is almost invariably present and is a fairly reliable guide to the severity as well as to the response to therapy. In mild cases, the pulse may be upto 120 per minute while in severe cases it may go upto 150—160 per minute. Mild fever is frequent. The presence of central cyanosis indicates severe disease. The optic fundi show venous engorgement and retinal haemorrhages may be seen in around 10% of cases, and this is more common in cases occurring at extreme altitude. The presence of papilloedema indicates associated cerebral oedema. The blood pressure is normal in all but the critical cases in whom it may show a fall. The administration of large doses of lasix also leads to a drop of blood pressure. The JVP is not raised and there are no signs of congestive failure. On auscultation of the chest, there are usually bilateral medium to coarse crepitations and rhonchi are uncommon; fine crepitations may be heard in very early cases as well as during recovery. The right side is usually more involved than the left, but any distribution may be seen. In mild cases crepitations are heard in the interscapular and infrascapular areas only but, at times, anterior involvement may be more marked; hence a detailed auscultation of the entire chest should be carried out in all cases. There is no cardiomegaly but evidence of pulmonary arterial hypertension is present in almost all with an accentuated pulmonary component of the second sound, with or without an ejection sound.

33. Investigation usually reveals a mild to moderate polymorphonuclear leucocytosis, though occasionally counts of 30000/cmm may be seen in severe cases. Despite fever and leucocytosis, there is no infection in the majority and so no indication for antibiotics. Radiologically there is bilateral but asymmetrical involvement of the lungs with the right side being more often involved than the left; unilateral lung involvement does occur but is less common. The mid and lower zones are more commonly affected than the upper zone and medial more than lateral lung fields. The density of the lesions varies from soft to dense with the former being more frequent. The lesions are patchy and all types of shadows may be seen including non-homogenous, mottled, straeky, cotton wool or confluent lesions. Pleural effusions, contrary to earlier reports, are very rare. The cardiothoracic ratio is within normal limits and this serves to differentiate it from cardiogenic pulmonary oedema. The main pulmonary artery as well as the right and left pulmonary arteries are dilated and prominent. Kerley lines are not seen. There is no relationship between the clinical severity and the extent of radiological involvement. The electrocardiogram shows sinus tachycardia and a rightward axis and often inverted T waves in the right-sided precordial leads. Other findings which may be seen include a qR pattern in aVR, S waves in V_5, V_6 and P pulmonale. Occasionally a normal graph may be seen apart from the tachycardia.

34. *Diagnosis.* The diagnosis is based on the temporal profile of the onset of illness in relation to induction to HA and the clinical findings of tachycardia, tachypnoea, and bilateral lung involvement. A high degree of suspicion is essential and any case presenting with the above signs and symptoms within the first week of induction should be taken as HAPO unless proved otherwise. The main differential diagnosis include

pneumonia (in view of the fever and leucocytosis), pulmonary embolism and cardiogenic pulmonary oedema. Pneumonia is ruled out by the bilateral involvement. The absence of deep vein thrombosis, the subacute onset and the absence of pleuritic chest pain as well as the ECG help to exclude pulmonary embolism. The history and the absence of orthopnoea, cardiomegaly and gallop sounds and the ECG reasonably rule out a cardiogenic cause for the pulmonary oedema.

35. *Treatment.* Ideally, the patient should be deinducted; however, this is not usually possible in our setting. If HAPO occurs at a post, evacuation to a lower height helps in the management. The mainstay of threapy is oxygen therapy. Oxygen should be given freely and at high flow rates (of at least 8 L/minute) with a face mask to prevent rebreathing. The use of nasal catheters to administer oxygen is not of much value. In case of shortage of the gas, which is likely in the periphery, lower flow rates may be given. However, there should be provision for the admixture of atmospheric air in such cases by interposing a reservoir bag between the mask and the cylinder with an outlet to the atmosphere. This is essential as the patient is tachypnoeic and has a high minute ventilation. In mild to moderate cases this usually suffices and the patient reports subjective improvement within an hour or two and this is corroborated by the fall in the pulse rate. Oxygen therapy should be continued till the patient becomes asymptomatic and the pulse falls below 100/min and irrespective of the presence of crepitations or of the radiological findings, which tend to lag behind. On an average, this means 12—24 hours of oxygen. In severe cases, frusomide (lasix) may be given. When required, 20—40 mg given intravenously usually suffices and larger doses are not recommended and generally not more than 2 to 3 doses (8 hourly) are required. Morphine, bronchodilators and antibiotics are not indicated in the routine case. In severe cases, morphia (5 mg intravenously, slowly) may be given. Deriphyllin has no role and antibiotics can be safely withheld for the first 2 to 3 days when the picture becomes clearer. Where

available, a recompression chamber can be used in severe cases; the patient is placed in the chamber and the usual duration of recompression is about 24 hours. If there is evidence of cerebral oedema, this is managed accordingly.

36. Though there is subjective and objective improvement within a few hours of initiating treatment, radiological clearance takes longer and complete resolution takes about a week. Evidence of pulmonary arterial hypertension on auscultation and in the X-Ray may persist for longer.

37. *Disposal.* All cases should be sent on six to eight weeks sick leave and thereafter reviewed by a medical specialist. In view of the likelihood of recurrence in an individual who has had an episode of HAPO, such individuals should be made unfit for service at HA. Downgradation of the medical category is not necessary as long as re-entry to HA can be avoided. 'Unfit for HA' should be endorsed in the individual's pay book AB 64/Officers' Health Card.

38. Clinical evidence of pulmonary arterial hypertension and radiological evidence of a prominent main pulmonary artery in these patients after recovery correlates poorly with invasive or echocardiographic evidence of pulmonary hypertension and so should not be used as a basis for deciding his medical category.

High Altitude Cereoral Oedema (HACO)

39. This is the most dreaded of all the high altitude illnesses; fortunately, it is also the least common. In Inder Singh et al's series (1969) only 4 of the 1925 cases of AMS revealed frank papilloedema.

40. *Pathophysiology.* The pathophysiology of this condition is similar to that of AMS with fluid retention due to secretion of ADH and activation of the renin-angiotensin-aldosterone system along with increased cerebral blood flow.

41. *Pathology*. The brain in fatal cases shows increased weight and flattening and swelling of gyri and narrowing of the sulci. There are punctate as well as larger haemorrhages particularly in the white matter and the corpus callosum. Western workers have also reported the presence of subarachnoid haemorrhages, areas of haemorrhagic infarction and thrombosis in the dural venous sinuses. These changes indicate that recovery may not always be total since some of the changes may be irreversible, and there may be residual deficits.

42. *Clinical Features*. The onset is with AMS and so headache, as a sign, is of no importance where the diagnosis of HACO is concerned. Isolated HACO is very uncommon, and it is usually seen in combination with HAPO. Alteration in the level of consciousness is the most important feature in the diagnosis. The patient may complain of dimness of vision, dizziness, vomitting and may progress to stupor and coma. On examination, apart from the level of consciousness, there may be focal deficits in the form of cranial nerve palsies (Particularly the VI and the VII cranial nerves), hemiparesis, abnormal plantar reflexes and alterations in muscle tone. The optic fundi show blurring of disc margins and there may be frank papilloedema. Though papilloedema is diagnostic, it is not essential for the diagnosis, as cases may have changes in the level of consciousness but without frank papilloedema.

43. *Management*. Rapid evacuation to a lower altitude is the most important aspect of management. Thereafter oxygen given by mask (as for HAPO) or a recompression chamber, if available, should be initiated. Decongestive measures should be simultaneously started including parenteral steroids and mannitol and oral glycerol. Frusomide (lasix) may be given, but in small doses in view of the recent description of dural venous sinus thrombosis in HACO, since dehydration may

favour thrombus formation. The prognosis in severe cases is poor and is related to intracerebral haemorrhages and/or dural venous sinus thrombosis. The disposal is as for HAPO.

Pulmonary Arterial Hypertension of High Altitude

44. This condition was originally described by Indian Army physicians in the latter half of the 1960s, but was not well recognized till recently. In 1988, a similar condition affecting infants in Tibet was reported as 'Infantile Subacute Mountain Sickness'. Thereafter (in 1990) a syndrome of congestive cardiac failure due to high altitude pulmonary arterial hypertension was reported among Indian soldiers serving at extreme HA as 'Adult Sub-acute Mountain Sickness' (1990).

45. This syndrome consists of features of right heart failure consequent to pulmonary arterial hypertension and is akin to Brisket Disease of cattle. In the original description, the onset of the disease was after 5—42 months of stay at mild to moderate altitudes and hence classified as chronic. At extreme altitude, the onset is much earlier (8—12 weeks after ascent to these altitudes) and is, therefore, classified as sub-acute. This earlier onset at extreme altitude is probably due to the greater hypoxic stress.

46. The exact pathogenesis of this disorder is not known. Pulmonary arterial hypertension is a normal phenomenon at high altitude due to the hypoxic pulmonary vasoconstriction. However, when such hypertension is severe in susceptible individuals (hyperreactors to hypoxic stress) it can lead to symptoms and finally to cardiac failure.

47. The possibilities in the pathogenesis of this disorder include pulmonary vascular obstruction since hypoxic vasoconstriction alone cannot explain the pulmonary hypertension as the administration of oxygen does not reverse it completely. At the same time, recovery is almost complete within a few weeks to months of deinduction to the plains. In the original description in 1965 by Inder Singh et al. autopsy showed multiple thrombi in the pulmonary vasculature and they postulated that breakdown of the fibrinolytic system may play a role. In necropsy studies in the Tibetan infants, there was

severe right ventricular hypertrophy and a dilated pulmonary trunk along with increased muscularization of the pulmonary arteries, arterioles and venules; in the venules the proliferating myofibroblasts may bulge into the lumen, thereby compromising it. Failure to acclimatize was postulated as a cause for these features.

48. Clinically, the onset is usually with effort intolerance, anginal chest pain, haemoptysis and swelling of the feet and face along with diminution in the urine output. At extreme altitude, this usually starts after a period of relatively strenuous exertion e.g. carrying loads uphill ('link duties'). The breathlessness on exertion is gradually progressive and an occuasional patient may give a history suggestive of paroxysmal nocturnal dyspnoea. On examination, there is evidence of congestive cardiac failure with a raised JVP and prominent 'a' waves in the venous pulse, pedal oedema with facial puffiness and, at times, generalized anasarca and a tender hepatomegaly. There is evidence of pulmonary arterial hypertension with a loud pulmonary component of the second sound, an ejection sound and, rarely, a murmur of tricuspid regurgitation. There may be evidence of pleural and pericardial effusions as well as ascites. There is usually polycythaemia and the urine examination does not reveal any abnormality. The ECG shows right axis deviation, right ventricular hypertrophy and deep, symmetrical T inversions in the right sided precordial leads. The X-Ray of the chest shows cardiomegaly with a prominent main pulmonary artery. Pleural effusions may be seen.

49. The management consists of evacuating the patient to lower altitudes. It has been seen that as little as a descent of 500 m results in diuresis and amelioration of symptoms. All cases should preferably be evacuated to the nearest service

hospital. In case the individual cannot be evacuated early, small doses of oral frusemide (lasix) may be given (40 mg once a day) along with potassium supplements. In hospital, conservative management with only rest results in remarkable recovery. Diuretics are rarely required and digoxin is not indicated. All cases should be finally deinducted to the plains. They may be initially sent on a spell of sick leave and thereafter placed in low medical category till cardiomegaly in the X-Ray and ECG right ventricular hypertrophy subsides.

Chronic Mountain Sickness (Monge's Disease)

50. This disease was first described by Monge in 1928 from the South American Andes and, till recently, was thought to be non-existent in the Himalayas. In 1982, Nath first described this syndrome from the Western Himalayas and in 1989, it was reported from Tibet. In both these reports, chronic mountain sickness was seen in low landers who had stayed at HA for prolonged periods.

51. Chronic Mountain Sickness (CMS) is seen in young or middle-aged men, particularly in smokers and has not been described in children and is rare in women. The long latent period of 15—20 years or more before the manifestations of the disease appear explains the absence of the disease in children, and the regular menstrual loss of blood is probably why it is uncommon in females.

52. Three types of CMS have been described. In the first, a lowlander who ascends to HA shows a chronic lack of adjustment to HA; in the second, a highlander who has acclimatized, develops a respiratory disease which exaggerates the hypoxaemia (such as emphysema, gross obesity, pneumoconiosis, etc)—conditions which can cause chronic hypoxia even at sea level and hence this type has been called 'Secondary CMS'. The third type is a native highlander or a lowlander who has successfully acclimatized and develops symptoms of CMS without any pulmonary disease—this has been termed 'True Monge's Disease'.

53. CMS has been considered to be due to loss of acclimatization in a previously acclimatized individual. These patients have an increased blood volume, pulmonary arterial hypertension and a high haematocrit. It has been postulated that these individuals develop a loss of sensitivity of their respiratory centres to CO_2 leading to a loss of respiratory drive and so to alveloar hypoxia. This hypoxia, in turn, results in polycythaemia which is responsible for most of the symptoms. So far there has been no autopsy reported on an unequivocal case of true Monge's disease.

54. The dominant and early symptoms are referable to the central nervous system with headache, somolence, loss of memory, dizziness, paraesthesias and neuropsychiatric symptoms. Other symptoms include effort intolerance, bleeding manifestations such as epistaxis, haemoptysis and purpura and, later, symptoms of mild cardiac failure. The majority of these patients are smokers. On examination, they have a plethoric, florid facies, suffused, dark red conjunctivae, cyanosis, clubbing and there may be macroglossia and signs of pulmonary arterial hypertension and congestive cardiac failure. A mild rise in systemic blood pressure is common. Haemorrhage under the finger nails has been considered to be characteristic. Investigations show a high haemoglobin and haematocrit and often there is gross proteinuria. The X-Ray shows an increased cardio thoracic ratio, with a prominent main pulmonary artery and the ECG may show right atrial enlargement, right axis deviation and right ventricular hypertrophy.

55. Treatment consists of evacuation to the plains where the symptoms and signs disappear over a period of time. Cases diagnosed should be referred to the High Altitude Medical Research Centre, 153 GH or to Army Hospital, Delhi Cantt for further studies.

HA ILLNESS UNRELATED TO ACCLIMATIZATION

High Altitude Retinopathy (HAR)

56. Apart from the retinopathy associated with papilloedema in HACO, retinal haemorrhages may occur at HA unrelated to papilloedema. This is being increasingly recognized now and its importance lies in the fact that these changes which are visible through the ophthalmoscope may

reflect the vascular responses in the vital organs such as the brain. The incidence increases with increase in altitude and so will be increasingly seen with troop deployment at extreme altitudes.

57. The exact incidence of retinal haemorrhages at HA is not known but about a third to a half of all those exposed to heights above 5000m are likely to develop it, but there is no threshold in altitude for the development of this complication. The exact cause is not clear; there is an increase in retinal blood flow with vasodilatation and this may play a role. In addition, sudden surges in blood pressure on exertion may aggravate or precipitate ratinal haemorrhage.

58. Retinal haemorrhages at HA are more common in the central fundus and usually spare the macula so that vision is not affected. The haemorrhages may be diffuse, punctate or flameshaped and are usually associated with retinal venous engorgement, venous tortuosity and disc hyperaemia, which are the normal changes seen on ascent to HA.

59. Retinal haemorrhages have usually been detected on routie examination since the patient is asymptomatic. These haemorrhages resolve spontaneously and completely over a few days to weeks and so do not require any active treatment. The individual should be reassured and the haemorrhages followed up till they are absorbed.

60. 'Cotton Wool' or soft exudates are very rare but have been described at HA and are unrelated to any systemic disease.

Snow Blindness

61. This condition is less common than is believed and is due to damage of the corneal epithelium caused by exposure to ultraviolet radiation which is relatively higher at HA, apart from the increased reflection of such radiation from the snow surface.

62. There is severe photophobia and blepharospasm so that the individual is unable to open his eyes and hence the term

snow blindness'. There is intense pain and severe congestion of the conjunctivee. The affected area of the cornea stains with fluorescein.

63. Treatment consists of application of an antibiotic ointment and pad and bandage for 24 hours. Analgesics should be given for the relief of pain. Prevention is by the use of protective goggles with side protectors.

Thrombotic Episodes

64. The incidence of thrombotic episodes is directly related to the altitude and so is more common at extreme altitude. These episodes include thrombophlebitis, pulmonary thromboembolism and cerebrovascular accidents due to cerebral thrombosis. The basis for these include the polycythaemia which increases blood viscosity, and the hypercoagulable state at HA. This hypercoagulable state is due to raised levels of factors X and XII as well as an increased platelet count. This is further aggravated by the dehydration due to increased water loss through the increased ventilation and by the enforced inactivity during extreme cold.

65. Thrombotic episodes have been implicated in the genesis of NAPO, HACO and pulmonary arterial hypertension of HA. Pulmonary embolisms are more common at extreme altitude and the presentation is similar to that of HAPO; however, HAPO is relatively less common at extreme altitudes as mentioned earlier.

66. The management of these conditions include administration of oxygen and evacuation to the nearest hospital/field ambulance where definitive therapy can be initiated. Imbibing of fluids liberally is advocated, particularly at extreme altitudes, to help prevent these episodes.

EFFECTS OF COLD

Hypothermia

67. HA is invariably accompanied by severe cold during winters and exposure to such low temperatures in an inadequately clothed individual can lead to hypothermia. In addition, hypothermia may be seen in survivors of avalanche accidents and falls into rivers/lakes, particularly in winters.

68. Hypothermia is diagnosed when the core body temperature falls below 35°C. Between 35 and 37°C, the condition is termed as 'cold stress'. Below 25°C is lethal. The usual clinical thermometers available in MI Rooms cannot record temperatures below 35°C.

69. In mild hypothermia (33°C), the onset is subtle and there is pallor due to peripheral vasoconstriction, in coordination and decrease in the shivering activity. As the core temperature falls further, the individual becomes careless about his clothing which can lead to a vicious circle of increasing hypothermia. The individual becomes uncooperative memory is affected, there is somnolence leading on to stupor and coma and, finally, death. The skin is ice cold, the respiratory and heart rates fall and the blood pressure may be difficult to measure. The immediate danger to life is ventricular fibrillation and this can be precipitated by any muscular activity which causes anaerobic blood from the muscles to reach the heart. Terminally there is pulmonary oedema.

70. The management in mild cases consists of gentle rewarming by giving hot beverages to drink, applying hot water bottles, protection from further cooling by extra blankets or a sleeping bag and, if possible, by submersion in a warm bath.

71. In contrast, in severe hypothermia, the primary aim is to prevent death by preventing ventricular fibrillation. This should be considered a medical emergency. Rough handling should be avoided at all costs so as to prevent the products of anaerobic metabolism from the muscles from reaching the heart. The patient should be transported gently to the nearest ADS/Field Ambulance/Hospital. Extra heating en route may be provided by blankets/hot water bottle. Rapid rewarming should be avoided as it can lead to peripheral vasodilatation causing blood to be shunted to the periphery from the centre, aggravating the shock. In hospital, gentle rewarming is continued, IV fluids warmed to 40°C is given to correct the hypothermia and the dehydration, oxygen is supplemented and cardiac resuscitation for ventricular fibrillation is kept ready.

72. The prevention of hypothermia consists of the wearing of adequate clothing in multiple layers keeping the clothes dry, avoiding direct contact wi h snow, avoiding exposure to wind which removes the layer of insulating warm air around the body, keeping the exposed parts of the body to a minimum, increasing heat production by exercise and consumption of high caloric foods.

Local Cold Injury

73. This includes chilblains, trench foot and frost bite and usually affects peripheral structures such as fingers, toes, tips of the nose and the ear lobes. The reasons for this include the peripheral vasoconstriction that occurs on exposure to cold as well as the fact that there are no muscles in these parts which can generate heat. Predisposing factors to local cold injuries are the wearing of tight constricting clothing including tight cramp on straps, contact with cold objects, smoking, imnobility, older age, poor personal hygiene, prolonged exposure to cold and moisture, intercurrent illnesses, fatigue, mental apathy and a past history of cold injury. Alcohol, by causing vasodilatation and by a direct central effect on the hypotnalamus, can aggravate heat loss and thereby predispose to frost bite. Cold injuries are freequently associated with generqalized hypothermia.

74. *Chilblains.* This is described as non-freezing injury of the skin, is seen in suceptible individuals and consists of an abnormal reaction to cold; it occurs at temperature just above the freeging point and pressure sites are vulnerable. The affected part is red and there is intense irritation. Desquamation may follow, but tissue loss is rare. Though by itself it is not serious, the importance of chilblains lies in the fact that they are common and so affect the battle preparedness of soldiers. Also, patients with chilblains are particularly prone to frost bite. Management consists of reassurance, keeping the part dry and warm, immersion in warm water followed by thorough drying and application of vaseline, and symptomatic treatment.

75. *Trench Foot.* This occurs after prolonged contact with moist cold such as water or mud at temperatures above freezing. This condition is uncommon at HA.

76. *Frost Bite.* This is the most serious of the local cold injuries and is seen usually at temperatures below the freezing point. There is freezing of extracellular fluid with the formation of ice crystals leading to a rise in the osmotic pressure which draws water from the intracellular compartment causing intracellular dehydration. This is aggravated by freezing of water in the cells and inhibition of enzyme systems. To this is added ischaemic injury due to vasoconstriction which decreases blood flow and structural damage to the small vessels leading to leakage of plasma and intravascular sludging and local thrombus formation. Arteriovenous shunts open up to bypass the frozen part with the aim of preserving the organism at the cost of aggravating the local injury. Thawing leads to the resumption of circulation but this is followed by stasis and the formation of microthrombi in the arterioles.

77. *Clinical Features.* The onset is usually insidious with pain and numbness followed by loss of sensation in the part especially when there is immobility e.g. lying or sleeping. This leads to a vicious circle where the damage is ignored and further damage occurs. The severity of frost bite depends upon the duration of exposure and the temperature. In very early stages there is freezing of the most superficial layer of the epidermis producing a blanched wheal which is called 'frostnip'. More severe injury is graded into the following four degrees depending on the depth involved :—

(a) *First Degree Frost Bite.* Here there is redness and oedema with delay in capillary filling. Initially the part may be pale but becomes red on rewarming. This stage is fully reversible though symptoms may persist for days.

(b) *Second Degree Frost Bite.* Here again the part is pale and numb and there is no evidence of capillary filling. On rewarming, the part becomes red or violaceous and there is swelling and blister formation. Pain may be

severe. Partial thickness of the skin is involved and a black eschar may form after 2-3 weeks and this separates in about 4 weeks. This stage is still reversible but there may be long lasting sequelae in the form of pain, paraesthesia and hyperhidrosis.

(c) *Third Degree Frost Bite.* There is complete necrosis of the skin and the area is pale and without sensation. On rewarming, the pallor persists. Later, a gangrenous thick eschar forms which peels away over weeks to months leaving behind tender new epihelium. This stage is irreversible with tissue death in the affected skin and subcutaneous tissue layers.

(d) *Fourth Degree Frost Bite.* In this stage there is total freezing of varying depth of deeper tissues. Connective tissue and tendons may be involved though bone is more resistant. Tissues can break off and there may be complete loss of toes/fingers. Demorcation betwen dead and healthy tissue takes about a month.

78. *Extent of Cold Injury.* In all cases of 2nd, 3rd and 4th degree cold injuries, the extent should also be recorded when evident as under :—

(a) ***Hands and Feet.***

(i) One plus (1+) upto distal, inter phalangeal joint.

(ii) Two plus (2+) upto middle inter phalangeal joint.

(iii) Three plus (3+) upto metacarpo-phalangeal or metatarsophalangeal joint.

(iv) Four plus (4+) upto middle of metacarpals of metatarsals.

(v) Five plus (5+) upto wrist or ankle.

79. Treatment of frost bite consists of preventing further injury to the affected part. As first aid, at the level of the RAP, general warmth should be provided by hot fluids and food, sleeping bag or extra blankets and hypothermia, if present, should be dealt with (see above). The patient should be reassured, given mild analgesics such as disprin, dry dressing applied to the part, prophylactic antibiotics started, booster does of tetanus toxid given and the patient evacuated to the nearest field ambulance or hospital. Blisters should not be opened. The primary aim should be to evacuate the patient to a Field Hospital/GH at the earliest and to achieve this, the more severe degrees should be evacuated as Priority I cases. Local warming of the part is not advised at this level unless evacuation is likely to be delayed. Local warming leads to thawing which is painful and makes the individul unable to walk due to the discomfort. Also, thawing and refreezing causes more tissue damage. On the other hand, it has been shown that walking on frostbitten feet does not increase tissue loss.

80. In the Field Ambulance or hospital, IV Lomodex 3 units daily for 3 days continous rapid rewarming of the part in a water bath at 37—41°C over 30 minutes is carried out. The temperature of the bath should be maintained by adding hot water and not by heating the container and, while adding the water, the limb should be removed. Grudual, spontaneous thawing should only be done for superficial degrees of frost bite. During rewarming, pain is common and may be intense necessitating analgesies such as aspirin though, at times, morphia may be required. Blisters should not be opened deliberately. If they have burst, they can be covered with dry gauze dressings. Blisters are a potent source of infection. Abscesses may form and require drainage. Eschar, if present, protects the underlying tissue and will gradually separate. Debridement and amputation should be delayed till demarcation is complete which may take upto 3 months. The role of medical or surgical sympatholysis is limited.

81. Disposal of cold injuries (frost bite) is to observe the individual in low medical category till finality is reached after which his category will depend on the residual deficit. Since the occurrence of frost bite predisposes to recurrences

of cold injury, such individuals should be considered unfit for HA extreme cold areas and such as endorsement should be made in their AB 64/Health Card.

Other Problems at HA

82. These are problems which have been found to be relatively more common at HA. Their management does not differ from that at sea level, but the reason for the increased incidence at HA is unclear at present.

Systemic Hypertension

83. Prolonged stay at HA tends to bring about a fall in the blood pressure. However, work in the Himalayas has shown that during the first week there is a rise in the diastolic and systolic blood pressures. Others has shown that area the blood pressure tends to be on the higher side in both temporary residents at HA as well as in native highlanders. In our experiences with hospital admissions, it is seen that admissions for hypertension is common and is usually among NCOs. JCOs and Officers and in the age group of 30—50 yrs. In addition, many of these have additional risk factors particularly obesity. The behaviour of the BP on deinduction to the plains cannot be predicted; in some the BP tends to return to normal value without treatment, while in others it remains high or may necessitate increase of drug dosage. They require close observation initially after deinduction and, for those with persistent hypertension, the disposal is as for essential hypertension.

Gastrointestinal Problems

84. Gastric flatulence on induction to HA is a normal and temporary phenomenon and due to expansion of the air in the stomach due to fall in barometric pressure. Other problems include an increased incidence of haemorrhoids, peptic ulcers with its complications and pancreatitis.

85. Peptic ulcer, both gastric and doudenal, are common and they often present with haematemesis or as perforation.

Bleeding may also be due to spontaneous haemorrhage caused by the increased capillary fragility which is seen at HA; another reason could be the consumption of analgesics for symptoms of AMS. Management does not differ from that in the plains.

Infections

86. Viral infections are frequent at HA and include herpes zoster, chicken pox, viral hepatitis and mumps. Pneumonias are also frequent and respond rapidly to antibiotics with complete radiological resolution usually by the end of two weeks. Localized infections such as perianal and ischiorectal are also common. Amoebiasis is also another frequent reason for hospital admission.

Cutaneous Reactions

87. At HA there is increased exposure to ultravioilet radiation particularly those of short wavelengths, which are responsible for producing erythema. The acute effects on the skin are similar to keratitis and consists of sunburn which presents as erythema which then leads on to vesiculation and crusting. Pain may be present. Even a short period of exposure may result in blistering, crusting and desquamation which is seen in unprotected areas of the skin such as the scalp and face. The erythema usually reaches its maximum in 12 to 24 hours and fades after 1 to 3 days leaving behind pigmentation. Other effects include delayed pigmentation caused by formation of new melanin ('suntan') and increased thickness of the epidermis. The treatment of the acute erythema consists of rest, cold compresses and analygesics for pain. Prevention is by protective clothing and the application of barrier creams such as para-aminobenzoic acid compounds.

Other Problems

89. These include the nephrotic syndrome, weight loss particularly at extreme altitude, epistaxis, carbon monoxide poisoning due to exposure to fumes from bukharis and koilonychia in native highlanders. The management of these does not differ from the standard.

PREVENTION

High Altitude Illnesses

89. The cadrinal principle for the prevention of high altitude illnesses is not to go too high too fast. The acclimatization protocol to be followed by troops being inducted to high altitude is laid down in AO 110/80 and which has been reproduced as Appx 'A'. The present protocol is under review but, for the present, the same protocol applies for fresh as well as reinductees and air as well as road inductees.

90. Other preventive measures include a thorough preinduction medical examination, prompt treatment of respiratory infections and avoidance of strenuous exertion during the first week of induction. During the first week of induction, all individuals should be encouraged to report sick if they develop any symptom. The role of prophylactic drugs, particularly acetazolamide, on a large scale has not yet been established. However, acetazolamide (250 mg three to four times a day for a day or two) may be given on an individual basis especially for those who have experienced symptoms of HA illness during past inductions. For troops on patrolling duty at HA, it is recommended that after patrolling at higher reaches, they descend to a lower altitude for rest/sleep.

Effects of Cold

91. Prevention of cold injury is far better than cure. The aims are to increase heat production and decrease heat loss. This includes adequate hydration and nutrition, good personal hygiene, proper clothing and shelter, exercise, avoidance of smoking and alcohol and protection from contact with cold objects.

92. In cold climates, the tendency is to ignore personal body care and hygiene. Special care should be given to this aspect. Fluids should be taken liberally and food should be nutritions, hot and appetizing. Alcohol should be avoided, particularly when the individual is likely to be exposed to cold.

There should be adequate shelter from cold and wind in the living area as well as in the latrines and bathing places. Clothing should be loose and in manylayers so that air is entrapped between the layers and adds to the insulation. The outer layer of the clothing should be wind-proof. Two pairs of socks should be worn and an extra pair provided for change. Socks and shoes should not be tight. All clothing should be dry and should be changed at the slightest sign of dampness. Feet should be inspected each night for any sign of cold injury. Foot powder may be sprinkled before wearing socks to keep the feet dry. Regular, moderate exercise is advised and during periods of prolonged immobility, there should be frequent movement of limbs, fingers and toes. Metal parts such as the trigger quard and trigger of weapons should be taped.

93. The prevention of other problems at HA has been mentioned under the respective diseases and will not be elaborated upon further.

CONCLUSION

94. The problems at HA are many and, by and large, preventable. A thorough knowledge of HA physiology and of the clinical features of these illnesses along with a high index of suspicion will help in the early diagnosis and thereby institution of therapy on time. Many of the problems are as yet poorly understood, particularly those occurring at extreme altitudes and a lot of work remains to be carried out in the field of HA medicine. It is here that the sincere RMO, by constant, careful observation and meticulous collection of data under the guidance of the medical/surgical specialist, can contribute greatly to the better understanding of these fascinating problems.

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APPENDIX 'A'

ACCLIMATIZATION PROCEDURE

1. Acclimatization will be carried out in 3 stages depending on the height that the individual is finally going to stay.

(a) *First Stage Acclimatization* : This will be applicable to individuals posted above 2700m and upto a height of 3600m. The acclimatization period will be for 6 days as under :—

(i) *First and Second day* : Rest except for short walks in the unit lines only, not involving any climbs.

(ii) *Third and Fourth day* : Walk at slow pace for 1.5 — 3 Km. Avoid steep climbs.

(iii) *Fifth and Sixth day* : Walk upto 5 Km and climb upto 300m at a slow pace.

(b) *Second Stage Acclimatization* : (Above 3600m and upto 4500m). This is carried out for 4 days as under :—

(i) *First & Second days* : Slow walk for a distance for 1.5 – 3 Km; avoid steep climbs.

(ii) *Third day* : Slow walk and climb upto 300m.

(iii) *Fourth day* : Climb 300 m without equipment.

(c) *Third Stage Acclimatization* : (Above 4500 m). This also lasts for 4 days and is on the same lines as second stage acclimatization.

2. *Re-entry of High Altitude* : Individuals who have left high altitude area will require acclimatization again if they are away for more than 10 days. Individuals who are away for more than 4 weeks will require complete acclimatization

as stated in para 1 above while those who have been away for more than 10 days but less than 4 weeks will have acclimatization for 4 days at each stage like fresh inductees as under :—

(a) *First & Second day* : Rest except short walk.

(b) *Third day* : Walk at slow pace for 1-2 Km. Avoid steep climb.

(c) *Fourth day* : Walk 1-2 Km with climb upto 300m.

APPENDIX 'B'

ABBREVIATIONS USED IN THE TEXT

1. ADH : Anti-diuretic Hormone.
2. AMS : Acute Mountain Sickness.
3. AO : Army Order.
4. CMS : Chronic Mountain Sickness.
5. ECG : Electrocardiogram.
6. HA : High Altitude.
7. HACO : High Altitude Cerebral Oedema.
8. HAPO High Altitude Pulmonary Oedema.
9. HAR : High Altitude Retinopathy.
10. HVR : Hypoxic Ventilatory Response.
11. PO_2 : Partial Pressure of Oxygen.
12. RMO : Regimental Medical Officer.
13. SL : Sea Level.