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Neurophysiological Problems in Snow Bound High Altitude Areas

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Abstract. A series of studies have been conducted to evaluate the neurophysiological responses in young healthy soldiers during acclimatization at 3,500 m altitude in Western Himalayas. The responses of autonomic nervous system, electroencephalogram hypothalamic thermoregulatory efficiency, orthostatic tolerance, sleep profile and effects of sleep deprivation have been studied in fresh inductees during three to five weeks of acclimatization at high altitude and compared with those of one year acclimatized lowlanders and high altitude natives. Physiological significance of these neurophysiological responses in the process of altitude adaptation is discussed in the light of current knowledge in the field.

1. Introduction

The snow bound high altitude (HA) areas pose several problems to sojourner soldiers, mountaineers and tourists visiting high altitude locations. Hypoxia and cold are the two main stressors which are primarily responsible for many of the altitude problems. The nervous system, being highly susceptible to hypoxia, manifests several interesting neurophysiological responses on induction and during acclimatization at high altitude. Most of the maladaptation syndromes appear to have neurophysiological basis for their aetiology. Unfortunately, there is a big dearth of knowledge in the field of neurophysiology of altitude adaptation. Advancement in this field of research would perhaps, lead us closer to solving many of the altitude problems. An attempt has been made to briefly review some of the salient findings of our field trials at 3,500 m altitude in Western Himalayas and laboratory investigations, in the light of the current developments in this field.

2. Autonomic Responses

Hypoxic stress is known to disturb the stability of the balance between sympathetic and parasympathetic activity[1]. Autonomic arousal level was studied in a group of

20 sojourner soldiers on induction by aeroplane and during three weeks of acclimatization at an altitude of 3,500 m in Western Himalayas. The responses of sojourners (SJ) were compared with those of acclimatized lowlanders (AL) staying at HA for more than one year and high altitude natives (HAN) born and brought up at high altitude[2]. Autonomic arousal level was measured by monitoring heart rate, blood pressure, oral temperature, mean skin temperature, cold pressor response at 4°C water, cardiovascular responses to 70° head-up tilt on a tilt table, alpha index of electroencephalogram and 24 hours urinary excretion of catecholamines. Autonomic responses of SJ were suggestive of an augmented sympathetic activity, which showed peak response during 24-72 hours after arrival at high altitude[3]. This is the most susceptible period for the occurrence of pulmonary oedema. There was a gradual recovery of autonomic equilibrium after one week of acclimatization, however, sympathetic activity remained elevated for about three weeks. In HAN, on the other hand, there was relative preponderance of parasympathetic activity, and the responses of AL were intermediate between those of SJ and HAN. There is certain degree of controversy regarding some of the autonomic responses during the initial phase of acclimatization, which could be to some extent, attributed to the mode of induction to HA and other experimental procedures. In general, most of the initial autonomic responses at HA indicate sympathetic over activity[4,5]. Studies also lend support to this concept and the earlier findings of others who have also observed a disturbance in the autonomic equilibrium at high altitude[4,5] which persists for about a week even after the sojourners return to the plains after a prolonged stay at high altitude[6].

The relative dominance of parasympathetic activity observed in HAN as an adaptive characteristic appears to be primarily dependent on environmental factors rather than on the genetic traits, as they tend to lose this feature during two months of their sojourn at the plains[7]. Experimental studies conducted on albino rats supported the view that relatively enhanced parasympathetic activity may be beneficial in improving hypoxic tolerance[8].

3. Electroencephalogram Studies

After our initial studies on autonomic responses at altitude, experiments were conducted to study the central nervous system (CNS) response to altitude adaptation by monitoring electroencephalogram (EEG). The resting (awake) EEG was recorded in A-P temporal run, on a group of young healthy soldiers initially at the plains and after air-induction to 3,500 m for four weeks[9]. EEG responses of these sojourners were compared with those of AL and HAN. There was cerebral cortical depression in the initial phase of induction due to hypocapnia resulted by altitude-induced hyperventilation, which changed to cortical desynchronization in the later part of the first week of stay at HA as a result of sympathetic hyperactivity while the cortical neurones gradually adapted to lower $PaCO_2$. During acclimatization, there was gradual build-up of EEG as observed in AL and HAN. Foster *et al*[10] also observed cortical

depression, as seen in EEG patterns, in the subject who developed acute mountain sickness (AMS). The other available reports on the changes in EEG under hypoxia are confined to studies using gas mixtures[11,12] or during short-term exposure to altitude hypoxia[13].

4. Physiological and Behavioural Alterations

Changes in the activity of autonomic nervous system (ANS) and central nervous system (CNS) at high altitude lead to the manifestation of certain physiological and behavioural changes. Orthostatic tolerance shows a transient deterioration during the first week of arrival at high altitude probably due to hypocapnic influence on the vasomotor centres[14]. It, however, improves after one week of acclimatization as a result of an augmented sympathetic activity and adaptation of the vasomotor centre to reduced $PaCO_2$ level, within this period. Another function which is disturbed at HA is hypothalamic thermoregulatory efficiency which shows deterioration on arrival at HA and remains 'poor' even after a long stay[15,16]. On the other hand, repeated exposure to moderate simulated altitudes (1,500-2,500m) is reported to improve the thermoregulatory efficiency in asthmatic patients who show signs of prognosis after moderate altitude therapy[17,18]. The role of sympathetic hyperactivity as a cause for the deterioration in thermoregulatory efficiency[19,20] does not rule out the direct effect of hypoxia on the neurones of hypothalamic thermoregulatory centre and the physico-chemical effects of the respiratory centre in the brain on the composition of blood passing through the hypothalamic area[17]. Even the cold-induced vasodilation i. e. peripheral vascular response to local cold exposure of hand at 4°C water for 30 minutes, show deterioration which may be explained by the sustained vasoconstriction on account of increased sympathetic activity[21]. This accounts for the increase in the susceptability of cold injuries at high altitude.

5. Sleep Problem

Sleep disturbance is one of the common complaints of sojourners at high altitude. The neurophysiological mechanism underlying the aetiology of this problem is not yet clearly understood, nevertheless, a few human and experimental studies have been attempted in this direction[22,23,24]. Extensive studies were conducted in this laboratory to assess the nature and magnitude of sleep problem at altitude with emphasis on exploring the neurophysiological basis of the problem.

Initially, the subjective evaluation of the problem was done by a questionnaire method on 2,000 soldiers of different age, ethnic and trade groups, staying at different altitudes ranging from 2,790 m (9,000 ft) to 5,150 m (17,000 ft) in Eastern and Western Himalayas. In the second phase, the alterations in sleep patterns were electrophysiologically studied on a group of SJ during two weeks of acclimatization at 3,500 m in Western Himalayas and compared with those of AL and HAN. In the third

phase, experimental studies were undertaken on albino rats to understand the basic neurophysiological mechanism associated with the causation of sleep disturbance at high altitude.

The opinion survey was conducted on 2,000 soldiers using a questionnaire to assess the nature and magnitude of the problem at different altitudes and to find out whether the sleep problem is related to the altitude of stay, duration of acclimatization, age, physical activity, drinking or smoking habits, and psychological profile of the individuals. Subjects included for the study were SJ, AL and HAN, who were of the age between 19 and 50 years. Subjects were asked to grade the quality of their sleep in a three point scale as 'normal, light or disturbed'. The SJ and AL were instructed to keep in mind, the nature of their sleep at the plains as reference while grading the sleep at high altitude.

The percentage of subjects who reported their sleep to be 'normal' 'light' or 'disturbed' at different altitudes are shown in Fig. 1. There was proportionate increment in the magnitude of the sleep problem with increasing altitudes. The problem was severe above 4,000 m altitude and during winter months. The complaints of sleep disturbance were more acute among the fresh inductees, relatively less in AL and rare among HAN (Table 1). The magnitude of sleep complaints was not well correlated

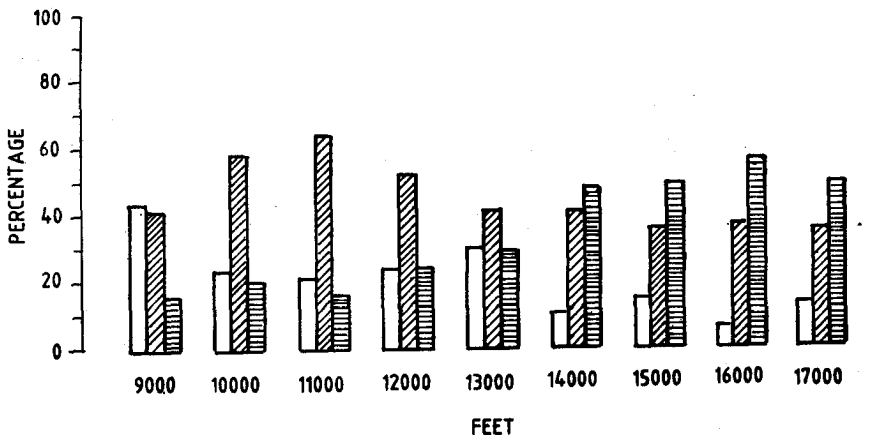

Figure 1. Histogram showing the percentage of subjects reporting their sleep to be 'Normal (blank), light (diagonal lines) or disturbed (vertical lines)' at various altitudes.

Table 1. Percentage of subjects who reported their sleep to be 'normal, light or disturbed' at high altitudes

Subjects	Nature of Sleep		
	Normal	Light	Disturbed
Fresh Inductees	12	50	38
Acclimatized Lowlanders	26	47	27
High Altitude Natives	70	18	12

with age, physical activity, habituation to smoking, alcoholism, frequent intake of coffee or tea, and mode of induction to HA by air or road (Table 2). Most of the subjects who complained of sleep disturbance reported that they were woken up with some respiratory distress during sleep, especially during the initial phase of acclimatization. This suggests the association of periodic breathing, commonly observed in fresh inductees, with disturbance in sleep[25]. Majority of the subjects with sleep disturbance also complained of deterioration in memory and appetite, and increased irritability. Inspite of the consensus of opinion on the existence of sleep problem at high altitude[26,27], there has not been any attempt to quantify the magnitude of the problem at different altitudes. This simple study fills up this lacuna to some extent.

Table 2. Correlation of habitual factors and age with sleep problem

Group	Nature of Sleep		
	Normal	Light	Disturbed
Smokers	21%	45%	34%
Non-smokers	19%	52%	29%
Regular drinking habit	24%	44%	32%
Non-alcoholics	28%	42%	30%
Physically active jobs	22%	45%	33%
Sedentary jobs	27%	43%	30%
Disturbed hunger	16%	30%	54%
Normal hunger	30%	49%	21%
Age groups			
19-30 years	19%	48%	33%
31-40 years	21%	44%	35%
41-50 years	26%	41%	33%

Percentage of subjects who reported their sleep to be 'normal, light or disturbed' is presented in different sub-groups.

6. Sleep Patterns at High Altitude

In the next phase, electrophysiological monitoring of sleep was done on a group of 15 soldiers initially at the plains and thereafter on air-induction to 3,500 m altitude in Western Himalayas for two weeks, and on return to the plains. The sleep scoring of the whole night sleep records was done by the standard method[28] and the duration of various sleep stages and indices of sleep efficiency were also calculated[23]. The sleep profiles of SJ were compared with those of AL and HAN at HA. All the subjects were in the age group ranging from 22 to 30 years. The autonomic arousal and psychological profile (anxiety level and cognitive functions) were also measured by various standard tests, in wakeful condition, during altitude acclimatization.

The typical sleep pattern of a SJ at the plains and HA is shown in Fig. 2a. Frequent short bursts of arousal and reduction of the amounts of slow wave sleep (stage-3 & 4 of Non-REM sleep) were observed during the first night sleep at high altitude.

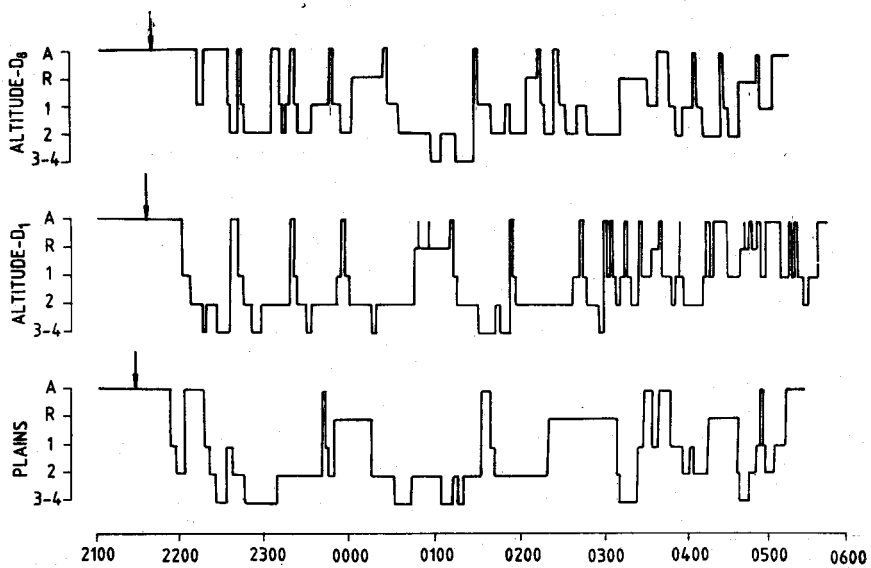

Figure 2a. Sleep patterns of a normal subject (sojourner) at the plains and on days 1 and 8 at 3,500 m. Time in hours is plotted below each graph; time at which lights were switched off is indicated by a small arrow (vertical). Stage awake (A), REM (R) and sleep stages 1, 2, 3 & 4 are shown.

These responses persisted even during the second week of stay at high altitude. Five of the SJ showed complete elimination of stage-4 during the first week at high altitude. Four of the SJ who manifested signs and symptoms of acute mountain sickness (AMS) did not exhibit either frequent arousals or curtailment of slow wave sleep (SWS) as

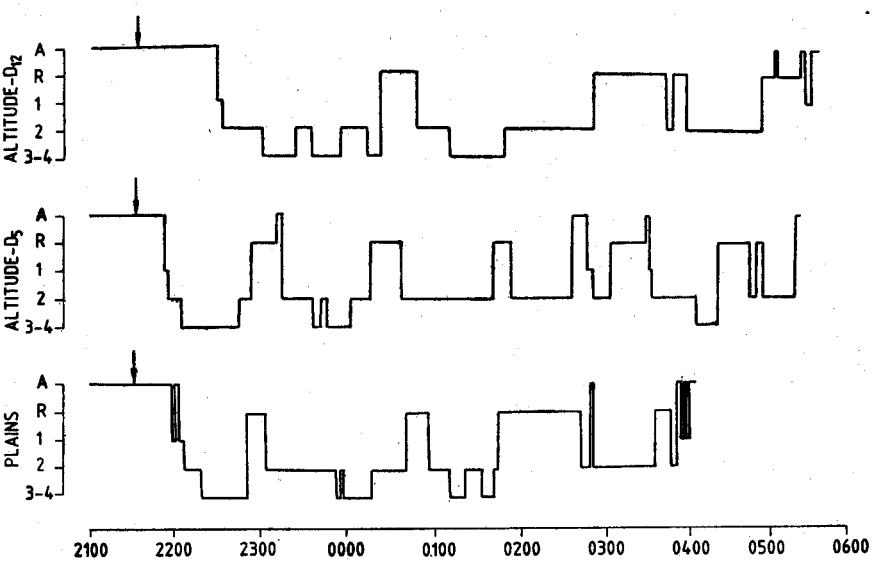

Figure 2b. Sleep profile of a sojourner who suffered from acute mountain sickness.

shown in Fig. 2b. The AL and HAN had relatively less frequent arousals while exhibiting a reduction in SWS (Fig. 3). Total sleep time (ISI) was not significantly altered at this altitude, however, the duration of SWS was significantly ($p<0.05$)

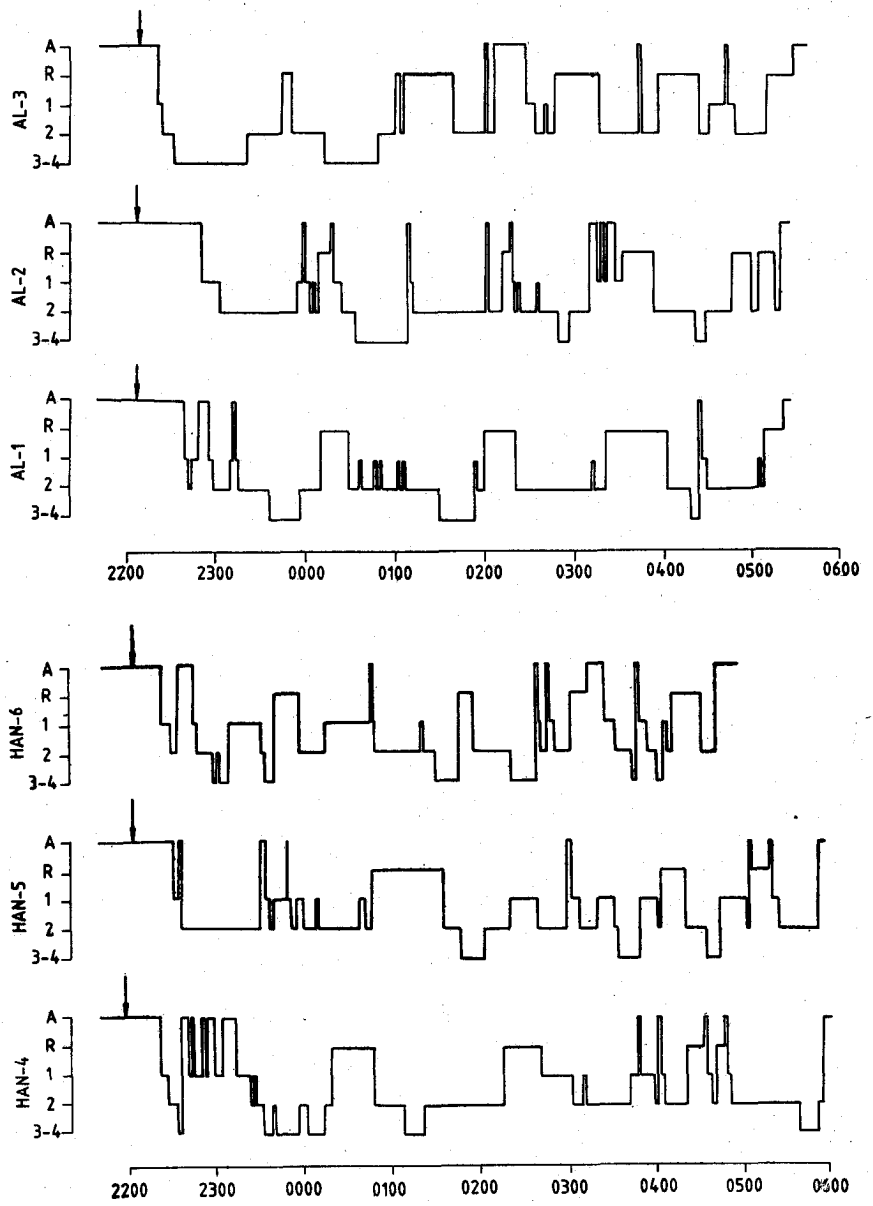

Figure 3. All night sleep plots of (Top) three acclimatized lowlanders (AL), and (Bottom) three high altitude natives (HAN) at 3,500 m.

reduced. Even on return to the plains, the reduction in SWS persisted for two weeks (Fig. 4). Sleep data expressed as percentage of SPT also showed significant reduction in all the three groups of subjects (Fig. 4).

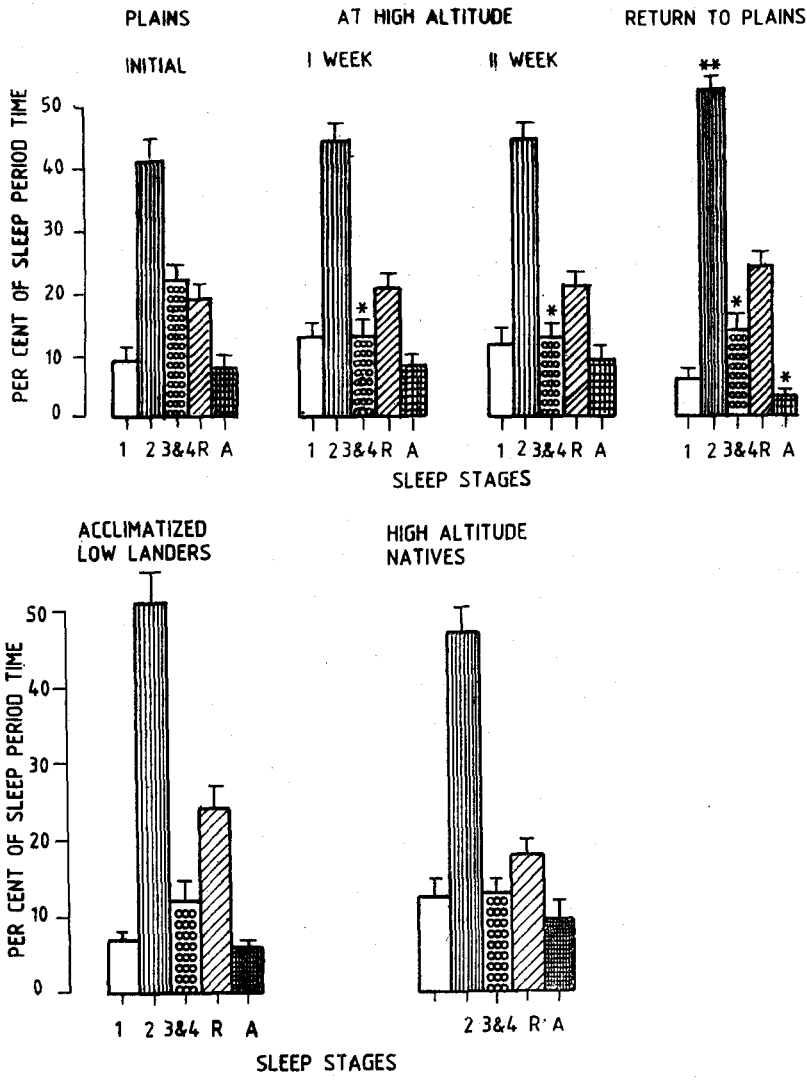

Figure 4. Amounts of each sleep stage expressed as percentage of sleep period time in (Top) sojourners, and (Bottom) in acclimatized lowlanders and high altitude natives.

Sleep latencies of SJ were not significantly altered at this altitude, however, on return to the plains, they had significantly ($p<0.01$) shorter latencies of all the sleep stages (Table 3). Latencies of stages 1,2, and rapid eye movement (REM) phase of sleep were significantly lower ($p<0.01$) in AL than those of SJ. On the other hand, HAN had latencies comparable to those of SJ at SL. Indices of sleep efficiency (Table 4) did not show any significant change at this altitude (3,500 m); on return to the SL sleep efficiency improved ($p<0.05$). If the altitude values were compared with those of return to the plains, the deterioration in sleep efficiency at HA became evident. Body movements during sleep were more ($p<0.01$) in SJ and AL at HA.

Table 3. Latencies of various sleep stages (minutes)

	Plains	Altitude (weeks) I	II	Return to Plains	AL	HAN
Stage 1	18.6	15.1	18.1	1.0***	3.8**	17.2
	±4.53	±3.48	±3.83	±0.31	±1.38	±2.65
Stage 2	31.2	33.6	27.2	7.7**	11.8**	29.2
	±6.71	±5.20	±4.27	±4.27	±3.20	±5.83
Stage 3	51.8	55.2	51.6	28.7**	55.8	51.4
	±10.13	±9.92	±5.28	±6.51	±16.85	±12.70
Stage 4	82.2	86.3	114.8*	65.3	67.3	90.6
	±15.04	±15.43	±22.50	±11.65	±19.86	±11.03
Stage 5	137.4	134.7	167.9	80.4**	91.8**	137·5
	±13.79	±16.43	±13.18	±8.63	±4.09	+11.81

Values are Mean ± SEM *p<0.05 **p<0.01 ***p<0.001
AL : acclimatized lowlanders; HAN : high altitude natives

Table 4. Indices of sleep efficiency

Index	Plains	Altitude (weeks) I	II	Return to plains	AL	HAN
TST/TIB	0.855	0.849	0.854	0.952***	0.914	0.840
	±0.024	±0.021	±0.021	±0.009	±0.015	±0.020
% SREM ÷	0.669	0.618	0.665	0.793	0.762	0.612
(SREM+S1)	0.055	±0.059	±0.065	±0.056	±0.037	±0.077
CL (min)	123	107	111	93**	91	98
	±10.1	±10.7	±8.9	±5.2	±6.8	±5.6
SS (number)	41	48	48	36	44	49
	±2.7	±3.3	±6.4	±3.0	±2.2	±3.5
BM (number)	22	38***	34**	26	33*	31
	±2.5	±3.9	±2.7	±3.3	±3.3	±1.8

AL : acclimatized lowlanders; HAN : high altitude natives; TST : total sleep time; TIB : time in bed; SREM: stage REM; S1 : stage 1; CL : mean cycle length; SS : stage shifts; BM : body movements; Values are means ± SE.
*p<0.05 **p<0.01 ***p<0.001

Sleep data of AMS subjects differed from those of the normals only in the abscence of a reduction in SWS at high altitude (Table 5). All the SJ complained of 'poor' and 'disturbed' sleep at HA after the sleep monitoring schedule. Autonomic indices measured in this study indicated the enhanced sympathetic activity (Table 6) and a little increase in anxiety level (Table 7) during two weeks of stay.

The curtailment of SWS and frequent short bursts of arousal during sleep observed in SJ at HA appear to be adaptive features to prevent the accentuation of hypoxemia which is known to occur due to sleep hypoventilation[29]. Those who do not manifest

Table 5. Sleep profile of subjects with symptoms of acute mountain sickness as compared to normals

Parameter	Acute Mountain Sickness (n=4)				Normals (n=11)			
	Plains	Altitude (Weeks) I	Altitude (Weeks) II	Return to Plains	Plains	Altitude (Weeks) 1	Altitude (Weeks) II	Return to Plains
TST (min)	363 ±22.2	391 ±19.8	407 ±3.4	433 ±8.5	379 ±17.9	383 ±18.6	377 ±20.0	425 ±8.4
S_1 (TST)	6	8	10	8	13	19	16	6*
%	±2.42	±3.5	±5.3	±4.9	±3.2	±2.4	±3.4	±1.6
S_2 (TST)	49	41	49	52	42	53	49	56
%	±1.6	±5.3	±1.7	±3.8	±6.1	±2.5	±4.1	±2.9
$S_{3\ \&\ 4}$ (TST)	24	24	18	21	24	9**	12*	10**
%	±5.0	±4.7	±2.3	±4.8	±4.1	±1.8	±3.0	±2.4
SREM (TST)	21	28	23	19	21	19	28	28
%	±4.8	±2.9	±3.8	±1.9	±2.7	±2.2	±1.9	±3.4

Values are means ± SE; *$p < 0.05$ **$p < 0.01$
TST : Total sleep time; S: Sleep Stage expressed as per cent TST. Statistical comparisons of altitude values of normals and AMS subjects were made with their own values of the plains.

Table 6. Autonomic response during altitude acclimatization—recorded during day in awake state

Parameter	Plains	Altitude (Days)			Return to Plains	AL	HAN
		2	7	14			
HR (bpm)	67	83***	80***	80***	65	69	57***
	±1.4	±1.9	±1.7	±1.2	±1.2	±1.3	±1.6
BP (mmHg)	117	126***	122*	124**	111**	111**	110**
Systolic	±1.2	±0.9	±0.7	±0.7	±0.5	±1.1	±1.2
Diastolic	78	85***	82*	81	70***	75	61***
	±1.7	±1.8	±1.3	±0.9	±1.6	±2.6	±2.2
Tor (°C)	36.9	36.6	36.7	36.7	36.8	36.9	36.8
	±0.10	±0.03	±0.03	±0.02	±0.02	±0.05	±0.04
Tsk (°C)	34.2	31.3***	31.2***	31.2***	32.0***	31.8***	32.4*
	±0.21	±0.19	±0.16	±0.19	±0.14	±0.20	±0.30
RR $(min)^{-1}$	18	19	20	19	20	20	17
	±1.0	±1.4	±1.3	±0.8	±1.1	±0.8	±0.7
CPR-Syst (mmHg)	21	11***	9***	10***	14***	11**	11**
	±1.6	±1.8	±1.2	±1.4	±1.7	±1.8	±1.5
Diast. (mmHg)	22	14***	10***	13***	16**	12**	10**
	±1.7	±0.7	±0.9	±1.7	±1.7	±2.0	±2.0
HR-Tilt (bpm)	24	28	30*	27	15**	18*	16**
	±1.5	±3.0	±2.4	±1.9	±2.0	±1.6	±1.4
AI (%)	30	39**	24*	32	36*	44**	56***
	±3.0	±4.1	±3.2	±2.6	±2.3	±3.1	±2.4

Values are mean ± SEM, *$p<0.05$ **$p<0.01$ ***$p<0.001$; AL : Acclimatized lowlanders; HAN : high altitude natives; HR : Heart rate; BP : blood pressure; Tor : oral temp; Tsk : skin temp; RR : respiratory rate; CPR : cold pressure responses; HR-Tilt : Increase in heart rate during tilt; AI : Alpha index of EEG.

Table 7. Changes in psychological parameters during altitude adaptation

Parameter	Plains (Initial)	At altitude (weeks) I	II	Return to plains
Anxiety level	32	39*	41*	34
(Score)	±2.1	±1.5	±2.1	±1.5
Concentration	24	25	25	27
(Score)	±1.1	±1.9	±1.3	±1.2
Psychomotor	143	109*	128	136
Performance (Score)	±9.8	±10.7	±14.3	±13.4

Values are mean ± SEM *$p<0.001$

such sleep response are likely to suffer from AMS. It is interesting to note that even AL and HAN have reduced amounts of SWS as compared to the sleep profile of SJ recorded at SL. This further supports the thesis that the curtailment of SWS is an adaptive feature at high altitude[30].

Earlier studies showed more frequent arousals at 4,300 m altitude[22] than those observed in the present study conducted at 3,500 m. The increased number of arousals which occur mostly during SWS is likely to be due to the augmented chemoreceptor input into the mid-brain reticular formation[31] resulted from sleep hypoxemia[29]. Periodic breathing and respiratory alkalosis are the other factors likely to be associated with sleep disturbance. It is rather difficult to emphasize the role of any of these factors as these are likely to interact. Pappenheimer[24] suggests that the mechanism by which hypoxia affects sleep may be biochemical in the sense of direct interference of hypoxia with some unknown metabolic process underlying sleep mechanism. Augmented sympathetic activity, elevated levels of catecholamines in blood[5,7], and anxiety observed during the initial phase of altitude induction may contribute, in a limited extent, to the sleep problem through the excitation of reticular formation.

Subjects who suffered from AMS had normal amounts of SWS and less frequent arousals than the asymptomatic normals. This, in turn, would lead to the accentuation of hypoxemia during sleep and manifestation of the symptoms of AMS. The reason for the lack of normal sleep response in AMS cases is not clear, nevertheless, the following explanation seems plausible. These individuals may possess attenuated chemoreceptor sensitivity[32] which fails to sense the accentuation of hypoxemia during sleep. A lag in oxygen delivery system resulted from a decrease in cardiac output observed especially during the early morning hours of sleep[33] could be a contributory factor for the manifestation of symptoms of AMS.

7. Neurotransmitters Under Hypoxia

Experimental studies conducted on albino rats also showed more frequent arousals and reduction in SWS as observed in man, but also increase in REM sleep under

hypoxia (Table 8). The brain levels of 5-hydroxy tryptamine (5-HT), norepinephrine (NE) and dopamine (*DA*) estimated in this study showed significant reduction ($p<0.05$) on hypoxic exposure (Table 9). The frequent awakenings observed in rats during sleep in hypoxic environment were mostly associated with intermittent gasping type of breathing. These observations are in conformity with those of Pappenheimer[24]

Table 8. Percentage of slow wave sleep (SWS), rapid eye movement (REM) sleep and stage awake, and number of awakenings per hour during 1100-1500 hrs in normoxic and hypoxic condition

Sleep stage	Normoxia	Hypoxia
SWS (%)	53.6 ± 4.0	32.7 ± 6.3**
REM (%)	18.0 ± 3.8	34.0 ± 7.4*
Awake (%)	28.4 ± 1.1	38.3 ± 6.4*
Number of awakenings	20.2 ± 4.2	34.8 ± 5.3**

Values are means ± SEM *$p<0.05$ **$p<0.01$

Table 9. Brain levels of dopamine, norepinephrine, 5-*OH* tryptamine, homovanilic acid and 5-*OH* indole acetic acid in normoxic and hypoxia (Mean ± SEM)

Variables	Normoxia	Hypoxia
Norepinephrine ng/g wet tissue	370.63 ± 74.85	291.88 ± 36.37*
Dopamine ng/g wet tissue	791.38 ± 84.12	584.25 ± 35.28*
5-*OH* Tryptamine ng/g wet tissue	737.63 ± 48.65	591.13 ± 24.21*
Homovanillic Acid ng/g wet tissue	20.88 ± 0.69	21.38 ± 1.19
5-*OH* Indoleacetic Acid ng/g wet tissue	436.00 ± 30.56	370.88 ± 28.04

*$p<0.05$

who had described respiratory changes during sleep in rats under hypoxia. The decrease in the total brain content of 5-HT and catecholamines is, perhaps, due to the inhibitory effects of hypoxia on the enzymes, such as tyrosine hydroxylase and tryptophane hydroxylase, associated with the synthesis of these neurotransmitters[34,35,36] which influence the sleep physiology a to larger extent[37]. Further studies are required on the effects of hypoxia on the electrical activity of subcortical areas closely associated with sleep physiology and the specific regional distribution of monoamines in these areas. These studies would lead us to a better understanding of the intricate neurophysiological mechanism which is affected by hypoxia resulting in alterations in the sleep physiology at high altitude.

8. Effects of Sleep Deprivation at Altitude

The troops during war or exercise are likely to be subjected to total or partial sleep deprivation for a prolonged period. This may affect the normal physiological respon-

ses during altitude acclimatization leading to an altered acclimatization status. A study was conducted on young soldiers to evaluate the effects of 48 hours total sleep deprivation (SD), initially at Delhi (260 m altitude) and later after three weeks of acclimatization at 3,500 m altitude. Autonomic responses, thermoregulatory efficiency and physiological functions were measured one day prior to SD, after 24 hours and 48 hours of SD and after 48 hours recovery sleep following SD, both at the plains and at high altitude.

Autonomic arousal level as assessed by a battery of tests showed depression in the sleep deprived subjects both at the plains and at high altitude (Table 10). However, the sleep deprived subjects exhibited an augmented sympathetic reactivity to an additional stress, as evidenced from the increased cold pressor response in them following sleep deprivation. Critical flicker frequency (CFF) response showed deterioration after SD at the plains and at HA, the decrement was more at high altitude. Thermoregulatory efficiency showed marked deterioration after SD at HA, while this response was not affected to any appreciable extent at the plains (Fig. 5). Another interesting observation was the slower recovery in all these responses following 48 hours of

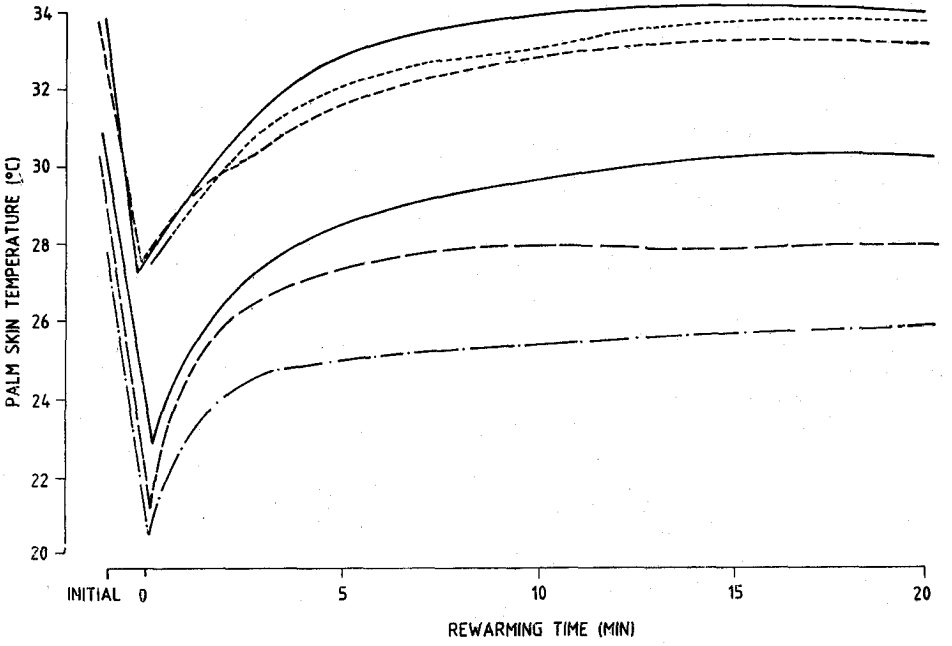

Figure 5. Rewarming pattern of right hand after cold immersion at 10°C water for two minutes.

	At Plains	At Altitude
Pre-deprivation	———	———
After 48 h sleep deprivation	....	— — —
After 48 h recovery sleep	----------	—.—.—.

Table 10. Physiological effects of sleep deprivation at high altitudes

Parameter	At Plains				At high altitude			
	Initial	Sleep 24 h	Deprivation 48 h	After recovery sleep	Initial	Sleep Deprivation 24 h	48 h	After Recovery sleep
HR (ppm)	67±1.9	66±2.1	59±1.6***	67±1.8	81±3.3	73±3.0**	74±3.0**	77±3.1
BP systolic (mm Hg)	117±1.7	116±1.9	110±2.4*	112±1.2*	124±2.5	119±3.1	118±2.7**	115±1.9***
Diastolic (°C)	80±2.3	76±1.5	74±2.7	74±1.3	89±1.1	88±2.3	86±2.7	84±3.1
Tor	36.8±0.04	36.7±0.07	36.4±0.09***	36.8±0.05	36.7±0.03	36.4±0 07**	36.3±0.07***	36.5±0.10**
RR/Min	19±0.5	21±0.9	20±0.8	20±0.8	21±0.5	19±0.4*	19±0.7*	18±0.8**
CPR (mmHg)	16±1.4	26±2.2***	—	21±2.3***	13±2.1	17±2.9*	—	7±1.6*
Diastolic	18±1.2	27±1.8***	—	18±1.9	13±1.7	18±2.2*	—	10±1.3
CFF	45.4±0.9	—	43.0±0.7***	45.3±0.9	42.6±0.7	—	37.9±0.4***	40.4±0.5***

Values are mean ± SEM *$p < 0.05$ **$p < 0.01$ ***$p < 0.001$
The heat rate (HR), blood pressure (BP), oral temperature (T_{or}), respiratory rate (RR), cold pressor response (CPR) and critical flicker frequency show significant changes as a result of sleep deprivation.

recovery sleep at high altitude. On the other hand, at the plains, the recovery was almost complete within 48 hours.

The augmented sympathetic reactivity to a superimposed stress following SD at HA may draw our concern, as our previous study has suggested sympathetic hyperactivity to be a probable predisposing causative factor for the genesis of HAPO[38]. The decrement in thermoregulatory efficiency and CFF show that SD at HA affects CNS functions also. Mountaineers and troops operating at HA should take a special note of this SD response, as severe cold exposure following SD may prove to be detrimental due to deterioration in thermoregulatory efficiency resulted from SD. This effect may partly explain the increased incidence of accidental hypothermia and cold injuries during mountaineering, military exercise and war operations, as the individuals engaged in such manoeuvres are likely to suffer from partial or total SD,

9. Neurogenic Involvement in HAPO

The concept of centrogenic (neurogenic) pulmonary oedema in head injuries, cerebral compression and brain lesion is known for over 30 years. However, the possible involvement of central neural mechanism in the genesis of HAPO has, recently, been suggested by us[38] demonstrating the role of hypothalamus and sympathetic activity in the aetiopathology of HAPO (Fig. 6). The mechanism is not as simple as it has been shown in this schematic diagram; but, perhaps, it is a part of the complex neural

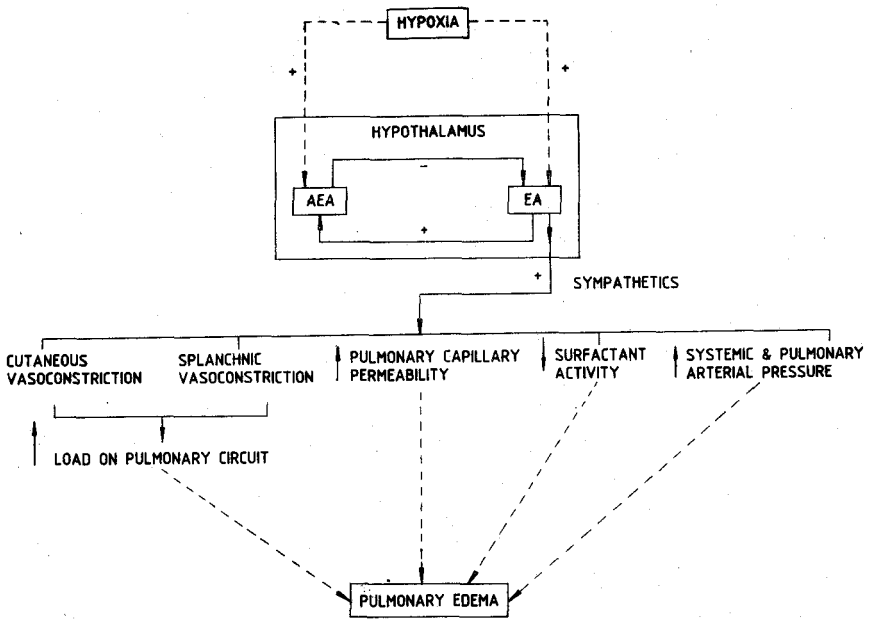

Figure 6. A new concept to illustrate the role of Edemagenic area (EA) and Anti-Edemagenic area (AEA) of hypothalamus in the genesis of high altitude pulmonary oedema (HAPE).

neurophysiological mechanism underlying its aetiology. The edemagenic area of hypothalamus shows increased excitation followed by sympathetic hyperactivity in animals which develop pulmonary edema on exposure to simulated altitude[39]. Studies are currently in progress in our laboratory to assess the effects of sympathetic blockers in the prevention of the sequence of events leading to the genesis of HAPO.

Type-J receptors of Paintal[40] and lung irritant receptors which are known to be stimulated by pulmonary congestion and oedema, may play a significant role in the manifestation of some of the pathophysiological changes associated with HAPO such as cough, bradycardia, muscle weakness and respiratory distress. These are clearly demonstrated to be the respiratory and visceral responses to stimulation of J-receptors and irritant receptors[41]. It is likely that in patients of HAPO the J-receptor mechanism is less efficient to elicit muscular inhibition and other protective cardiovascular and respiratory responses. The processing of the inputs of J-receptors and other pulmonary receptors at the CNS especially at the respiratory complex of the brain stem is perhaps abnormal in the susceptible individuals. These are some interesting possibilities which need further elucidation.

The neurobiology of altitude adaptation is a fertile field of research as very little is known about the neurophysiological responses to altitude adaptation. Effects of altitude on learning, memory, hunger, taste, reflexes, cognitive function and developmental neurobiology could be some areas of interest for neurobiologists.

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