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Sleep Patterns at an Altitude of 3500 Metres

by

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ABSTRACT. – Alterations in sleep pattern during acclimatisation at an altitude of 3500 m were studied on 27 healthy men (20-30 years of age). Of these, 15 were sojourners (SJ), 6 were acclimatised lowlanders (AL) and 6 were high altitude natives (HAN). Baseline sleep profile of SJ was electrophysiologically monitored, initially at Delhi (260 m) and later at 3500 m altitude in Western Himalayas for 2 weeks. At high altitude (HA) the sleep patterns of AL and HAN were also monitored for comparison. There were 4 cases of acute mountain sickness (AMS) among SJ, whose sleep profiles were also recorded. The state of autonomic arousal was assessed by a battery of indices, while the psychological arousal was measured by the anxiety scales. On completion of studies at HA, the SJ were flown back to the plains and re-tested within one week of return. SJ showed curtailment of slow wave sleep (SWS) and frequent short episodes of arousal during sleep at HA. AL and HAN also had lesser amounts of SWS; however, the arousals and awakenings during sleep were less frequent. Subjects who experienced AMS had normal amounts of SWS at HA. There was sympathetic hyperactivity and slight increase in anxiety level in SJ, while HAN and AL had relatively reduced level of sympathetic activity. The curtailment of SWS and frequent arousals observed in SJ during the initial phase of acclimatisation at HA, appear to be adaptive features to prevent the accentuation of arterial hypoxemia due to sleep hypoventilation.

INTRODUCTION

Sleep disturbance is one of the common complaints of sojourners (SJ) at high altitude (Barcroft, 1925; Pugh and Ward, 1956). The objective evidence to show that the duration and/or quality of sleep are altered at high altitude (HA) is rather limited. The mechanism of causation of sleep disturbance at HA is not yet clearly understood. Similar problem has also been observed during short and prolonged stay at South polar plateau in Antarctica where the barometric pressure ranges from 64.6 Kla to 69.3 Kla (Joern et al., 1970; Natani et al., 1970). Reduction in the amounts of slow wave sleep (SWS) and frequent arousals have been reported at 4300 m and these changes are attributed to sleep hypoxemia and respiratory alkalosis (Reite et al., 1975). Studies conducted under simulated altitude conditions, however, reveal that most of the subjects have normal synchronised sleep at 65.7 Kla (Miller and Horvath, 1977a). Sleep hypoxemia and decrease in

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oxygen delivery system are regarded as possible factors responsible for the sleep problem (Miller and Horvath, 1977b; Edelman, 1980). Later, the experimental studies have shown a reduction in SWS with concurrent increase in REM sleep and wakeful state in rats during sleep in hypoxic environment (Pappenheimer, 1977). Changes in sleep profile at HA appear to have a direct bearing on adaptation (Malkin et al., 1980).

Our earlier studies (Malhotra et al., 1976; Selvamurthy et al., 1981) have indicated the elevation of sympathetic activity and catecholamine levels in sojourners at 3500 m, which result in alterations in EEG patterns in wakeful state (Selvamurthy et al., 1978). The magnitude of sympathetic arousal, and alterations in psychological profile (Sharma, Baskaran and Malhotra, 1976) may have a role in causation of disturbance in sleep at HA, which has not been studied so far. Moreover, sleep patterns of high altitude natives and acclimatised lowlanders who have been staying at HA for a long period, have not been clearly illustrated.

Hence, in the present study, emphasis has been directed towards evaluating the changes in sleep pattern of the sojourners during two weeks of acclimatisation at 3500 m and compare their sleep profile with those of high altitude natives and acclimatised lowlanders. Attempts have also been made to correlate the changes in the status of autonomic arousal and psychological profile with alterations in sleep pattern at HA.

MATERIALS AND METHODS

Twenty seven healthy men (soldiers) served as the experimental subjects who belonged to three categories: 15 were sojourners (SJ) – residents of the plains taken to HA for the first time during the study; 6 were acclimatised lowlanders (AL) – residents of the plains stationed at HA for more than one year; 6 were high altitude natives (HAN) – natives born and brought-up at HA (3300 m-3800 m). The SJ and AL were of the same ethnic group (Rajpuths) while the HAN were Ladakhis. The mean ($\pm$ s.e.m.) age, body weight and height of SJ were 27.1 $\pm$ 1.2 years, 57.6 $\pm$ 0.85 kg and 172.3 $\pm$ 1.5 cm respectively, while those of AL were 25.3 $\pm$ 1.4 yr, 57.7 $\pm$ 1.68 kg and 168.8 $\pm$ 1.8 cm, and those of HAN were 26.8 $\pm$ 3.0 yr, 56.2 $\pm$ 2.04 and 166.3 $\pm$ 2.3 cm respectively. Subjects were maintained on a diet supplying 3900 kcal and controlled physical activity schedule throughout the period of the study. Sleep history of all subjects was recorded by standard questionnaire method. They were also briefed about the experimental procedures prior to the commencement of the study, and their consent to extend maximum cooperation in the study was also obtained. They were later subjected to routine clinical examination and were found to be free from any ailment.

Baseline sleep pattern of SJ was monitored at Delhi (260 m) in a sleep laboratory maintained at 25 $\pm$ 2C dry bulb (DB) with 40-50% relative humidity (RH) and 30-45 dB background noise level. Thereafter, SJ were flown to an altitude of 3500 m in Western Himalayas in three batches of five each at an interval of five days to facilitate the recording of the initial altitude responses in many of them. Sleep monitoring was repeated at HA during two weeks of acclimatisation, in a sleep laboratory developed at HA, with 23 $\pm$ 2C DB, 30-40% RH and 25-40 dB. Only non-consecutive sleep data could be obtained as the sample size was large and only one subject could be handled every night for sleep monitoring. At HA, sleep profile of AL and HAN were also recorded for comparison. On completion of altitude studies, SJ were flown back to the plains and re-tested.

Subjects reported to the laboratory at 2100 h after normal dinner. They were prohibited from alcohol intake one day prior to and on the day of sleep monitoring. Surface

electrodes were fixed on the scalp at C_3, P_3, C_4, P_4, P_z, O_1, O_2 and A_2 locations as per the 10-20 International system of electrode placement (Jasper, 1958), for EEG recording. Electro-oculogram, electromyogram and electrocardiogram were monitored on a GRASS – Electroencephalograph (model-6). Sleep recording started at the time when the lights were switched off and lasted upto their awakening on the next day morning. The paper (chart) speed was maintained at 15 mm sec^{-1} throughout the night. Two dummy recordings were done on each subject during two consecutive nights to get them accustomed to the instrumentation and sleep in the laboratory conditions. Actual recording of the baseline sleep profile was made only on the third night. Sleep scoring was done by analysing every page of the whole night sleep record and 30 sec epoch was recorded using the conventional criteria (Rechtschaffen and Kales, 1968). Following sleep indices were calculated from the sleep data: time in bed (TIB), sleep period time (SPT), total sleep time (TST), latency of different sleep stages, amounts of each sleep stage, mean cycle length (CL), stage shifts, indices of sleep efficiency i.e. TST/TIB, and SREM/(SREM + S_1) according to the established methods (Miller and Horvath, 1977a). Each awakening was counted as arousal which was characterised by EMG activation, occasionally by eye movements and alpha activity.

Autonomic and psychological indices were recorded when the subjects reported to the laboratory in the morning after a light breakfast. The following autonomic indices were monitored in thermoneutral conditions after one hour of complete rest: heart rate, blood pressure, oral temperature, mean skin temperature, respiratory rate, cold pressor response at 4°C water, cardiovascular responses to 70° head-up tilt and alpha index of EEG were recorded by standard methods (Selvamurthy et al., 1981). Anxiety levels were quantified using the questionnaire of R. B. Cattell. In addition, concentration and psychomotor performance (eye-hand coordination) were also measured using standard psychological methods (Sharma, Baskaran and Malhotra, 1976). Subjective assessment of the quality of sleep was also recorded at HA by interview.

Data was analysed by the method of Analysis of Variance on a IBM computer-360/44. Comparisons were made between the data obtained at the plains on SJ with their own response at HA, and also with those of AL and HAN. Intergroup comparisons were also made in the data of SJ, AL and HAN.

RESULTS

All the three groups of subjects had comparable physical characteristics. Sleep data obtained at the plains in SJ were comparable to the previously established norms for young adults (Williams, Agnew and Webb, 1964). The typical sleep pattern of a SJ at the plains and at HA is shown in figure 1-A. Frequent short bursts of arousal and reduction in the amounts of slow wave sleep (stages 3 and 4) were observed on day-1 and were present even during the second week of stay at HA. Five of the SJ showed complete elimination of stage-4 during the first week at HA. In four of the SJ, signs and symptoms of acute mountain sickness (AMS) manifested during the first week (Sutton, 1971). These subjects did not exhibit either frequent arousals or reduction in slow wave sleep (SWS) as seen in figure-1B, as compared to the asymptomatic normals. AL and HAN had relatively less frequent arousals while exhibiting a similar curtailment of SWS (Fig. 2).

Sleep latencies of SJ were not significantly altered at this altitude, however, on return to the plains, they had significantly shorter latencies of all the stages of sleep (Table 1). Lat S_1, S_2 and REM were significantly lower ($P < 0.01$) in AL as compared to the sea level

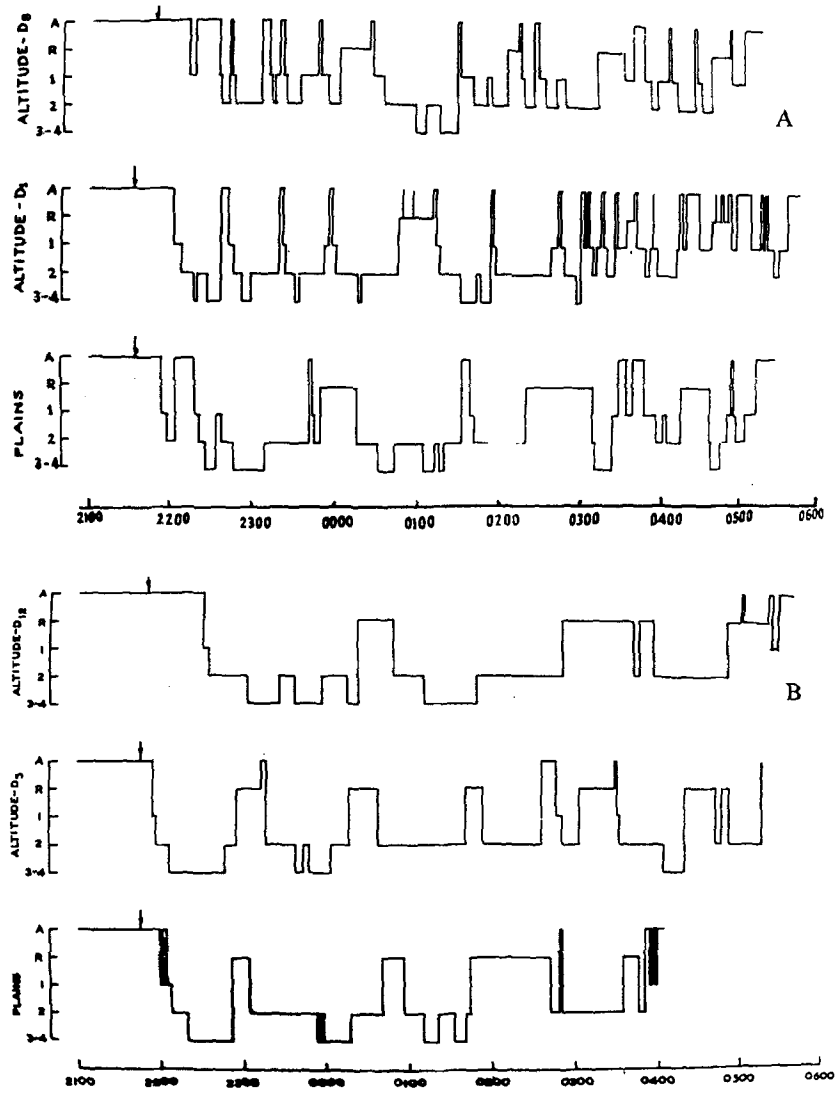

Fig. 1 A. Sleep patterns of a normal sojourner at the plains and on days 1 and 8 at 3500 m. Time in hours is plotted below each graph; time at which lights were switched off is indicated by small vertical arrows. A, awake; R, REM; sleep stages 1, 2, 3 and 4 combined, are shown. "D_1" – Day 1.
B. Sleep profile of a sojourner who suffered from acute mountain sickness (sleep recording at the plains was interrupted at 0425 h due to technical problems).

(SL) values of SJ. Sleep latencies of HAN, on the other hand, were comparable to those of SJ at SL.

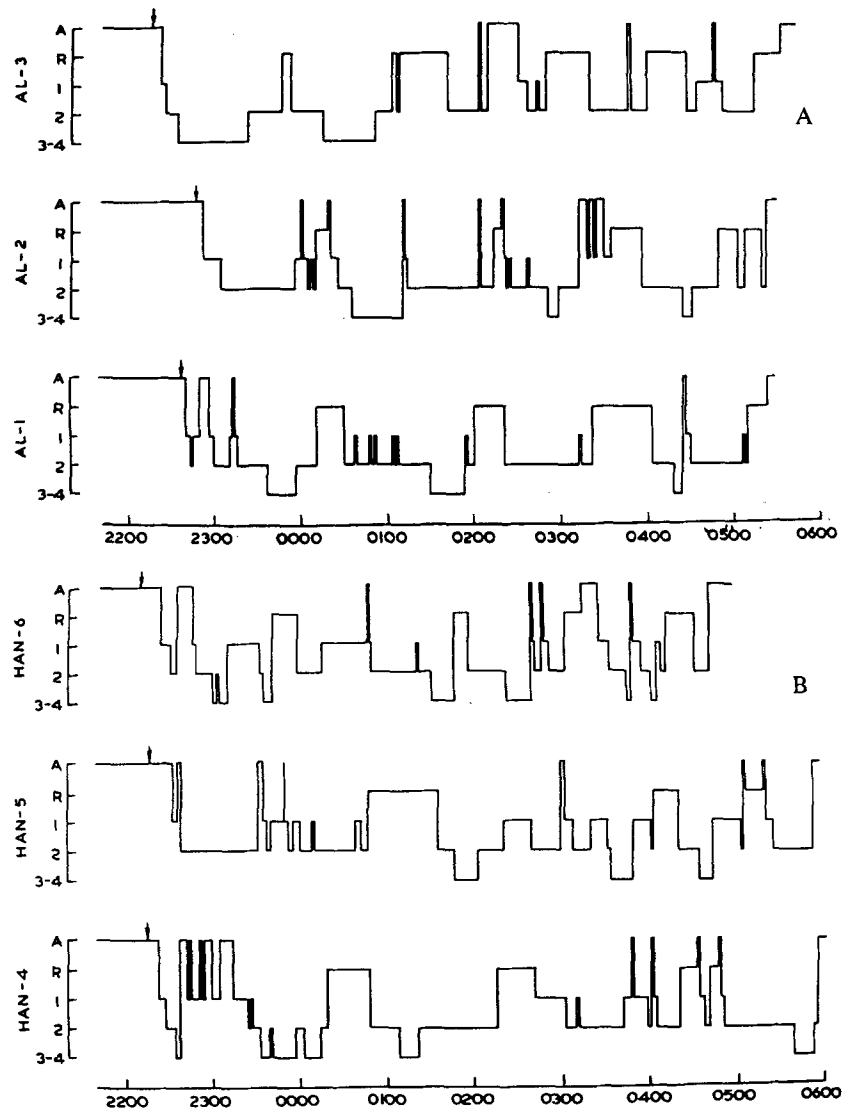

Fig. 2 All night sleep plots of (A) three acclimatised lowlanders (AL) and (B) three high altitude natives (HAN).

SPT and TST were not significantly altered in SJ at HA, however, there was a significant reduction ($P < 0.05$) in SWS at HA, which persisted even on return to SL for 2 weeks (Table 2). AL and HAN also had significantly less amounts of SWS ($P < 0.05$) at HA as compared to SL data of SJ. Sleep data expressed as percentage of SPT also showed significant reduction in SWS in all the three groups of subjects at HA as compared to the SL data of SJ (Fig. 3).

Table 1. Latencies of various sleep stages (in minutes)

Stage	Plains	Altitude (weeks) I	Altitude (weeks) II	Return to Plains	AL	HAN
S_1	18.6 ± 4.53	15.1 ± 3.48	18.1 ± 3.83	1.0*** ± 0.31	3.8** ± 1.38	17.2 ± 2.65
S_2	31.2 ± 6.71	. 33.6 ± 5.20	. 27.2 ± 4.27	7.7** ± 4.27	11.8** ± 3.20	29.2 ± 5.83
S_3	51.8 ±10.13	55.2 ±9.02	51.6 ±5.28	28.7** ±6.51	55.8 ±16.85	51.4 ±12.70
S_4	182.2 ±15.04	86.3 ±15.43	114.8* ±22.50	65.3 ±11.65	67.3 ±19.86	90.6 ±11.03
S_{REM}	137.4 ±13.79	134.7 ±16.43	167.9 ±13.18	80.4** ±8.63	91.8* ±4.09	137.5 ±11.81

Values are means ± SE
AL, acclimatized lowlanders; HAN, high altitude natives.
* $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$.

Table 2. Grouped sleep data (in minutes)

Parameters	Plains	Altitude (weeks) I	Altitude (weeks) II	Return to Plains	AL	HAN
TIB	437 ±11.1	454 ±9.9	454 7.6	448 6.5	425 13.7	438 7.8
SPT	406 ±10.2	421 ±10.6	427 ±7.7	440 ±6.0	414 ±13.5	408 ±13.1
TST	373 ±13.5	386 ±13.3	388 ±13.3	427 ±5.9	389 ±12.1	368 ±14.7
S-A	32.5 ±7.0	34.6 ±8.2	38.5 ±9.6	13.8* ±3.0	25.0 ±4.0	40.2 ±12.5
S-1	39.2 ±8.3	55.7 ±9.0	49.7 ±9.6	27.9 ±8.0	30.0 ±4.4	51.2 ±11.3
S-2	165 ±17.2	188 ±15.8	192 ±14.8	232** ±10.9	209* ±15.8	192 ±17.3
S-3 and 4	90 ±12.7	56* ±10.6	56* ±9.6	61* ±11.1	49* ±14.3	52* ±8.3
S-REM	79 ±10.1	86 ±8.8	91 ±8.0	106* ±11.0	101 ±14.8	73 ±8.03

Values are means ± SE. * $P < 0.05$, ** $P < 0.02$
TIB, Time in bed; SPT, sleep period time, TST, total sleep time; S-A, Stage awake, AL, acclimatised lowlanders; HAN, high altitude natives.

Indices of sleep efficiency did not show any significant change at this altitude (Table 3); but on return to the plains, sleep efficiency improved ($P < 0.05$). Body movements during sleep were more ($P < 0.01$) at HA in SJ and AL at HA. Sleep data of AMS subjects differed from those of the normals only in the absence of a reduction in SWS at HA

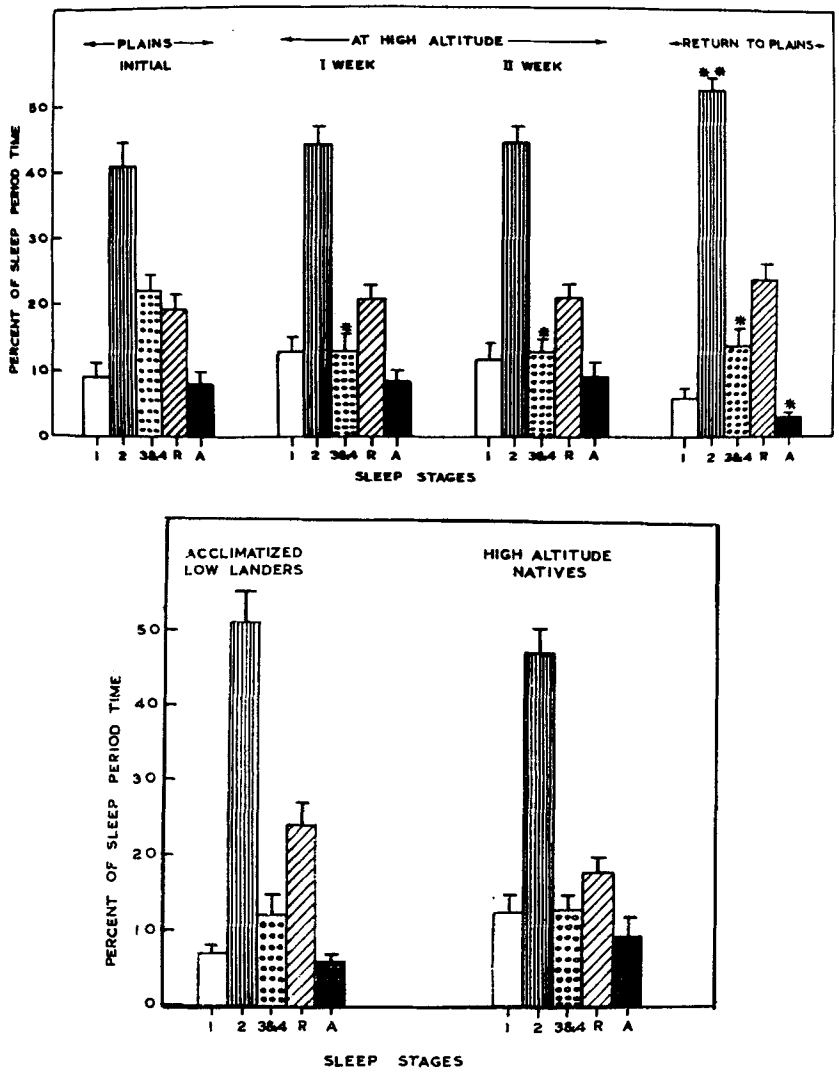

Fig. 3 Amounts of each sleep stage expressed as percentage of sleep period time in (A) sojourners and (B) in acclimatised lowlanders and high altitude natives.

(Table 4). All the SJ complained of "poor" and "disturbed" sleep at HA, during subjective assessment of sleep, which was done in the morning following sleep monitoring. They also reported "tiredness" in the morning, which points to the lack of recovery value of sleep.

Autonomic indices measured in this study indicated sympathetic overactivity during two weeks of stay at HA (Table 5). In addition, the anxiety levels of SJ were also higher ($P < 0.001$) during the first week and improved during the second week of acclimatisation (Table 6). Psychomotor performance showed deterioration ($P < 0.001$) during the first

Table 3. Indices of sleep efficiency

Index	Plains	Altitude (weeks) I	Altitude (weeks) 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	6.618	0.655	0.795	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, Acclimatised 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 mean ± SE.
* $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$

DISCUSSION

The salient findings of the present study are the curtailment of SWS and frequent short episodes of arousal observed in SJ at 3500 m, which appear to be adaptive features to prevent the accentuation of hypoxemia which is known to be resulted from sleep hypoventilation. Those who do not manifest such sleep response and have normal synchronised sleep, 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 HA.

Earlier studies showed more frequent arousals at altitude of 4301 m (Reite et al., 1975) than observed in the present study, probably due to the higher altitude at which those studies have been conducted. The increased number of arousals which occur mostly during SWS is likely to be due to the augmented chemoreceptor input into the midbrain reticular formation (Dell, Huzelin and Bonvalet, 1961; Oswald, 1962) resulted from sleep hypoxemia (Powles et al., 1978; Edelman, 1980). Periodic breathing and respiratory alkalosis are also suggested as other factors likely to be associated with this sleep response. It is rather difficult to emphasise separately the role of any one of these factors as these are likely to interact. Pappenheimer (1977) 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 and elevated catecholamines observed at this altitude (Malhotra et al., 1976; Selvamurthy et al., 1981) may also contribute to the increased stimulation of reticular formation. Even though anxiety level has shown increase, its impact on the sleep profile should not be stressed, as the subjects in the present study are well motivated as assessed by the psychological questionnaire.

Sleep latencies, TST, SPT and indices of sleep efficiency are not altered at this altitude. Nevertheless, most of the subjects complained of sleeplessness and "poor" quality of sleep. It may be due to the frequent arousals during sleep as well as reduction in SWS, as the curtailment of SWS may interfere with the recovery process from physical fatigue (Shapiro et al., 1975).

Table 4. Sleep profile of subjects with symptoms of acute mountain sickness as compare to normal

Parameter	Acute Mountain Sickness (n = 4)				Normals (n = 11)			
	Plains	Altitude (weeks) I	Altitude (weeks) II	Return to plains	Plains	Altitude (weeks) I	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 (% of TST)	6 ±2.42	8 ±3.5	10 ±5.3	8 ±4.9	13 ±3.2	19 ±2.4	16 ±3.4	6* ±1.6
S_2 (% of TST)	49 ±1.6	41 ±5.3	49 ±1.7	52 ±3.8	42 ±6.1	53 ±2.5	49 ±4.1	56 ±2.9
S_3 and 4 (% of TST)	24 ±5.0	24 ±4.7	18 ±2.3	21 ±4.8	24 ±4.1	9** ±1.8	12* ±3.0	10** ±2.4
SREM (% of TST)	21 ±4.8	28 ±2.9	23 ±3.8	19 ±1.9	21 ±2.7	19 ±2.2	23 ±1.9	28 ±3.4

Values are means ± SE, * $P < 0.05$, ** $P < 0.01$

TST, Total sleep time; S, Sleep Stage. Statistical comparisons of altitude values of normals and AMS subjects were made with their own values of the plains.

Table 5. Autonomic response during altitude acclimatization – recorded during day in awake state.

Parameter	Plains	Altitude (Days) 2	7	14	Return to Plains	AL	HAN
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) Systolic	117	126***	122*	124**	111**	111**	110**
	±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
T_{or} (°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
T_{sk} (°C)	33.2	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, acclimatised lowlanders; HAN, high altitude natives; HR, Heart rate; BP blood pressure; Tor, oral temp.; Tsk, skin temp.; RR, resp. rate; CPR, Cold pressure response; HR-Tilt, Increase in heart rate during tilt; AI, Alpha index of EEG.

Table. 6. Changes in psychological responses of sojourners

Parameter	Plains (Initial)	Altitude (weeks) I	II	Return 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 Performance	143	109*	128	136
(Score)	±9.8	±10.7	±14.3	±13.4

Values are means ± SE; * $P < 0.001$.

Sleep in natural altitude conditions is likely to be affected by cold stress also which is known to depress the REM phase (Buguet et al., 1976; Buguet, Roussel and Radomski, 1977). In the present study, effect of cold has been eliminated as the experiments have been conducted in thermoneutral conditions. However, while considering sleep in natural altitude conditions, effect of cold also should be borne in mind. Reduction in REM sleep due to cold will interfere with the recovery of plastic activity of the human physiological systems (Moruzzi, 1966). This also could, to some extent, account for the deterioration in the recovery value of sleep at HA and the general complaints of lack of "freshness" on awakening in the morning. The decline in the psychomotor performance observed in the initial phase of altitude acclimatisation may be due to this effect (Angus et al., 1979).

Stage 2 of non-REM sleep is relatively more at HA in SJ, AL and HAN as compared to the SL values of SJ. This may be an attempt by the physiological system to compensate for the loss of SWS. Sleep profiles of AL and HAN are comparable to each other. Both the groups show less amounts of SWS as seen in SJ, but have less frequent arousals. The amount of stage 2 is more and sleep latency is less in AL and HAN as compared to the SL values of SJ. But for these features, sleep patterns of AL and HAN are not different from those of SJ at HA.

AMS subjects have normal amounts of SWS and less frequent arousals than the asymptomatic normals. This, in turn, would lead to the accentuation of hypoxemia and manifestation of the symptoms of AMS. The association between the severity of AMS and impairment of pulmonary gas exchange has already been demonstrated (Sutton et al., 1976). The reason for the lack of normal sleep response, i.e. curtailment of SWS, in AMS subjects is not clear. The following explanation seems plausible. These individuals possess attenuated chemoreceptor sensitivity (Mathew et al., 1983) which fails to sense the accentuation of hypoxemia during sleep, because the chemoreceptor sensitivity is further reduced during sleep even in normal individuals (Bulow, 1963; Honda and Natsui, 1967; Phillipson, Murphy and Kozar, 1976). Our earlier studies indicate the dampening effect of hypocapnia resulted from altitude induced hyperventilation on reticular activating system, which leads to cerebral cortical synchronisation (Selvamurthy et al., 1978). Forster et al. (1975) have shown that in AMS subjects cortical synchronisation is more marked. Linking of these two observations leads us to think that AMS subjects perhaps have greater degree of reticular dampening due to hypocapnia and respiratory alkalosis. As a result of this, even the augmented chemoreceptor input into the reticular structures fail to elicit EEG arousal and or awakening. A lag in the oxygen delivery system resulted from a decrease in cardiac output observed especially in

the morning hours could be a contributory factor for AMS (Miller and Horvath, 1977b).

Sleep characteristics of SJ observed on arrival at the plains after three weeks of sojourn at HA, present some interesting findings. Sleep latencies and arousals during sleep are much reduced. The amounts of stage 2 and sleep efficiency index are increased, while the duration of stages 3 and 4, and the mean cycle length still remain reduced as compared to their initial plains values. These responses are noticed for two weeks on return to the plains.

It may be concluded that the curtailment of SWS and frequent short bursts of arousal during sleep at HA may be important adaptive features, at least in the initial phase of altitude acclimatisation, to protect the fresh inductees from accentuation of hypoxemia resulted from sleep hypoventilation. An individual who fails to show this sleep response may manifest the symptoms of AMS. Reduction in SWS is observed in AL and HAN.

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