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EFFECT OF HIGH ALTITUDE ON HUMAN AUDITORY BRAINSTEM RESPONSES

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Abstract : The effect of hypobaric hypoxia on Brain Stem Auditory evoked potentials (BAERs) were studied. BAERs were recorded in 30 volunteers at sea level (SL) and then at high altitude (HA) of 3200 m (HA I) and 4300 m (HA II) in Eastern Himalayas and on return to sea level (RSL). The BAERs were recorded using Nicolet Compact – 4 (USA) in response to monaural auditory stimuli consisting of clicks of 100 μs square pulse at a rate of 15/sec. The BAERs were recorded on day 4 of their stay at 3200 m and 4300 m respectively. Findings indicated an increase in absolute peak latencies of wave V at 3200 m, which was statistically significant. On further ascent to 4300 m there was an increase in absolute peak latencies of wave I and III indicating delay in sensory conduction at the medullo-pontine auditory pathways.

Key words : auditory evoked potential, high altitude, absolute peak latency, interpeak latency

INTRODUCTION

Earlier we have reported that high altitude stress causes delay in peak latencies of wave I of Brain Stem Auditory Evoked Responses (BAERs) recording during the first week of induction to HA which normalized during further stay at HA (1). In these studies the Brainstem Auditory Evoked Responses were recorded at 3500 m in the Western Himalayas where the subjects stayed for 3 weeks. In the present study, the subjects were inducted to high altitudes in Eastern Himalayas for a total duration of two weeks. The Brainstem auditory evoked potentials were recorded at two altitudes i.e. 3200 m and 4300 m in an ascending order to see the effect of increasing altitude on BAERs.

METHODS

The study was carried out on 30 healthy male volunteers, between the ages of 21–32 years. All subjects were informed about the nature of experiments. They had never been

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to HA before. Clearance from the ethics committee of Defence Institute of Physiology and Allied Sciences, Delhi was obtained to carry out these experiments. All the subjects had thorough ENT check up including audiometery to exclude ear pathology. The experimental procedure was explained to the subjects in detail and their written consent was obtained. The BAERs was recorded at sea level (SL) and HA at 3200 m (HA I) and 4300 m (HA II) in Eastern Himalayas and on return to sea level (RSL). In a total duration of 2 weeks of stay at HA, the volunteers stayed at 3200 m for 6 days for acclimatization before ascending to 4300 m, where the stay was for 8 days. The BAERs were recorded on the 4th day after their arrival at HA I and HA II. The subjects were inducted in two batches consisting of 15 subjects each so that recording of BAERs in all the 30 subjects falls uniformly on 4th day at HA I and HA II. The BAERs were recorded in the morning between 0800 hrs to 1300 hrs.

Brainstem auditory evoked response (BAERs)

Recording of BAERs was carried out in a quiet and dimly lit room with subject in a comfortable supine position. Electrodes were attached at the vertex (C_Z) and the ear lobes, with the ipsilateral lobe serving as the reference and the contralateral lobe as the ground. Monaural auditory stimuli consisting of clicks of 100 µs square pulse were delivered through an electrically shielded earphone at a rate of 15/sec. The contralateral ear was masked by pure white noise. The intensity was 70 dB above the click hearing threshold level (HL). The evoked electrical activity was amplified 10,000 times. A band pass of 150–3000 Hz was used to filter out the undesired frequencies and the response to 2000 click presentations were averaged for 10 msec sweep time by a computer averaged Nicolet Compact – 4 (USA) and printed on paper by the printer. The sampling rate was 1280 samples/sec. At least two trials were obtained to ensure reproducibility of the responses. The peak latencies of wave I, III and V, the interpeak latencies of I–III, III–V and I–V were analysed. The method used for recording the BAERs was similar to one reported earlier from our laboratory (2).

Statistical analysis was carried out using Student's t-test.

RESULTS

The representative recording of BAERs of a subject is shown in Fig. 1. The latencies and the interpeak latencies of BAERs of the subjects at sea level (SL), on induction to HA I (3200 m), HA II (4300 m) and after return to SL are given in Table I. The Recording of BAERs from right and left year did not show any significant change in absolute peak latencies and interpeak latency of wave I, III and V and hence the data has been pooled. The mean ± SD value of peak latencies of wave I at SL was 1.82 ± 0.165 msec and a HA I and HA II were 1.82 ± 0.139 msec and 1.89 ± 1.74 msec respectively. The peak latency of wave I was significantly increased at HA II ($P<0.05$) as compared to SL. The peak latencies of wave III increased significantly ($P<0.01$) at HA II as compared to SL (Table I). On return to SL, it remained significantly at a higher level as compared to SL ($P<0.01$). The peak

TABLE I: Effect of hypobaric hypoxia on BAEPs at different altitudes.

	Peak latencies (ms)			*Interpeak latencies (ms)*		
	I	*III*	*V*	*I–III*	*III–V*	*I–V*
SL	1.82±0.165	3.91±0.192	5.81±0.203	2.09±0.207	1.89±0.166	3.98±0.262
3200 m	1.82±0.139	3.91±0.218	5.85±0.226*	2.09±0.213	1.94±0.251	4.04±0.22
4300 m	1.89±0.174*	3.99±0.215**	5.82±0.169	2.10±0.217	1.83±0.172	3.94±0.187
RSL	1.76±0.095*	3.96±0.182**	5.87±0.201*	2.20±0.2193**	1.91±0.154	4.10±0.193**

All values are Mean ± SD
*P<0.05, **P<0.01
All values are compared with respect to sea level

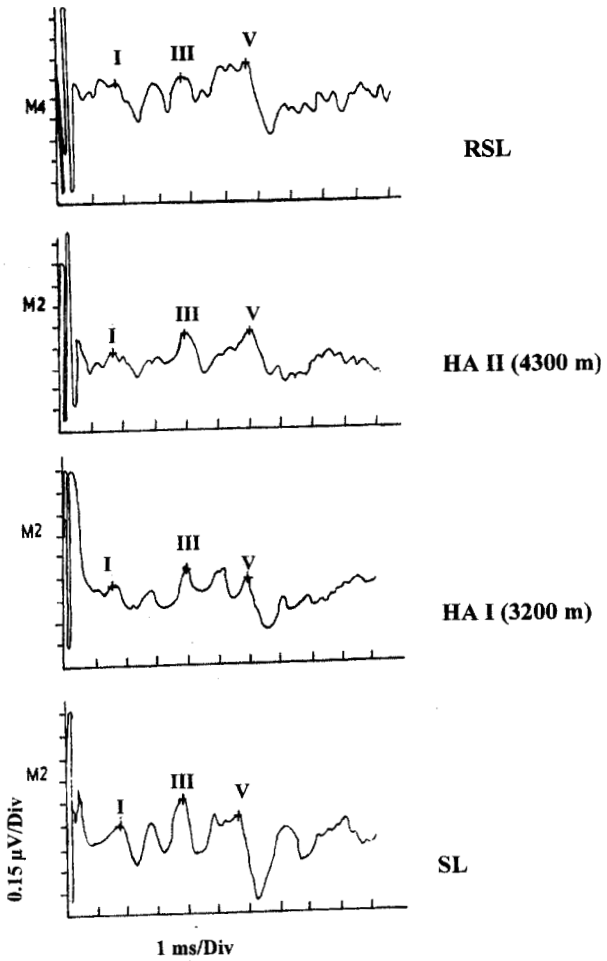

Fig. 1: Shows the representative wave form of the Brainstem auditory evoked response of the subject at Sea Level (SL), HA I (3200 m) and HA II (4300 m) and on return to Sea Level (RSL). Figure shows an increase in peak latency of wave I & III at HA II.

latencies of wave V showed an increase at HA I and the increase was significant (P<0.05).

There was a significant increase in interpeak latencies at RSL of wave I–III and wave I–V (Table I).

DISCUSSION

There was an increase in the peak latencies of wave I and III on induction to HA of 4300 m. The peak latencies of wave V showed an increase at 3200 m (HA I), which was significant. The BAERs peak latencies at the sea level did not show any difference and were within the values reported by others (3). Urbani and Lucertini (4) reported significant decrease of the brainstem transmission time (I–V interval) in volunteers exposed to 90 min of hypobaric hypoxia (5184 m) in an altitude chamber, which the authors said may be due to slow decay of compensatory mechanisms acting during hypoxia and/or a transient neuronal hyperexcitability at the end of the hypoxic stress. In another study (5) authors examined the effect of prolonged periods of hypoxia at high altitude on the latency of the auditory brain stem evoked response at sea level, 3500 m and 4310 m. The results

showed wave V latency was prolonged after 24 hrs ascent from 1300 m to 3900 m. After 72 h at the latter altitude, wave V latency returned to normal value. No further change was observed after the second rapid ascent from 3500 m to 4310 m. Authors have interpreted increase in wave V latency during hypoxia in terms of decline in auditory sensitivity.

The above observations point out that time of recording of BAER during HA induction is important. HA induced symptoms are generally more severe during first 72 hrs of induction and recede rapidly thereafter but reappear if up hill climbing continues at rapid pace (6). These HA induced symptoms causes behavioural and physiological changes i.e. anorexia, insomnia, emotional irritability and increased ventilation in sojourn which are caused by altered functions of the CNS (7, 8, 9, 10). HA exposure causes cortical cerebral depression in the initial phase due to hypocapnia resulted by altitude induced hyperventilation, which changed to cortical desynchronization in the latter part of the first week of stay at HA (3500 m) as a result of sympathetic hyperactivity while the cortical neurons gradually adapted to lower $PaCo_2$. During acclimatization, there was gradual build up of EEG as observed in acclimatized low landers (9). In the present study the BAERs were recorded on the 4th day at HA I and HA II. There may be partial inhibition of hyperventilation process caused by hypocapnia and subsequent alkalosis on day 4th at HA I and HA II which causes dampening effect on reticular activating system and leads to increase in peak latencies of wave I and III at HA II (11, 12).

Similarly there was an increase in peak latency of wave V at HA I (3200 m), which may again be due to hypocapnia caused by respiratory alkolosis leading to neural hyperpolarization and causing decrease in excitability (13, 14, 15).

From the present study, it may be concluded that hypoxia results in decrease in auditory sensitivity leading to increase in wave latencies which may be due to altered excitability of CNS occurring in response to HA acclimatization process.

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