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SHORT COMMUNICATION

BRAINSTEM AUDITORY EVOKED POTENTIALS (BAEPs) IN TOBACCO SMOKERS

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Abstract : Brainstem auditory evoked potentials (BAEPs) were studied in 10 chronic male tobacco smokes (average age 26.55 years) with history of smoking 3 to 14 years to detect early subclinical impairments in auditory pathway. The values of BAEPs were compared with those of 28 age matched normal male non-smokers (control). The results revealed significant prolongation of latencies of wave I and III indicating that conductivity of sensory impulse along acoustic nerve and pons is affected in chronic smokers which may be attributed to adverse effect on myelination by nicotine and toluene present in tobacco smoke.

Key words: BAEPs tobacco smokers

INTRODUC TION

Tobacco smoking is an intentionally invited health hazard affecting both active as well as passive smokers. The overall death rate for male smokers is 70 percent greater than for male nonsmokers (1). During burning of tobacco in a cigarette various processes such as pyrolysis pyrosynthesis, distillation, sublimation, hydrogenation, oxidation, decarboxylation, dehydration result in the generation of more than 4000 identifiable compounds present in tobacco itself or new compounds generated thereof.

Many of these constituents' concentrations exceed the industrial threshold values. Out of these constituents tobacco-tar, nicotine, carbon monoxide and other gas phase chemicals have been given more attention with respect to their adverse effects such as cardiovascular diseases, cancer promotion, chronic obstructive pulmonary diseases and teratogenic effects. Many of these constituents are known individually to be neurotoxic. Since sensory function is prerequisite for self and awareness of the environment for normal functioning of brain, it was interesting to know .how tobacco smoke constituents affect the sensory function. It is in this connection evoked potential study was done in smokers. The measurement of evoked potentials - somatosensory, visual and auditory, is a promising technique for assessment of neurotoxicity including subclinical state as a result of exposure to chemicals. These potentials reflect functional integrity of sensory tract in the brain and help in identifying the sites impaired due to neurotoxic factors. Hence, the main object of the present study was to detect subclinical abnormalities induced in auditory pathways in a group of tobacco smokers using BAEP technique.

METHODS

Study population comprised of ten chronic male smokers having average age 26.55 ± 3.36 years. Seven of them were bidi smokers with

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average consumption of 7.10 bidi per day (range 3 to 24) and average history of smoking 8 years (range 3 to 14). Remaining three were cigarette smokers consuming average 3.33 cigarettes per day (range 1 to 5) with history of smoking 3.2 years (range 3 to 7). Twenty eight age matched male non-smokers served as control. These were subjected to neurological examination to exclude any symptomatically abnormal subject. For recording BAEPs click stimuli (2048) of intensity 70 dB above normal hearing threshold, at the rate of 10/sec and 0.1 msec duration were presented monoaurally. The other ear was masked by white noise - 40 dB H.L. These clicks were generated by passing 0.1 msec square pulses through shielded headphones with the alternating polarity. The active (+) electrode was placed at CZ position (vertex) and the reference electrode at the ipsilateral ear lobe (A1 or A2). The electrodes were plugged to a junction box and skin to electrode impedance monitored and kept below 5 K Ohm. The signals picked up by these electrodes were filtered (6 dB point 100 Hz and 3 KHz) amplified, averaged and displayed on the screen of MEB - 5200 (Nihon Kohden, Japan) Evoked Potential Recorder. The absolute peak latencies, amplitude and interpeak latencies (IPL) were monitored with the help of digital cursors. Two trials of 2048 clicks were sufficient to give reproducible recordings of BAEPs. The latencies of peaks I to V, the IPLs (I-V), (I-III), (III-V) and amplitude of wave I and V calculated. Studies using the similar technique in our laboratory have also been published else where (2, 3, 4).

RESULTS

Data pertaining to BAEPs in smokers and non-smokers are given in Table I. It has been observed that peak latencies of wave I and III are significantly prolonged in the smokers in comparison with non smokers. However, no stastistically significant differences were seen in other parameters.

DISCUSSION

It is seen that main effect of tobacco smoking is on wave I and III component of BAEP among smokers (Fig.1). It needs to be mentioned that the wave I, III & V components of BAEPs primarily represent volume conducted electrical activity from the acoustic nerve, pons and midbrain respectively.

The interpeak latencies between these three components reflect neural conduction in the corresponding segments of the brain-stem auditory pathway (5, 6). Excepting latencies of

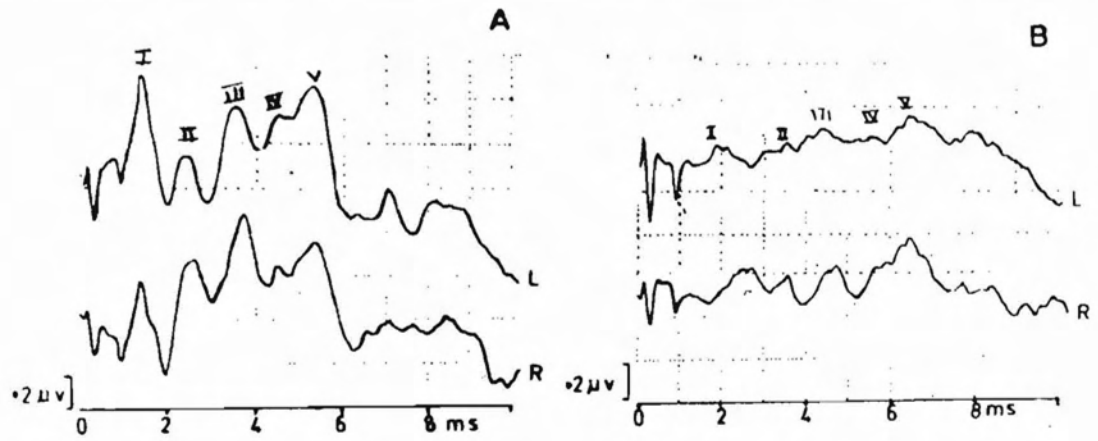

Fig. 1 : Normal (A) and representative abnormal BAEP in a tobacco smoker (B).

TABLE I : Composite data of Brainstem auditory evoked potentials (BAEPs) in smokers (n = 10) and non-smokers (n = 28).

Parameters	*Smokers Mean ± SD*	*Non-smokers Mean ±SD*	*P value unpaired 't' test*	*Significance*
Peak latencies(ms)				
Wave I	1.68 ± 0.21	1.55 ± 0.16	0.045	S
Wave II	2.71 ± 0.32	2.59 ± 0.13	0.101	NS
Wave III	3.79 ± 0.30	3.63 ± 0.16	0.033	S
Wave IV	4.87 ± 0.33	4.78 ± 0.22	0.375	NS
Wave V	5.62 ± 0.34	5.49 ± 0.23	0.189	NS
Amplitude (μv)				
Wave V	0.29 ± 0.13	0.25 ± 0.14	0.467	NS
Wave I	0.32 ± 0.11	0.32 ± 0.17	0.989	NS
V : I	0.94 ± 0.36	0.97 ± 0.76	0.889	NS
Interpeak latencies (ms)				
IPL I-III	2.08 ± 0.16	2.11 ± 0.16	0.650	NS
IPL III-V	1.88 ± 0.17	1.86 ± 0.24	0.772	NS
IPL I-V	3.94 ± 0.20	3.98 ± 0.29	0.712	NS

S = Significant; NS = Non Significant

wave I & III, the values of latencies and IPLs of all other waves obtained by us are almost similar to ones reported for normals by other authors (7, 8). This indicates that conductivity of sensory impulse along acoustic nerve and pons is affected in smokers as evidenced by significant prolongation of latencies of wave I and III. This may also indicate changes in excitability of neural pool of these generators in the lower brainstem i.e. medullopontine region. Nicotine and toluene present in tobacco smoke may perhaps be implicated as culprit chemicals inducing changes in BAEPs. Radioactive studies have demonstrated that the nicotine preferentially gets concentrated in the nuclei of the diencephalon and lower brainstem (9). It has further been reported that large doses of nicotine affect the sensory motor system (9). Similarly, toluene has also been detected in all brain regions, with highest concentration in the brainstem followed by the midbrain region and cerebellum both in animal and human studies. The initial uptake of toluene was significantly correlated with total lipid content of each brain region (10, 11). A further study on rotogravure printers exposed to toluene has shown a stastistically significant alteration in the evoked responses, visible for all waves and all the intervals studied indicating that auditory nervous system modification occurs as a result of toluene before the occurrence of clinical signs. From the foregoing discussion one may infer that because of affinity of nicotine and toluene towards lipid rich tissue of brain, prolongation of latencies of BAEPs occur as a result of their adverse effect on myelination of sensory pathway. However, it is difficult to pinpoint particular chemical or exclude other chemical responsible for the changes.

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