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Short communication

Short- and middle-latency auditory evoked potentials in abstinent chronic alcoholics: preliminary findings

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Summary Short- and middle-latency auditory evoked potentials (BAEPs and MAEPs) were studied in 15 chrooic alcoholic pa tien ts after 1 month's abstinence and compared with those of 15 heaJthy controls. matching the patients pairwise for sex and age. Most of the parameters studied varied more within the alcoholic group than within the control group. The BAEP results agree with previous reports; in the alcoholic group, BAEP peak V was significantly delayed and the inter-peak intervals. lll-V and I-V, were lengthened. The latencies of the MAEP components Na and Pa, on the other hand, were significantly shortened. These findings suggest that chronic abusive consumption of alcohol may bring about structural and/or neurochemic:iJ alterations at various levels in the auditory pathway.

Key words: Short-latency auditory evoked potentials; BAEPs; Middle-latency auditory evoked potentials; MAEPs; Alcoholism

Abusive consumption of alcohol bas been shown by a variety of techniques to cause alteration in both the function and the structure of the nervous system. In particular. since the late 1970s, functional nervous degeneration in chronic alcoholics has been investigated by means of evoked potentials.

Da ta have been obtained concerning short-latency auditory evoked potentials, or BAEPs (Chu and Squires 1980; Begleiter et al. 1981; Chu et at. 1982; Touchon et al. 1984; Chan et al. 1985; Chu 1985; Mabin et al. 1985); visual evoked potentials, or VEPs (Dustman et al. 1979; Posth uma and Visser 1982; Posthuma et al. 1983; Chan et al. 1986); and slow cogniti ve potentials, such as P300 (Begleiter and Porjesz 1979; Pfefferbaum et al. 1979; Begleiter et al. 1980, 1981; Pfefferbaum et aI. 1984; Porjesz and Begleiter 1985; Porjesz ec al. 1987) and contingen t negative variation (Skerchock and Cohen 1984). Though not entirely mutually consistent, the results of all these studies confirm the alteration of traces. Most studies have employed auditory stimuli and collected data concerning changes in the early and lace s1ages of auditory information process ing; hi therto, no research has been done on middle-latency auditory evoked potentials (MAEPs) in alcoholic subjects.

MAEPs. or 'middle-latency responses' (Picton et al. 1974; Davis 1976), appear between the early components produced in the acoustic nerve and brain-stem and the late cortical components produced by cognitive processes (Deiber e1 al. 1988). The MAEP components usually recognized in the literature are No, Po, Na. Pa, Nb and Pb (Picton et al. 1974; Ozdamar and Kraus 1983; Polich and Scarr 1983; Sainz et al. 1983).

It was originally thought that these potentials correspond to the reflex activation of auricular and lemporal muscles rather than to a cerebral response to auditory stimuli (Bickford et al. 1964; Mast 1965). and though it was shown later that they persist in the absence of myogenic activity (Mendel and Goldstein 1969; Mendel and Hosick 1975; Harker et al. 1977; Kileny et al. 1983), the neurone groups generating them have still not been precisely identified. Most research on this front has concerned Na and Pa. Some authors have suggested that both these components are generated by a single structure in the auditory cortex (Scherg and Von Cramon 1986). but most studies point to their having different sources. A subcortical origin has been attributed to Na. probably in the medial geniculate nucleus, polysensorial thalamic nuclei and/or the thalamo-cortical connections, while the source of Pa has been

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placed both subcortically (Otero et al. 1986) and in the primary auditory area and neighbouring areas receiving from and/or projecting into it (Geisler et al. 1958: Vaughan and Ritter 1970; Picton et al. 1974; Davis 1976; Polich and Starr 1983; K.ileny et al. 1987). either contralaterally (Kaga et al. 1980) or bilaterally (Vivion et al. 1980; Buchwald et al. 1981; Ozdamar et al. 1982; Erwing and Buchwald 1986, 1987).

The hypothesis that Na and Pa originate at different locations is supported by recent 'sequential mapping' studies, which suggest that Na has a deep source lying in the mesencephalon or diencephalon, while Pa is produced bilateraly in the supratemporal auditory cortex (Deiber et al. 1988).

On the basis of the findings obtained in recent years, Na and Pa may be considered as proper indexes of activity in the intermediate level of the auditory information pathway. Accordingly, we fell that they might be useful for the evaluation of changes brought about at this level by chronic abusive consumption of alcohol, and hence complement the information provided by BAEPs and long-latency potentials.

Material and methods

Subjects and stimulus parameters

The 15 outpatients studied (13 men and 2 women) had been diagnosed as chronic alcoholics according 10 the DSM III criteria. They were aged 23-51 years (mean 36.2, S.D. 7.48), had histories of alcohol abuse for at least 8 years (mean J 8.5 years), and prior to the recording session had abstained for 25-35 days and received no medication for at least 3 days. Fifteen healthy volunteer controls matched the patients pair- wise for sex and age (mean 35.5 years, S.0. 7.12 years).

None of the subjects had an auditory threshold above 65 dB SPL, and none of the alcoholic patients had other addictions (except tobacco). or serious cardiopulmonary, renal or liver disease. or serious psychic and/or neurological disorders not attributable to their alcoholism. None of the patients had been diagnosed as suffering from Wernicke-Korsakoffs psychosis. Five suffered from blackouts and 4 bad had convulsions.

Evoked potentials were recorded between 10 a.m. and *2* p.m .. i.e., before the midday meal. The subjects sat in a comfortable chair in a partially soundproofed , electrically insulated room with dim lighting, and were instructed to relax but remain awake with their eyes closed.

Potentials were evoked by monoaural stimuli 70 dB above the subjective hearing threshold, 70 dB REL of white noise being applied contralateraliy. For BAEPs, 100 µ.sec clicks produced by rarefaction at a rate of 11.3 clicks/sec were used (a total of 2000/run in a single run per ear), while MAEPs were evoked by 10 msec 500 Hz rarefaction tones produced at a rate of 9.7 tones/sec with 4 msec ramps and 2 msec plateaux (a total of 1000/run in a single run per ear). Prior to MAEP recording, the subjects" adaptation to MAEP stimuli was facilitated by subjecting them to a series of 100 unrecorded preparative stimuli.

EEG recording procedure

EEG activity was recorded using a Nicolet Compact Four apparatus with Ag/AgCI electrodes. The active electrode was connected to the vertex, the earth (ground) to the forehead and the reference electrode to the ipsilateral earlobe. Impedances were kept below *5* k.!J. BAEP signals were amplified with a gain of 100 K and fed through a filter passing the 150-3000 Hz band. For MAEP signals, the gain was 20 K and the bandpass was 10-250 Hz with a 12 dB/octave roll-off filter.

Data analysis

The BAEP data recorded here are the latencies of peaks I, 111 and V (identified as the positive overall maxima in the intervals 1.2-2.2, 3.2-4.4 and 5.2-6.3 msec post stimulus respectively) ant the inter-peak intervals 1-111. 111-V and J -V . The MAEP data are the latencies of Na and Pa, the inter-peak

TABLE 1

Two-way analyses of variance (2 groups X 2 sides) for BAEP and MAEP parameters.

Variables	Source of variance	*df* = 1 *F*	*p*
BAEPS			
I	Group (G)	2.35	0.13
	Ear (E)	2.60	0.11
	GxE	1.27	0.26
III	Group	0.94	0.33
	Ear	0.24	0.62
	GxE	0.01	0.91
V	Group	9.13	0.004
	Ear	0.00	0.98
	GxE	0.02	0.87
I-III	Group	1.20	0.27
	Ear	3.69	0.06
	GxE	0.87	0.35
III-V	Group	13.35	0.000
	Ear	0.38	0.54
	GXE	0.24	0.87
I-V	Group	4.34	0.041
	Ear	1.01	0.31
	GXE	0.84	0.36
MAEPs			
Na	Group	47.13	0.000
	Ear	0.01	0.91
	GXE	0.48	0.49
Pa	Group	13.98	0.000
	Ear	1.49	0.22
	GxE	0.04	0.84
Na-Pa (interpeak)	Group	1.99	0.16
	Ear	3.14	0.08
	GXE	0.87	0.35
Na-Pa (amplitude)	Group	3.56	0.06
	Ear	0.28	0.59
	Gx E	0.12	0.72

TABLE II

Mean (± S.D.) BAEP and MAEP parameters for alcoholic patients and controls, together with, for each parameter, the number of patients with values more than 2.5 control group S.D.s from the control group mean. N = 15.

	Alcoholics Mean ± S.D.	Controls Mean ± S.D.	No. of alcoholic patients with clinical abnormality
BAEPs			
I	1.65±0.17	1.59±0.11	3
III	3.80±0.23	3.76 ±0.13	3
V	5.83±0.32	5.58 ±0.23	3
I-III	2.15±0.17	2.17±0.16	1
III-V	2.02±0.15	1.82±0.19	2
I-VI	4.18 ±0.23	3.99±0.21	2
MAEPs			
Na	18.46±2.38	22.14±1.40	11
Pa	28.42±2.82	31.01±2.03	5
Na-Pa (interpeak)	9.96±1.37	8.87 ± 1.33	1
Na-Pa (amplitude)	3.18± 1.60	2.49± 1.30	3

latency Na-Pa and the Na-Pa amplitude. The Na peak was identified as the negative overall maximum in the interval 15-25 msec after stimulus onset, and Pa as the positive overall maximum following Na in the interval 22-40 msec after stimulus onset. Since the 10 msec stimuli generated artifacts in the first few milliseconds of the traces. Po was not considered.

After visual identification of BAEP and MAEP components in the recorded traces, the data for each dependent variable were subjected to 2-way analysis of variance to test for any influence of side (right or left) or group (patients or controls). or the modulation of the group effect by side. The Pearson correlation matrix fo all the variables studied was also calculated.

Results

The components studied were clearly identified in all subjects. The general structure of both BAEP and MAEP traces was normal, though the alcoholic group exhibited greater variability.

Alcoholic group BAEP values were significantly greater than those of the control group for the peak V latency (F (1, 28) = 9.13; $P < 0.005$) and for the inter-peak intervals IJI-V (F (1, 28=13.35; $P < 0.001$) and I-V (F (1, 28) =4.34; $P < 0.05$) (see Table I and Fig. 1). The latencies of peaks 1 and III were also greater in the alcoholic group than in controls, though the difference was not statistically significant.

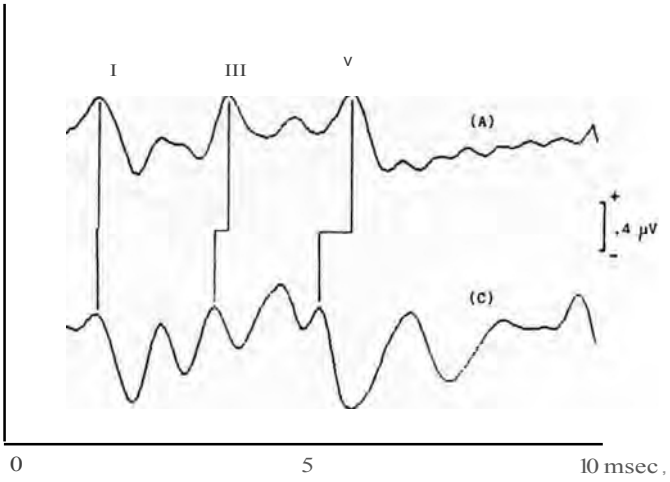

Fig. 1. Typical BAEPs of an alcoholic patient (A) and the corresponding control subject (C), showing the increased latency of peak V and the increased inter-peak intervals III-V and I-V.

Alcoholic group MAEP values were significantly smaller than those of the control group for the latencies of both Na (F (1, 28) = 47.13; $P < 0.001$) and Pa (F (1, 28) = 13.98; $P < 0.001$) (see Table I and Fig.2). The 2 groups did not differ significantly as regards either the inter-peak interval Na-Pa or the Na-Pa peak-to-peak amplitude (F (1, 28) = 3.56; $P = 0.06$), though the latter was on average greater in the alcoholic group.

No consistent Pearson correlation between BAEP and MAEP components was found.

In terms of normal ranges defined, for each variable, as the control group mean ±2.5 control group S.D.s, some kind of clinical BAEP abnormality was exhibited by 7 of the 15 patients (3 had prolonged peak I latencies, 3 prolonged peak III latencies and 3 prolonged peak V latencies) and some kind of MAEP abnormality by 11 (all 11 had shortened Na latencies, 5 shortened Pa latencies, 1 a prolonged Na-Pa interval and 3 enhanced Na-Pa amplitudes).

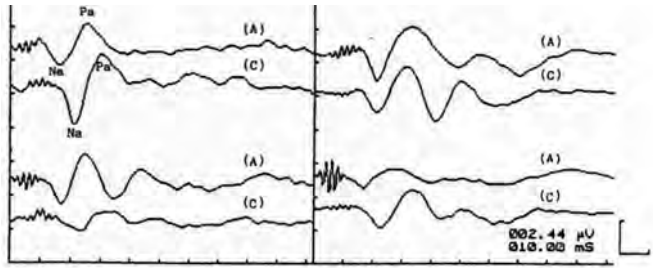

Fig. 2. MAEPs of 4 randomly selected alcoholic patients (A) and of the corresponding age and sex-matched controls (C). Three of the 4 patients exhibit considerable shortening of Na and Pa latencies.

Discussion

There is widespread agreement in the literature concerning the kind of change that is produced in BAEP traces by chronic alcohol consumption. Our results agree with those of most other authors in showing abnormal increases in the Ill-V and 1-V intervals (Begleiter et al. 1981; Chu et al. 1982; Touchon et al. 1984; Chan et al. 1985; Chu 1985; Mabin et al. 1985) and a significant delay in the appearance of peak V (Begleiter et al. 1981; Touchon et al. 1984; Mabin et al. 1985). We did not detect the increase in the I-III interval reported by others in alcoholics suffering from Wemicke-Korsakoff syndrome (Chan et al. 1985) or ethylic epilepsy (Touchon 1984), perhaps because our patients had few organic and/or psychic anomalies. The clinical incidence of BAEP alterations was likewise similar to that reported in other studies of alcoholics with similar characteristics, and less than that found in patients with associated neurological complications (Chu and Squires 1980).

The agreement between the present BAEP results and those of other authors and the kind of pathogenic mechanisms usually suggested to explain BAEP alterations (de-myelinization, axon death. changes in membrane properties) led us to expect that the Na and Pa latencies of alcoholics would be increased, whereas they were in fact consistently shortened in most of our patients. The only previous report of alcohol-induced shortening of evoked potentials concerns BAEPs in rats during withdrawal from chronic alcohol intoxication (Chu et al. 1978). However. in a study in which 6 stimulation conditions were employed (SO and 60 dB presented mon- and binaurally). Woods and Clayworth (1986) reported that, with respect to 20-40 year olds, 60-80 year olds had increased Na-Pa amplitude and increased Pa latency under all 6 conditions and shortened Na latency under three, and they tentatively attributed these changes lo structural or neurochemical alterations in the thalamic GABAergic system.

The available data concerning che effect of alcohol on GABA metabolism suggest that the MAEP alterations observed in alcoholics may come about through a mechanism similar to that proposed for aging-induced MAEP alterations. Alcoholics are known to suffer from GABA deficiency, either because its synthesis is prevented by lack of vitamin B6 (the cofactor of GAD) or because its degradation is accelerated by increased GABA-T activity (Supavilai and Karobath 1980), or both: and one or the brain's major GABAergic centres is the thalamic reticular nucleus, which strongly inhibits neurones in thalamic relay nuclei (Yingling and Skinner 1977), including the medial geniculate nucleus (Shosaku and Sumitomo 1983), which has been reported lo be involved in the generation of the Na wave (Polich and Starr 1983). Though the neural origin of Na is still not known in detail, the hypothesis of de-inhibition of Na due to GABA deficiency seems to be worth looking into in future studies.

Another mechanism possibly involved in alcohol-induced MAEP changes is loss of CNS neurones and neuronal connections, which is known to affect highly ramified circuits such as the thalamic reticular system, particularly acutely (Freund 1984); this hypothesis is supported by Acker et al.'s (1984) CT canning evidence or a reduction in thalamic density in alcoholics. The fact that the MAEP alterations observed in the present study were evident after a whole month's abstinence, when withdrawal had already been completed, suggests that in this case the chief cause may have been structural degeneration.

The significant shortening of Na and Pa latencies, observed in the alcoholics we studied, was accompanied by the lengthening of the Na-Pa interval. Although the latter effect was not statistically significant in this study, it suggests that the MAEP alterations described may be due chiefly to degeneration of the Na generator or generators. The absence of any correlation between BAEPs and MAEPs agrees with previous results for BAEPs, MAEPs, LAEPs, VEPs and CNV in alcoholics (Cadaveira et al. 1987; Diaz et al. 1988) and supports the notion that alcohol has different effects on different levels of information processing in the brain (Freund 1984). More light might be thrown on these phenomena by explorations using stimulation by clicks together with a filter system passing both MAEPs and BAEP peak V.

To sum up, the above data suggest that both BAEPs and MAEPs are useful for evaluating the effects of chronic alcohol consumption on the auditory pathway, and that further studies should be carried out to elucidate the mechanisms of the alterations observed.

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