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SOME OBSERVATIONS ON CLINICAL, COGNITIVE AND NEUROPHYSIOLOGICAL CHANGES IN SUBJECTS CONSUMING INDIGENOUS ALCOHOL

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Abstract: Sixtytwo subjects in age range of 25-50 years consuming more than 300 ml of alcohol daily, and an equal number of age matched non-alcoholic volunteers serving as control were tested. Their clinical and neurological evaluation, including electrophysiology was carried out. Their cognitive functions were measured using the modified WAIS system. In alcoholics there was a significant impairment of cognition, especially in orientation, attention and immediate recall. Their P_{300} wave was grossly abnormal as compared with the controls. Other electrophysiological investigations (EEG, NCV, EMG, BAER, VER) were normal. It is concluded that cognition may be grossly impaired in chronic alcoholics, which may not manifest clinically but is observed only after formal testing.

Key words: alcohol cognition P_{300} wave

INTRODUCTION

Many neurological problems and diseases have been associated with alcohol toxicity : such as neuropathy, myopathy, optic neuritis, encephalopathy, cerebellar and cortical degeneration, etc. A greater part (85%) of alcohol is metabolized in the liver to acetaldehyde. Its accumulation in brain, peripheral nerves and other nervous tissues can cause organ damage. Ethanol either alone or in combination with other agents is probably responsible for more toxic overdose deaths than any other agent (1). The mechanism of alcohol toxicity on nervous tissue is not fully understood, but presumably it is because of simultaneous increase in fluidity of neuronal cell membranes and change of neurotransmitters (2). Role of alcohol in producing dementia and alteration of cognition is controversial, with a variety of clinical circumstances being implicated, including toxic degeneration, gastrointestinal disease and above all dietary deprivation and neglect of nutritional needs especially by chronic alcoholics (1, 2). The present study was designed to observe the effects of intake of indigenous liquor on cognition and other neurophysiological changes.

METHODS

62 male alcoholics formed subjects of this study. An equal number of non-alcoholic volunteers were used as controls. Their age range was 25-50 years. Alcoholics had been consuming more than 300 ml of country liquor daily for more than 5 years prior to this study. They were residents of/or around the district of Lucknow. All alcoholics were randomly selected from the out patient section of Medicine and Neurology Departments. Nonwilling and with serious effects (eg. Hepatic coma) were excluded. The subjects were explained the protocol after obtaining their informed consent. A thorough clinical and neurological evaluation was done in all the subjects. Cognition was measured using the modified WAIS system. It consisted of a questionnair on a written format printed in Hindi, which comprised of questions as a test of Orientation,

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Attention, Concentration, Comprehension, Memory (Immediate, Recent and Remote) and GK (Intelligence and Calculation). The questions had been so designed to be easy to understand. Answers were recorded and scores awarded. P_{300} studies were also undertaken on them using Neuropack 4 machine (NIHON KOHDEN, Nishi-0-Chial Chome, Tokyo, Japan), using regular and odd auditory stimulus with subjects being asked to respond only to the odd auditory stimulus which produced the P_{300} wave. NCV, EMG, VER and BAER were also measured on the same machine. Averaging, analysing, cursor marking and measurements were all done by the computer. Repetition was done as and when required.

RESULTS AND DISCUSSION

The total number of alcoholics entering the study was 90. Out of them 28 dropped out because of -Fear of identification/report 6 (6.6%). Lack of interest 9 (10.0%), Lack of confidence 9 (12.2%), unknown 2 (2.2%) or they became seriously ill later on (Peptic ulcer and Hepatic coma) 2 (2.2%). The present study and observations pertain to 62 alcoholics.

It was observed that the maximum number of alcoholics were in 2nd and 3rd decade of life (64.1%). The age of first contact with alcohol was 22-25 years. In 85% the mode of introduction was via "friends".

The effects of alcohol are summarised in Table I. It was observed that GIT problems like dyspepsia. anorexia, epigastric pain, vomiting, etc., were the commonest ailment amongst the alcoholics.

Table II summarises the effects of alcohol on P_{300} wave. There was a significant difference in P_{300} potentials of alcoholics and controls ($P < .01$). Besides increased latency and poor reproducibility, the wave form abnormality prevented amplitude measurement in most of alcoholics (Fig. 1, 2).

Cognitive functions and their alterations have been represented in Table II. A decrement of more than 10% from max score was taken as abnormal. There was significant impairment of cognitive functions in alcoholics especially in calculation, immediate recall, and attention.

Alcoholism has been known to impair

TABLE I: Prevalence of different clinical features in the alcoholic patients.

N = 62

Complaints	*Alcoholics effected*	*Percentage*	*Comments*
Dyspepsia	26	41.9%	
Anorexia	16	25.8%	
Epigastric pain	12	19.3%	Amoebic liver abscess in 2 (Sonogram)
Ataxia	9	14.5%	Cerebellar atrophy in 6 (CT).
Vertigo	3	4.8%	
Vomiting	12	19.3%	
Tremors	5	8.0%	
Burning feet	6	9.6%	
Impotence	1	1.6%	
Bodyache/lassitude	16	25.8%	
Hypertension	9	14.5%	
Loss of weight	10	16.1%	
Released reflexes	14	22.5%	
None	3	4.8%	

TABLE II: Effect of alcohol on cognitive functions and P_{300} potentials.

Parameters	*Controls (N=62)*	*Alcoholic (N=62)*
Orientation	-	10 (16.1%)*
Attention	-	14 (22.5%)*
Immediate recall	-	18 (29.0%)*
GK	3 (4.8%)	6 (9.6%)*
Intelligence	3 (4.8%)	12 (19.3%)*
Calculation	4 (6.4%)	19 (30.6%)*
Comprehension	4 (6.4%)	12 (19.3%)*
P_{300} Max. latency	348.6 ms	486.4 ms*
P_{300} Min. latency	302.4 ms	368.2 ms*
P_{300} reproducibility	62 (100%)	23 (37%)*
P_{300} Abnormal wave form	Nil	45 (72.8%)*
P_{300} Average latency	322.6 ms	398.2 ms*
P_{300} Prolonged latency (more than 320 ms)	10 (16%)	56 (90.3%)*

*P_{300}<0.1

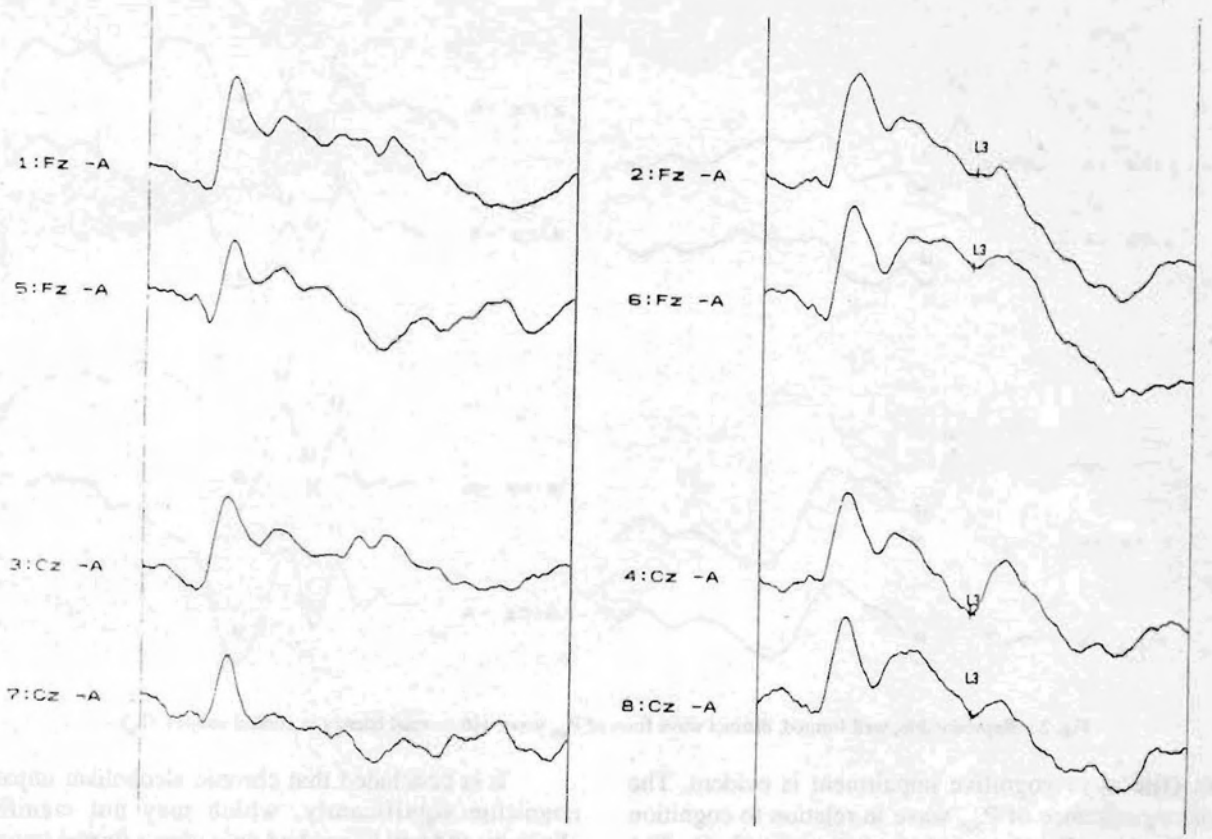

Fig. 1 : Non-reproducible, indistinct, ill formed wave form of P_{300} wave with prolonged latency in an alcoholic. (L_3)

cognition since long. Even current Neurobehavioural investigations on chronic alcoholics indicate that long continued use of alcohol produces intellectual deficits that are at least partially reversible. Clinically obvious dementia occurs in 3% of alcoholics, its typical characteristics being forgetfullness, psychomotor retardation, circumstantiality poor attention, and disorientation (3). Country made liquor may not have uniform composition and may contain several neurotoxic substances responsible for the present observations on cognitive dysfunctions, in which significant impairment of cognition after a detailed formal testing was observed. Other workers have demonstrated the impairment of cognition in 59% of alcoholic patients after conducting different tests of Neuropsychological functions (4). Though legal intoxication requires a blood level of alcohol concentration around 80-100 mg/dl, cognitive, psychomotor and behavioural changes are observed much earlier and at lower levels. These changes may not be obvious clinically in initial phases (3, 4).

In the present study, no significant alterations in peripheral nerves were observed as tested clinically or electrophysiologically by NCV or EMG. Even evoked potentials, viz., VER and BAER were essentially normal. The records were also normal in all. However, the P_{300} wave potential was distorted in various parameters in comparison to that of control subjects. Changes in P_{300} wave have been documented by several workers (5) and these changes may appear much earlier

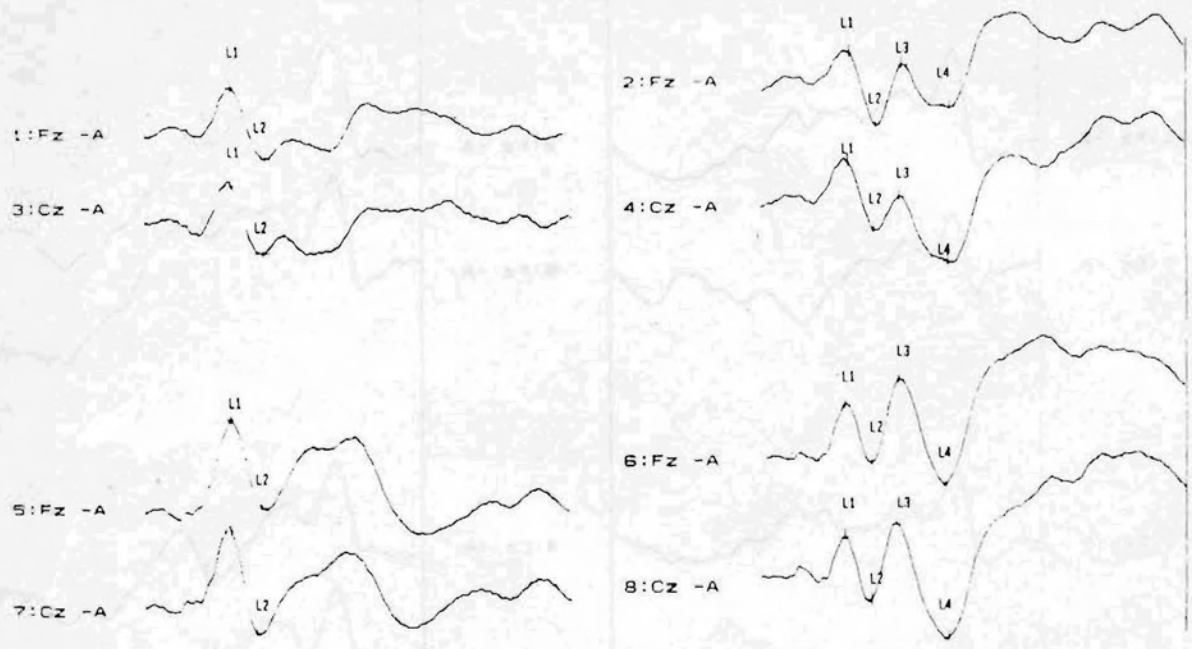

Fig. 2 : Reproducible, well formed, distinct wave form of P_{300} wave with normal latency in control subject. (L_4)

than clinical or cognitive impairment is evident. The exact significance of P_{300} wave in relation to cognition is still not well known, nor is its origin (5, 6). The sites postulated are the frontal, thalamic posterior parietal, cerebellar, hippocampus and limbic regions. It is presumed that degeneration in these regions may be occuring which may explain the high incidence of released reflexes, cerebellar signs, cognitive impairment, and the distorted P_{300} wave.

It is concluded that chronic alcoholism impairs cognition significantly, which may not manifest clinically and will be evident only after a formal testing. Also P_{300} wave presumably is the earliest to alter among electrophysiological investigations.

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