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ETHANOL INDUCED ELECTROENCEPHALOGRAPHIC CHANGES FROM CERTAIN CENTRAL LOCI OF CANINE BRAIN

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Abstract : Effect of intracerebroventricular (i.c.v.) ethanol on pre-optic area (POA) and hippocampus (HPC) was investigated by recording chronological changes in their electroencephalographic (EEG) activity, through chronically implanted cannulae and bipolar electrodes, in five concious male beagle dogs. Recordings were made for two hours after microinjection and compared with cortical (CO) tracings. The predominant pattern of EEG activity from subcortical loci was high voltage discharges though synchronization was also observed. Cortical EEG depicted slow waves with some potentiation. The study demonstrated similiar type of response form POA and HPC, which was different from CO, suggesting that ethanol produces different actions at subcortical and cortical level.

Key words : EEG Ethanol POA HPC CO

INTRODUCTION

The behavioural consequences of ethanol are well known. Only few studies with surface EEG recording after the use of ethanol are available (1). The activity of hypothalamic and limbic areas, is influenced by neuronal and humoral inputs (2,3). In the present study, locr from hypothalamic and limbic system were selected for observing their responses to i.c.v. ethanol, as compared to the response of the cortex.

METHODS

Five male beagle sexually mature dogs, weighing 8-10 kg, were domesticated in rooms which were well ventilated, and fed with bread, milk and water *ad-lib*. For stereotaxic implantation of electrodes and cannulae the animals were anaesthetized with intraperitoneal injection of sodium pentobarbitol (35 mg/kg). Bipolar stainless steel electrodes, with their tips scrapped about 1 mm, and cannulae, were stereotaxically implanted in POA and dorsal HPC, and third ventricle respectively, as per the stereotaxic atlas of dog (4), using techniques of Anand (5) and Antunes-Rodrigues and McCann (6). For cortical EEG screws were placed in parietal area and a third screw was implanted in nasal bone which served as reference ground. When the animals had recovered from the effects of surgical trauma, they were trained for EEG recording procedures in self restrained, undisturbed conditions. The dogs were observed through one-way viewing window of the room. EEG of different areas of brain was recorded on a polyrite after i.c.v. microinjection of vehicle (distilled water) and 10% ethanol (50 μl/dog). EEG was recorded for half an hour prior to and 30, 45, 60, 90 and 120 min after vehicle and ethanol injections. Each dog was first treated with the vehicle, and then next day at the same time with the use of ethanol. Each dog thus served its own control. After the completion of experiments electrode sites were confirmed histologically (3). The EEG records were analyzed using a procedure already described (3,7). Frequency and amplitude changes of EEG of ethanol treated group were statistically compared through Student's 't' test with basal and vehicle treated controls respectively. The analysis of maximum changes in frequency and voltage were made separately and illustrated by representative tracings of EEG record.

RESULTS

Pre-optic changes : After half an hour following

the application of Ethonol, E.E.G. record showed discharges (15-20 Hz, 50-200 μV), ranging from 0.5 to 2.25 sec in duration (Fig. 1). Such discharges were maintained upto one hour. After 90 min, slow waves (1.5-5 Hz, 200-300 μV) replaced these discharges. Conversion of slow waves into seizures (10-30 Hz, 75-150 μV) of 0.8-2.8 sec duration were observed after two hours.

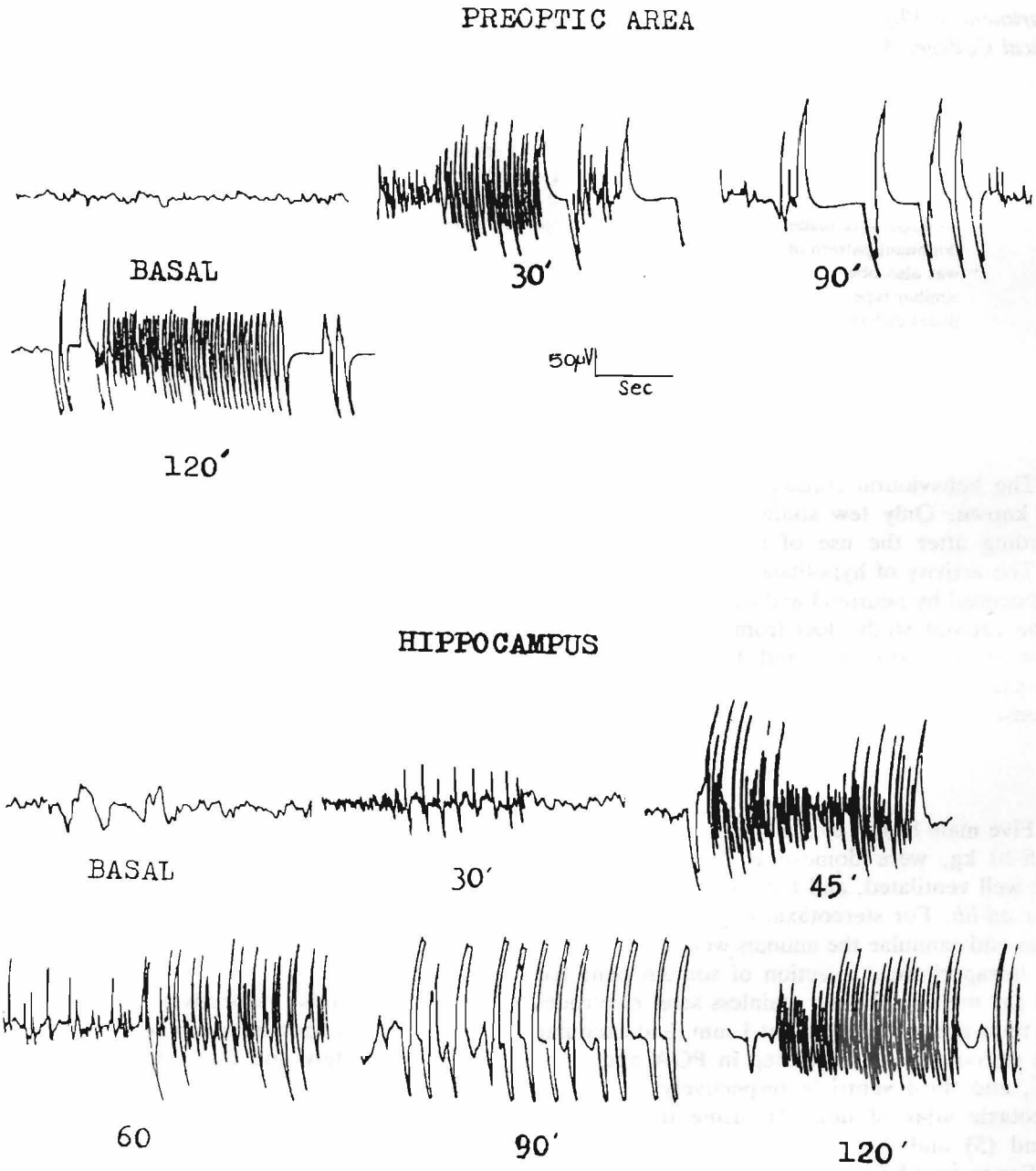

Fig. 1: Showing effect of intracerebroventricular ethanol on electrical activity of pre-optic area and hippocampus.

TABLE I : Mean frequency (cps) and amplitude (μV) of whole record taken from observations in dog after vehicle and ethanol treatment.

Area	*Group*	*Before treatment*		*After reatment (min)*									
		Basal		*30*		*45*		*60*		*90*		*120*	
		F	A	F	A	F	A	F	A	F	A	F	A
Pre-optic	Vehicle	9.78	10.27	9.82	10.18	10.02	9.65	9.95	10.67	8.89	11.72	8.51	14.82
		±1.32	±1.76	±1.21	±1.54	±1.45	±1.62	±1.43	±1.44	±1.36	±1.76	±1.52	±2.21
	Ethanol	10.12	11.24	18.66*	121.12**	18.41*	119.35*	17.19	128.26*	4.75	267.45***	18.22*	251.12**
		±1.29	±2.12	±2.33△	±18.79△△	±2.52△	±20.22△	±2.71	±25.67△	±0.62△	±29.11△△△	±3.12	±28.22△△
Hippocampus	Vehicle	7.60	25.90	7.54	22.34	8.21	21.56	8.74	20.77	6.81	26.82	6.52	28.72
		±1.21	±3.22	±1.32	±2.98	±1.46	±3.44	±1.32	±3.81	±0.86	±4.11	±0.94	±4.14
	Ethanol	7.80	26.29	14.62	75.26	19.50*	141.97**	10.25	122.41**	4.12	252.50**	18.55*	212.40**
		±1.26	±3.42	±2.28	±20.86	±3.01△	±22.11△△	±1.52	±18.22△△	±0.58	±30.92△△	±2.43△	±31.45△△
Cortex	Vehicle	9.86	12.52	10.11	12.68	9.62	12.42	8.89	15.33	8.72	15.81	7.82	18.46
		±1.42	±2.42	±1.34	±2.39	±1.22	±2.62	±1.17	±3.21	±1.25	±3.11	±0.91	±3.21
	Ethanol	10.21	11.81	10.77	11.54	10.12	10.89	9.42	18.29	7.49	40.76**	5.21	59.27**
		±1.51	±2.26	±1.62	±2.19	±1.42	±2.46	±1.31	±3.11	1.12	4.41△△	0.61△	6.49△△

Values are given as Mean ±SE
F = Frequency A = amplitude
Basal versus ethanol treated at corresponding time intervals △ P <0.05, △△P<0.01, △△△ P<0.001,
Vehicle treated versus ethanol treated at corresponding time intervals *P>0.05, **P<0.01, ***P<0.001.

Hippocampal changes : After half an hour, following the application of Ethanol low voltage discharge frequency coupled with spikes (15-20 Hz, 15-100 μV) was seen (Fig. 1). High voltage discharges (15-25 Hz, 50-275 μV) were observed after 45 min and slowing of EEG occured after 60 min. This slowing further progressed after 90 min and regular waves (2-5 Hz, 200-250 μV) became apparent. Seizures (15-25 Hz, 100-225 μV, 1.5-2.5 sec) replaced slow waves after two hours.

Cortical changes : From cortex no appreciable topographic change in EEG was observed, except for progressive slow slowing and potentiation (Table I).

Statistical evaluation of frequency and amplitude changes from the loci investigated are represented in Table I.

DISCUSSION

Both the central loci selected (POA and HPC), responded more or less similarly i.e., with repeated occurence of high voltage discharges alongwith short staying slow waves. Various inputs and modulations from anterior hypothalamus to HPC (8) might have aroused similar type of response in HPC as that of POA.

Some of the metabolites of ethanol, though not produced in functionally significant quantities in central nervous system, are reported to elicit, electrophysiological changes (9). Neuronal response maintained for more than two hours suggests its initiation by ethanol, later on being maintained by its metabolite acetaldehyde (10), which is known to be effective releaser of catecholamines (11). However, at the cortical level, some metabolite, other than acetaldehyde, may be operating (1), resulting in the observed synchronization. The present study suggests different mechanisms operating at subcortical and cortical levels. Further exploration is required to explain similar type of response from POA and HPC. Response of other loci from hypothalamic and limbic system will further add to the present observations.

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