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Effects of Moderate Alcohol Dose on Albino Rats Visual Cortex

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Abstract: The present study aimed to evaluate the possible effects of ethanol consumption on the photic response of the albino rats' visual system. Changes in Visual Evoked Potential (VEP) -amplitudes and latencies were measured after 1.75 g/kg dose of ethanol via oral rout administration. After ethanol consumption, rats showed increases in inter-hemispheric transmission times and as well as flash-lags. The present findings agreed with the suggestion says alcohol reduces neural processing.

Key words: Ethanol · Pattern-Shift · VEP · Occipital Cortex

INTRODUCTION

The shift/changes in visual evoked potentials patterns have been confirmed to be useful when lesions of the optic nerves or visual cortices are suspected [1]. Extensive clinical literatures exist telling their use in detecting and defining the neurologic effects of many disorders as glaucoma, optic neuritis and occipital lobe tumors. Other literatures describe the possible effects of drugs on pattern-shift visual evoked potentials.

Recent studies have assessed Pattern-shift visual evoked potentials in patients exposed to ethanol [2]. The changes of waveform and latency abnormalities have been reported [3]. Recent studies showed that the average PI00 latency difference between alcohol consumers and non-alcoholic was small and sometimes statistically insignificant [4]. We can say in general, the previous studies focused on middle-aged alcoholic patients during the first three weeks of abstinence. However, one study assessed alcoholic patients after a longer period of alcohol consumption termination where the majority of patients showed normalization in P100 latency within 6 months after alcohol consumption termination [5].

Using Pattern-shift visual evoked potentials to evaluate alcohol possible effects were suggested by many studies using photic flash-evoked electroence phalographic (EEG) responses in adult rats [6-8].

The present study was carried out to evaluate the possible effects of ethanol administration on pattern shift visual evoked potentials of albino rats.

MATERIALS AND METHODS

Male adult albino rats (n=20) *Rattus norvegicus* (above 150 g in weight) were used in the current study. Animals were picked up from the experimental animal breeding station at Helwan, Cairo, Egypt. Animals were housed as a single rat/cage in a standard Plexiglas cage [25 (w) × 20 (h) × 45 cm]with light metal mesh as covering part. Except during the EEG recordings there was free access to balanced standard maintenance of food and tap water.

Animals were then divided into two groups called control group (n=5) and alcohol group (n=15). Under deep sodium pentobarbital anesthesia (50 mg/kg, intraperitoneally), rats were implanted bilaterally with epidural stainless steel screw electrodes over the visual cortex (B: 2 mm, L: 3 mm) for EEG chronic recordings. A third electrode (reference electrode) was placed in the contralateral crest of the skull at the interaural line (asterisk in Fig. 1).

Electrodes were fixed to the skull via dental acrylic cement. One-week as a recovery and a training period was provided before the effects of alcohol were studied. This method was adopted after Skinner [9].

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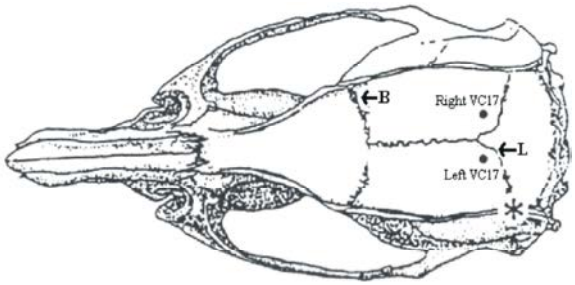

Fig. 1: Locations of the EEG recording electrodes

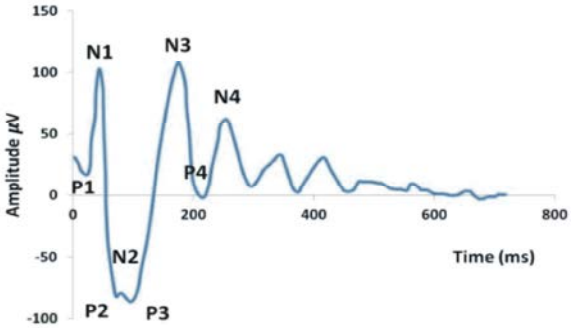

Fig. 2: The normal rats averaged visual evoked potential (VEP) recorded from the visual cortex. The graph represents an average of 20 responses.

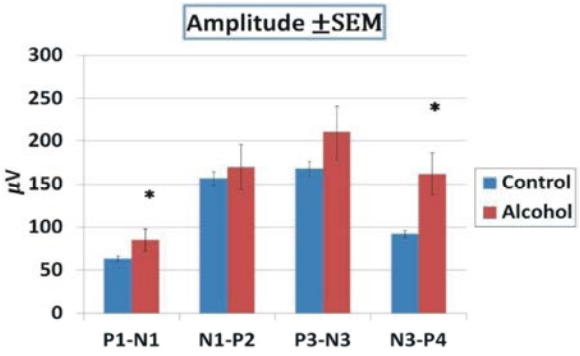

Fig. 3: Effects of alcohol on the amplitude of early and late VEP components. The star (*) indicates significantly difference from the control group with P< 0.05.

Rats were placed in a dark, and earthed metal box (60 cm×60 cm× 60 cm). Light flash photic stimulations were delivered by photic stimulator (Grass PS40, USA). The rate of stimulation was one flash each 0.75-1.25 s. VEPs patterns were stored on a computer disk with a sampling frequency of 512 samples/s. 15 single of VEPS per animal per channel were averaged off-line after a process of noise elimination. VEP patterns were recorded 30 min later after alcohol administration.

Each rat in the alcohol group (n=15) received 1.75 g/kg alcohol via oral rout administration. This dose of alcohol was used to test the moderate effects of alcohol on cortical EEG. Many recent studies reported that, this dose is considered to be a moderate dose [9-11]. All animals should be alcohol-free prior to acute alcohol administration which was followed by EEG and visual evoked potential recordings.

RESULTS

Fig. 2 represents the average normalized VEP wave form, mean peak latency values, as well as the amplitude. VEP wave form recorded from the rat visual cortex consisted of many well identified patterns namely P_1– N_1, N_1–P_2, P_2–N_2, N_2–P_3, P_3–N_3 and N_3–P_4 components.

However, since P_2–N_2, N_2–P_3 and P_4-N_4 components were highly unstable and difficult to be well recognized, therefore, we did not take them in to our concern to assess them. Those VEPs pattern components were identified visually then scored with a cursor on the monitor.

The amplitude of each component is the average of the first 15 ms of the recording epoch. Differences in the amplitude and latency of the VEP components were evaluated using analysis of variance (ANOVA) program. Statistical significance was assumed at $P < 0.05$.

Fig. 3 shows that alcohol consumption produced increases in the amplitude of P_1–N_1, N_1-P_2, P_3-N_3, as well as N_3-P_4.

Some authors prefer to call P_1-N_1 and N_1-P_2 as the early components. While they called P_3-N_3 and N_3-P_4 as the late components of the photic VEP. As we can see there were elevations in the amplitudes of both early and late VEP components after alcohol consumption. However the increases in P_1-N_1 and N_3-P_4 were significant.

Fig. 4 shows that alcohol administration yielded some changes in the VEP latency components. There were significant increases in the VEP latency of N_1 , N_3 and P_4 components.

Table 1 represents a comparison of one of the VEP components (N1) on both side of the occipital cortex. This comparison includes latency and amplitude between control and alcohol groups. Although alcohol group had a slightly prolonged VEP N1 latency compared to control group, this difference in VEP latency was not found to be significant. Higher VEP amplitude was observed in alcohol group, but the difference between groups was not statistically significant.

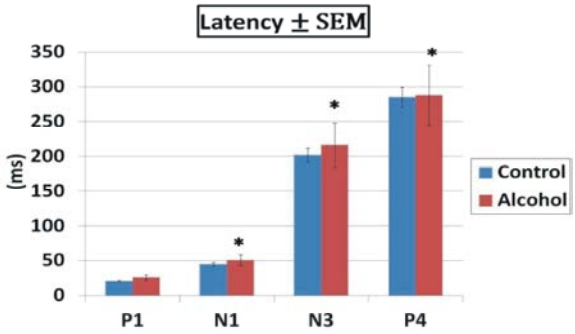

Fig. 4: Effects of alcohol on the latency of early and late VEP components. The star (*) indicates significantly difference from the control group with P< 0.05.

Table 1: Comparison of VEP latency and amplitude between control and alcohol groups

		Mean SD		
VEP-N1		Control (n=5)	Alcohol (n=15)	*P*-value
Amplitude	Right	153. 85 (3. 68)	164. 65 (11. 39)	0. 508
	Left	157. 70 (4. 22)	168. 95 (12. 45)	0. 808
Latency	Right	43. 1 (6. 5)	49. 5 (6. 5)	0. 254
	Left	40. 7 (7. 5)	45. 1 (8. 7)	0. 092

DISCUSSION

The present study revealed that, ethanol consumption elevated the amplitude of the VEP early components (P_1–N_1, N_1–P_2) as well as the late components (P_3–N_3, N_3–P_4).

It has been suggested that changes in the early VEP components reflect altered sensory input, while late component variations are related to behavioral state or psychological variables [11]. The visual evoked potential (VEP) recording is widely used in clinical practice to assess the severity of optic neuritis in its acute phase and to monitor the disease course in the follow-up period. Changes in the VEP parameters closely correlate with pathological damage in the optic nerve [12].

There is important clinical evidence suggesting that substance abuse may produce neurophysiologic disturbances particularly in relation to altered neural synchronization in Visual Evoked Potentials (VEP) [13, 14].

The variation of late VEP amplitudes seems to relate well to the attentional and behavioral changes found during alcohol intoxication. However, the VEP latency results indicate that the effects of alcohol are more complex than is indicated by the amplitude data alone. This means a greater sensitivity of the early components than the late components to the effects of alcohol, a pattern almost exactly opposite that found with variations in amplitude [15, 16].

It is generally recognized that VEP is generated in the visual cortex [13]. However the latencies of the early peaks of rat VEPs were more consistent, which may be due to their generation in the primary visual cortex via the retinogeniculate fibers [17].

It is well known that alcohol induces dose-dependent mydriasis in rats. From these findings, alcohol-induced mydriasis may be partially responsible for the increase of both early and late components of VEP amplitude [18-20].

The effects of alcohol on latency were more obvious than did the effects of alcohol on amplitude. Specifically, amplitude effects were readily apparent in all components following alcohol consumption. These findings may suggest that, the early component latency changes reflect altered sensory input. The increased VEP peak latencies measured in the current study were small and could easily have gone undetected without the aid of a digitized recording system.

CONCLUSIONS

The current study examined the possible alcohol effects on the VEP recorded from the visual cortex of chronically implanted rats. There were effects of alcohol consumption on the early and late VEP components. Also the latencies of both early and late components were increased. It is therefore possible that visual problems occurring as a result of alcohol intake could be assessed using the VEP technique.

Abbreviations:
EEG-: Electroencephalogram
VEP-: Visual evoked potential
ANOVA-: Analysis Of Variance
SD: Standard Deviation

REFERENCES

1. Chiappa, K.L., 1990. Pattern shift evoked potentials: Interpretation. Evoked Potentials in Clinical Medicine. Raven Press, New York, pp: 111-172.
2. Cadaveira, F., M. Roso, C. Grau and M. Sanchez-Turet, 1992. Effects of age on event-related potentials in chronic alcoholics: a multimodal study. Neuro-Psychobiology, 25: 166-171.

3. Cadaveira, F., C. Grau, M. Roso and M. Sanchez-Turet, 1991. Multimodality exploration of event-related potentials in chronic alcoholics. Alcohol Clin. Exp. Res., 15: 607-611.
4. Meinck, H.M., K. Rader, G. Wieditz and L. Adler, 1990. Afferent information processing in patients with chronic alcoholism: An evoked potential study. Alcohol., 7: 311-313.
5. Bauer, L.O. and C. Easton, 1996. Pattern shift visual evoked potentials in abstinent cocaine-dependent, alcohol-dependent and cross-dependent patients. Drug and Alcohol Dependence, 40: 203-209.
6. Bauer, L.O., 1993a. Photic driving of EEG alpha activity in recovering cocaine dependent and alcohol dependent patients. Am. J. Addict., 3: 49-57.
7. Bauer, L.O., 1993b. Electroencephalographic evidence for residual CNS hyper-excitability during cocaine abstinence. Am. J. Addict., 2: 287-298.
8. Bauer, L.O., 1995. Psychomotor and electroencephalographic sequelae of cocaine dependence. In, Neurotoxicity and Neuropathology Associated With Cocaine/Stimulant Abuse. Ed., Majewska, M.D., NIDA Res Monogr. US Government Printing Office, Washington, DC.
9. Skinner, J.E., 1971. Neuroscience: A Laboratory Manual.; Saunders Company, Philadelphia.
10. Charng, J., C.T. Nguyen, Z. He, T.M. Dang, A.J. Vingrys and R.L. Fish, 2013. Conscious Wireless EEG and Visual Evoked Potentials in Rats. PLoS ONE, 8: 1423-1437.
11. Kuroda, K., A. Fujiwara, Y. Takeda and C. Kamei, 2009. Effects of narcotics, including morphine, on visual evoked potential in rats. European J. Pharmacology, 602: 294-297.
12. You, Y., J. Thie, A. Klistorner, V.K. Gupta and S.L. Graham, 2015. Visual evoked potential recording in rat model of experimental optic nerve demyelination. J. Vis. Exp., 101: 1319-1328.
13. Corda, M. , G. Piras, M. Piludu and O. Giorgi, 2014. Differential Effects of Voluntary Ethanol Consumption on Dopamine Output in the Nucleus Accumbens Shell of Roman High- and Low-Avoidance Rats: A Behavioral and Brain Microdialysis Study. World Journal of Neuroscience, 4: 279-292.
14. Garg, S., R. Sharma, S. Thapar and Sh. Mittal, 2016 . Visual evoked potential response among drug abusers- a cross sectional study. Journal of Clinical and Diagnostic Research, pp: 23-29.
15. Kraut, M.A., J.C. Arezzo and H.G. Vaughan Jr., 1990. Inhibitory processes in the flash evoked potential of the monkey. Electroencephalogr. Clin. Neurophysiol., 76: 440-452.
16. You, Y., A. Klistorner, J. Thie and S.L. Graham, 2011. Improving reproducibility of VEP recoding in rats: electrodes, stimulus source and peak analysis. Doc Ophthalmol, pp: 109-123.
17. You, Y., A. Klistorner, J. Thie and S.L. Graham, 2012. Normalization of visual evoked potentials using underlying electroencephalogram levels improves amplitude reproducibility in rats. Invest Ophthalmol Vis. Sci., 53: 1473-1478.
18. Martin, A., C. Balachandran, A.L. Klistorner, S.L. Graham and F.A. Billson, 2003. Effect on pupil size on multifocal pattern visual evoked potentials. Clin. Exp. Optom., 31: 354-356.
19. Kim, J.T., C.M. Yun, S.W. Kim, J. Oh and K. Huh, 2016. The effects of alcohol on visual evoked potential and multifocal electroretinography. J. Korean MedSci., 31: 783-78.
20. Kaarre, O., E. Kallioniemi, M. Könönen, T. Tolmunen, V. Kekkonen, P. Kivimäki, N. Heikkinen, F. Ferreri, E. Laukkanen and S. Määttä, 2016. Heavy alcohol use in adolescence is associated with altered cortical activity: a combined TMS-EEG study. Addict Biol., published online doi: 10.1111/adb.12486.