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THE EFFECTS OF ALCOHOL ON THE SOMATOSENSORY EVOKED POTENTIALS IN MAN

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The effects of alcohol on the electrical activity of the brain have been studied in animals and in man. In a comprehensive review of the animal literature, Himwich and Callison (1972) reported that alcohol in adequate doses depresses the functions of the central nervous system. Begleiter and Platz (1972) reviewed the human literature and concluded that the effects of alcohol on human EEG appear primarily to be an increase in the total amount of alpha activity and as a slowing of its frequency.

In recent years a number of investigators have examined the effects of alcohol on evoked brain potentials in man. Gross et al. (1966) studied the effects of 100 cc of alcohol on the auditory evoked response of ten males. The findings established that alcohol significantly reduces the auditory evoked response. The maximal effects were obtained 15-30 minutes after ingestion of alcohol. Spilker and Callaway (1969) reported that the average evoked response to sine wave light is apparently related to "augmenting/reducing" since augmenters (as defined on a kinesthetic test) have visual evoked responses that increase in amplitude as the depth of modulation is increased. In contrast, "reducers" show a leveling off or an actual decrease in visual evoked responses at high depths of modulation. In an attempt to test the effects of arousal, these investigators studied the effects of various drugs on the amplitude of the visual evoked response. They found that alcohol significantly decreased the slope of the visual evoked response amplitude at the four highest depths of modulation.

Lewis, Dustman and Beck (1970) conducted a study on the effects of two doses of alcohol, 0.41 g/kg and 1.23 g/kg on the visual and somatosensory evoked responses of man. They found no effect with

the low dose of alcohol. With the high dose of alcohol, they observed a significant decrease in the amplitude of a number of late waves of both visual and somatosensory evoked responses recorded from central areas. Visual evoked responses recorded from the occipital area showed no such changes.

A recent report by Salamy, Rundell and Williams (1969) indicates that the late components of the somatosensory response are highly depressed by the administration of alcohol. This effect is highly correlated with the percentage of alcohol in the blood; at blood alcohol concentrations (BAC) as low as .02% this response is already somewhat diminished, at a BAC of .06% the amplitude may be reduced by 50%, at BAC of .12% it is virtually abolished. In contrast, the early components seen with post Rolandic parasagittal electrodes, appear to be little affected by the doses employed.

While it may be said that alcohol significantly reduces the late components of the visual, auditory and somatosensory evoked responses in man, some investigators have argued (Jones and Vega, 1972) that the rate of ascent of blood alcohol concentration is the most important factor in eliciting perceptual and behavioral impairment. The same authors have stated that "the rate of change and direction of change of the alcohol concentration in blood are probably more crucial variables than the length of time alcohol is in the blood stream."

The purpose of the present study was to examine changes in somatosensory evoked potentials with a fairly constant blood alcohol level for a period of three hours.

METHOD

The experiment was performed on 14 healthy adult male volunteers with a mean age of 33 years and a mean weight of 174.8 pounds. All were accustomed to very mild alcohol consumption. Each subject was tested in a sound attenuated, electrically shielded room.

Somatosensory responses were evoked by stimulating the median nerve of the right wrist through electrodes placed on the skin 3 cm apart (anode distal). A ground electrode was placed proximal to the cathode. The stimulus was a pulse of 1 msec duration at an intensity 3 ma above the subject's thumb twitch threshold. The source of the pulse was a constant current stimulator, triggered and timed by means of a Grass S8 stimulator and isolation unit. Recording electrodes were placed in the parasagittal plane 7 cm left to the midline. Bipolar recordings were obtained between one lead 2 cm behind a line from the vertex to external auditory meatus and the other was 6 cm anterior to it. The EEG was amplified with a

Grass Model 78 and fed into a computer (CAT 1000) for summation. Analysis time of 512 msec was used. In order to obtain accurate measurements of the responses, a calibration pulse was placed on each sweep in the last 20 ordinates.

Stimulus repetition frequency was variable from 1 to 3 sec. Each run contained a total of 50 stimuli, and each recording session consisted of 12 such runs over a period of three hours.

Alcohol Administration

Since it became important for us to maintain a fairly constant blood alcohol level for a period of 2 hours, we conducted a pilot study with the use of 8 subjects. Multiple dose administration with doses spaced so as to maintain a constant blood alcohol level was given in accordance with the method developed by Goldberg (1960, 1961).

In the evoked response experiment, the subjects received alcohol (80^{o} bourbon) on one morning and an equal amount of water on the other morning. These two conditions were counterbalanced for all 14 subjects. Before the electrodes were placed on the scalp, each subject was asked to drink an amount of alcohol in the ratio of 1.5 gm of alcohol per kg of body weight. The subject was given a period of 5 minutes to finish his drink. Approximately 60 minutes after the intake of alcohol, the first blood alcohol level was determined with the use of a Breathalyzer. The second dosage of alcohol given was calculated on the basis of the first blood alcohol level. Two hours after alcohol intake another blood alcohol level was obtained which determined the amount to be given in the third dose. The second and third doses of ETOH were always very small, never exceeding 30 ccs.

RESULTS

In order to determine the effect of alcohol in the somatosensory evoked potential, an analysis of variance was performed comparing the 12 alcohol with the 12 water runs. A statistically significant F value (6.22) at $p < .05$ was obtained between the two treatments. The 12 runs were not found to differ significantly from each other, while individual differences between subjects was found to be significant at $p < .01$ ($F = 6.99$).

The interaction between treatments and subjects yielded an F value of 12.58 which is significant at $p < .01$. The Run x Subjects interaction was statistically significant at $p < .05$.

Breathalyzer readings of blood-alcohol level were maintained at .05 - .06 throughout the experimental sessions, with a mean value of .055. The full range of all readings over the 14 subjects was .04 - .08.

DISCUSSION

The results of the present investigation confirm previous findings (Salamy, Rundell and Williams, 1969; Lewis, Dustman and Beck, 1970) that the somatosensory evoked potential is highly susceptible to the influence of alcohol. However, unlike previous studies, this experiment has focused on the effects of alcohol in the "primary" components of the somatosensory evoked potential, and a pronounced decrease in the amplitude of the early component was obtained. The component examined has a positive deflection at 20-25 milliseconds and a negative peak at 60-80 milliseconds after the stimulus.

It has been reported that the early somatosensory response (15-80 msec) arises from within the cerebral cortex (Allison, 1962), while later components are extra-lemniscally mediated and of more diffuse thalamic origins. A further distinction as to the origins of the two components forming the "primary" response has been made, based on a detailed analysis of all five components of the somatosensory evoked potential (Goff, Rosner and Allison, 1962). Within the "primary" response, the first component appears to indicate pre-synaptic thalamo-cortical fibers, and the second component to indicate post-synaptic potentials. The "secondary" response is akin to the "K-complex" or "V-potential" and is presumed to reflect extra-lemniscal activity. Our data indicates that alcohol affects the primary response as well as the later components of the somatosensory evoked cortical activity.

Blood alcohol levels were monitored throughout the experiment with Breathalyzer readings, and were maintained at fairly constant levels. Our data indicate that the amplitude of the somatosensory evoked potential is primarily influenced by the actual level of alcohol in the blood. The magnitude of the evoked potential was strikingly depressed on days when alcohol was administered, concomitant with elevated blood-alcohol levels. In addition, evoked potential amplitudes remained relatively constant throughout the testing session for all 12 runs, paralleling the blood-alcohol readings. The role of the rate of change in blood-alcohol level as a factor effecting evoked potential modifications still remains to be determined.

Great caution is suggested in comparing the results of different experiments dealing with the effects of alcohol on somatosensory evoked potentials, as the recording sites, type of recording (mono-

polar vs. bipolar), stimulating electrode sites, alcohol dosage and administration, etc. are often so dissimilar. Methodological differences very often may be the cause of divergent results, and the content of what is being investigated may not always be analogous. It is hoped that somatosensory evoked potentials will provide a tool to elucidate and differentiate the action of alcohol on the central nervous system.

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