Effect of Alcohol on the Cerebral Function Observed in Eye Movements during Tracking a Visual Target

EFFECT OF ALCOHOL ON THE CEREBRAL FUNCTION OBSERVED IN EYE MOVEMENTS DURING TRACKING A VISUAL TARGET

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Abstract

In this study we especially associate eye movements in tracking a visual target with dysfunctions caused by symptoms of intoxication in both frontal and parietal lobes. Visual targets used in this study were of apparent movements and of periodical triangular wave form on display. Two moving patterns of a visual target were provided for the experiment. One is designed so as to demand a perceptual task, and another for a cognitive task. Each subject carried out the tracking process under three conditions: of no-alcohol, of low and high concentrations. In the case of an intoxicated subject, the latency and the duration of saccade are more prolonged, the mean velocity is reduced, and the amplitude is enlarged, especially in the case of tracking a visual target with a moving pattern which requires a cognitive task. There is no difference between the two tasks on the neural network for setting up necessary parameters to start off a saccade, but the quality of eye tracking is definitely affected by high quantities of alcohol. This corroborates Terao's hypothesis that the neural network can compensate for the damage only at one area in the brain, but does not cooperate well if damage exists in multiple areas of the brain.
Key words: alcohol, visual search, cerebral function, saccade, driving

1. Introduction

Drinking is one of us human beings' most typical habits and has a strong influence on personal and social aspects of our lifestyles. Many researchers have investigated influences caused by alcohol on the human body. It is well-known that dysfunction of the brain induced by the symptoms of intoxication can lead to personal or social disaster in the form of a driving accident or a criminal act.

Some reports, indicating relations between intoxication and localization of functions in the brain, show that intoxication causes hypesthesisa in the frontal lobe, locomotors ataxia in the parietal lobe, visual disturbance in the occipital lobe and basal ganglia, and poor equilibrium in the cerebellum[1]. These dysfunctions were investigated by applying such methods as MEG, fMRI or PET, which can pick up responses caused in the brain while certain stimuli were given to the human body under symptoms of intoxication[2)~4)]. Other

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studies demonstrate that the influence of alcohol permeates throughout the brain in proportion to its quantity, as the density of alcohol affects the function of the frontal lobe with a concentration of breath alcohol (B.A.) of 0.5mg/l, the parietal lobe with a 1.0mg/l concentration, the occipital lobe and basal ganglia with 1.25mg/l, and the cerebellum with 1.5mg/l[5]. There have been many reports in which these dysfunctions were indirectly observed action of or symptoms seen in an effector of the human body under a condition of intoxication. One important aspect of effector's actions is eye movement, which is induced through some functions of the brain as a result of response to a stimulus[6,7]. It is clear that there is a relationship between the two kinds of eye movement in tracking and localization of function in the brain. For example, the most important feature of saccade "saccadic amplitude" is organized in the superior colliculus[1], "eye velocity" of smooth eye movement is regulated in the frontal lobe[8] and latencies of two kinds of eye movements are generated in the occipito-parietal association area[9].

In this study, we try to corroborate these above-mentioned dysfunctions through the eye movement behavior in tracking a visual target under symptoms of intoxication.

2. EYE MOVEMENT GENERATION AND THE INFLUENCE OF ALCOHOL IN THE BRAIN

2.1 Brain and Eye Movement

We find two kinds of eye movements, saccade and smooth eye movement, in tracking a visual target. Saccade is generated by a neural network through the frontal visual area, the LIP area, thalamus, basal ganglia, superior colliculus, cerebellum and the brain stem, and smooth eye movement through MT and MST in the cerebral cortex, nuclei Pontius, cerebellum and the brain stem[6].

Some areas in these neural networks have a specified function for creating eye movement. The frontal lobe, for example, has a switching mechanism for selecting either saccade or smooth eye movement, a generator for amplifying each eye velocity of the two kinds of eye movement[2], and a controller for managing an accommodation of all classes of voluntary eye movement[10]. The parietal lobe is crucial for responses to the most salient or behaviorally relevant visual objects[11], and for evoking saccade in catching a visual target.

2.2 Brain and Alcohol

In general, alcohol serves to cause a loss of self-restraint in the neo cortex, so that the human being has a feeling of euphoria, over-confidence, and mental flexibility. The influence of alcohol permeates with increasing amount by turns through the somatosensory cortex and the motor cortex in the parietal lobe, through the visual area in the occipital lobe and the thalamus[5].

The relationship of the concentration of breath alcohol to localization of function in the brain or to dysfunction caused by symptoms of intoxication is summarized in Table 1. Decline in the ability to make a decision, or to concentrate and loss of self-restraint are

Table 1 Brain area and dysfunction on alcohol

Concentration of B.A.	Brain Area	Dysfunction
0.50[mg/l]~	Frontal lobe	Loss of Self-Restraint Feeling of Euphoria Hypesthesia
1.00[mg/l]~	Parietal lobe	Locomotor Ataxia Language Disorder Reeling
1.25[mg/l]~	Occipital lobe Basal ganglia	Visual Disturbance Disturbance of Consciousness Vomoting
1.50[mg/l]~	Cerebellum	Difficulty in Walking Difficulty in Equilibration
1.75[mg/l]~	Pons	Coma Sweating
2.00[mg/l]~	Medulla	Inhibition of Respiration Temperature Fall Death

observed when the influence of alcohol permeates through the frontal lobe with a concentration of breath alcohol at or above 0.5mg/l. Locomotor ataxia arises when the influence of alcohol permeates through the parietal lobe with a concentration of breath alcohol at or above 1.0mg/l.

We especially associate eye movements in tracking a visual target with dysfunctions caused by symptoms of intoxication in the frontal and parietal lobes.

3. EXPERIMENTAL CONDITIONS

3.1 Visual Target and Task

The visual targets used in this study were of apparent movements and of periodical triangular wave form on display. Velocity and amplitude for each visual target were set to 20deg/sec and 20degrees in visual angle throughout the experiment. When a visual target appeared, the subject was required to pursue it with both eyes as smoothly as possible.

We provided two moving patterns of a visual target for the experiment. One moving pattern was designed to include discontinuous displacements (of 5degrees) in order to examine perceptual changes in cerebral function upon encountering sudden perceptual gaps. Another was designed to give a cognitive task (comparing two different numbers presented for 300msec at each point along the left and right sides (5degrees) from the center of the display) while the subject was tracking a visual target in order to examine cognitive changes. As a result, subjects continued to track a visual target in the direction of the larger of the two numbers.

3.2 Subjects and Drinks

A total of three healthy male undergraduate students participated in the study. The subjects' ages were 22-24 years and they weighed 62-70 Kgf. Drinks consisted of whisky (with 40% alcohol) and water. Amounts of whisky in each drink were either about 200ml to give a concentration of breath alcohol of 0.5mg/l ("low alcohol concentration"), or about

400ml to give a concentration of breath alcohol of 1.0mg/l ("high alcohol concentration"). The condition of low alcohol concentration was designed to affect the cerebral functions at frontal lobe, and that of high alcohol concentration would affect cerebral functions at both frontal and parietal lobes.

3.3 Experimental Method and Procedure

Each subject performed tasks under three conditions: no-alcohol, low alcohol and high alcohol concentrations. The same task was performed twice by each subject under conditions of low and high alcohol concentrations. Eye movements during tracking a visual target were measured by the Eye Link System, which samples at the rates of 250Hz. Each subject drank 200ml within 30minutes in consideration of the internal absorption velocity of alcohol in the body. The breath alcohol concentration was measured 30minutes from the time of drinking in the case of low alcohol concentration. After checking that the breath alcohol value was more than 0.5mg/l, the subject performed the tracking process designed with two moving patterns. Immediately after finishing these experiments for low alcohol concentration, the subject drank another 200ml within 30minutes, and the breath alcohol concentration was measured again to ensure a condition of high alcohol concentration of more than 1.0mg/l. The subject then performed the tracking process using the same procedure as before.

4. RESULTS

4.1 Moving Pattern Designed to Demand a Perceptual Task

Figure 1 shows the loci of a moving target (a broken line) and eye trajectories in tracking a visual target (a black line for smooth eye movement and a gray line for saccade). In this figure, latency, amplitude, duration and mean velocity of a saccade are defined, and a discontinuous point of a visual target is named a "saccadic point".

Figure 2 shows examples of eye trajectory in tracking a visual target with the moving pattern including discontinuous displacements under conditions of no-alcohol, low, and high alcohol concentrations, respectively. Eye trajectory under no-alcohol condition as shown in

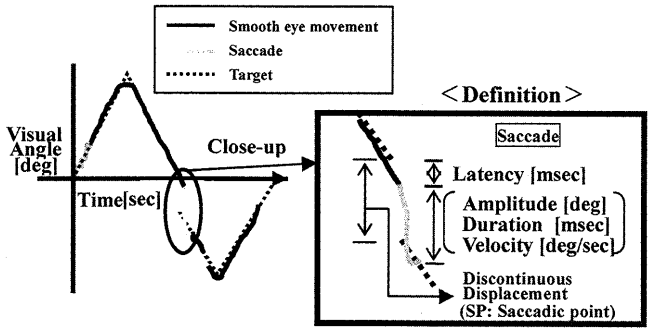

Fig. 1 Schematic loci of a visual moving target and of eye movement in tracking it

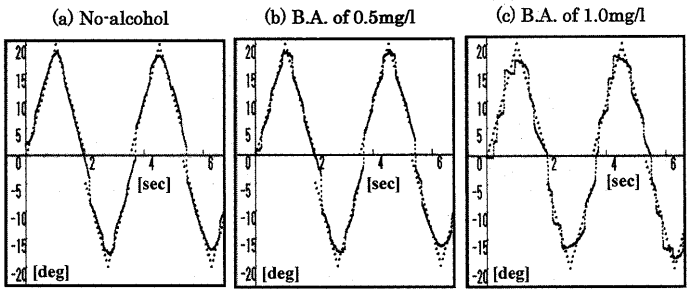

Fig. 2 Loci of moving targets including sudden perceptual gaps and eye trajectories in dual-mode tracking

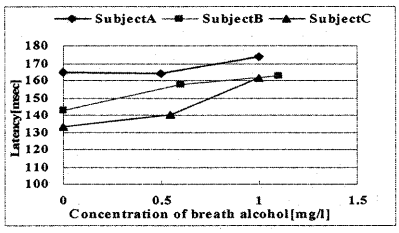

Fig. 3 Latency of saccade

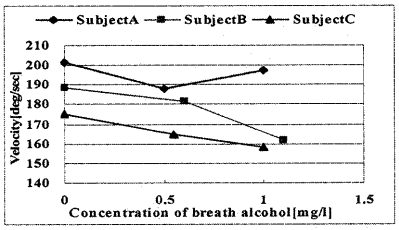

Fig. 4 Mean velocity of saccade

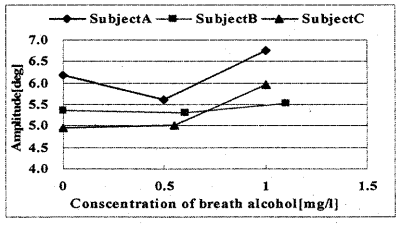

Fig. 5 Amplitude of saccade

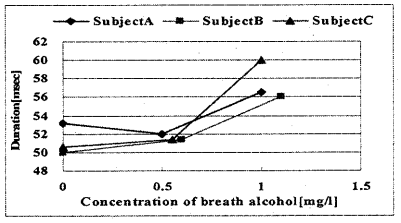

Fig. 6 Duration of saccade

Fig.2(a) consists of precisely smooth eye movements and saccades which are caused at only three points- the starting point where a target begins to move, the turning point where a target begins to move back and the saccadic point. The subject can quite easily perceive the moving target on grasping this eye trajectory. However, it becomes harder for the subject to perceive the moving target in proportion to the density of alcohol, as shown in Figs.2(b) and 2(c), where the frequencies of saccade and of insufficient smooth eye movement increase.

We assessed four features: latency (msec), amplitude (deg), duration (msec) and mean velocity (deg/sec) of saccade on eye trajectory which was induced just after the saccadic point on a locus of a moving target. These four features of saccade are summarized as a function of breath alcohol concentration in Figs.3-6. Three quantities of saccade; latency, amplitude and duration increase, but that of mean velocity decreases especially in concentration of breath alcohol 1.0mg/l.

4.2 Moving Pattern Designed to Demand a Cognitive Task

The subject's eyes are made to saccade the moment when a cognitive task is presented during tracking a visual target, since we want to know the consequences of alcohol on cognitive function in the neural network.

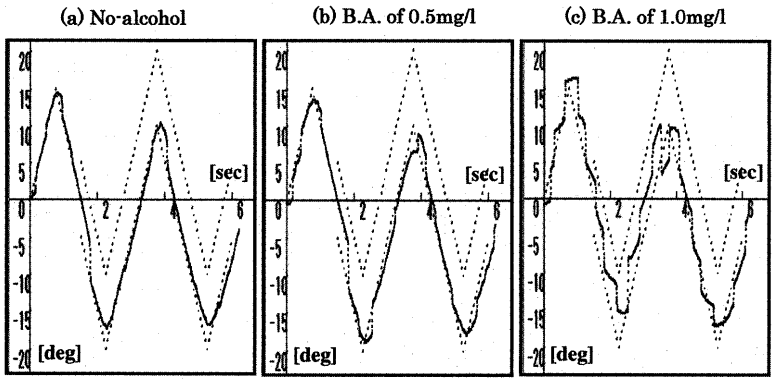

Fig. 7 Loci of moving targets given a cognitive task and eye trajectories in dual-mode tracking

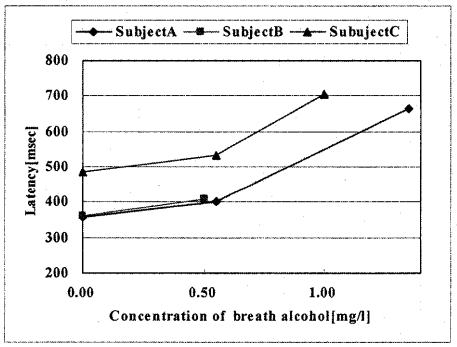

Fig. 8 Latencies of saccade when subject tracks a moving target designed to give a cognitive task

Figure 7 shows examples of eye trajectory in tracking a visual target with a moving pattern designed to demand a cognitive task under three conditions: no-alcohol, low, and high alcohol concentrations. The amplitudes of a saccade are enlarged in proportion to the concentration of breath alcohol, as shown in Figs.7 (b) and 7(c). The latencies of a saccade are exhibited graphically in Fig.8. Latency increases in proportion to the concentration of breath alcohol.

5. DISCUSSION

The experiments corroborate the results of Wegner[6]: the latency of saccade in this dual mode tracking becomes more and more prolonged and intoxicated subjects showed a more reduced mean velocity of the saccade than did no-alcohol subjects. Kato[7] reports that latency of voluntary eye movement becomes prolonged in proportion to the complexity of

the task. We too found this to be the case. The upshot is that there is no difference between the two tasks on the neural network for setting up necessary parameters to start off a saccade. As shown in Figs.2 and 7, the higher the concentration of breath alcohol, the more saccades are found on an eye trajectory, because symptoms of intoxication causes dysfunction of the switching mechanism for selecting saccade or smooth eye movement, and also the loss of self-restraint causes a decline in the function of smooth eye movement generation. Furthermore, hypesthesia causes a decrease in the mean velocity of saccade due to dysfunction in the brain's generator for amplifying eye movement[8].

Increase in amplitude, duration and latency of saccade arises from locomotors ataxia in the parietal lobe under symptoms of intoxication, as indicated by W. Becker[12] and S. Kato, et.al.[13]

However, the amplitude of saccade caused at a discontinuous displacement of 5 degrees in the visual target is closer to a condition of low alcohol concentration rather than to one of no-alcohol concentration, because the subjects are capable of focusing attention on accomplishing each tracking process under less self restraint in the frontal lobe[5].

Eye tracking is definitely affected by quantity of alcohol intake, for we can see its influence on the frontal lobe in the case of low alcohol concentration and on both the frontal and parietal lobes in the case of high alcohol concentration[5]. This corroborates Terao's work [14] which shows that the neural network can compensate for the damage only at one area in the brain, but does not cooperate well if damage exists in multiple areas.

It is understood that driver's eye movement is usually in a dual-mode when collecting visual information while driving. The temporal damage of perceptual or cognitive functions in the brain must be responsible for accidents caused by drunken drivers (see H. Moskowitz, et al.[15] and F. Andris, et al.[16]). Such results show that alcohol causes driver errors when checking a dashboard meter, reading a road signal, watching a pedestrian or an oncoming car when driving while over the legal limit of alcohol (0.15mg/l in Japan and 0.4mg/l in some foreign countries [17]).

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