THE EFFECTS OF ALCOHOL ON EYE MOVEMENTS DURING READING

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Abstract — In an experimental double-blind placebo study of 18 subjects (mean age 26.2 years), we investigated the effect of three blood alcohol concentrations (0.0%, 0.05% and 0.1%) on five visuo-motor reading parameters: (1) number of eye fixations per 100 words read; (2) the number of words read per minute; (3) the number of regressions per 100 words read; (4) the saccadic length; (5) the duration of eye fixations. The number of fixations and the duration of eye fixations increased significantly as a function of increased breath alcohol concentration. There were no significant changes in the other visuo-motor reading parameters.

INTRODUCTION

Several studies have documented that alcohol influences several perceptual functions relevant to the reading process (Jansen et al., 1985; Rohrbaugh et al., 1988). For instance, Jansen et al. (1985) and Rohrbaugh et al. (1988) found that alcohol significantly affected signal detection performance under conditions of low signal probability. Gustafson (1986) reported that alcohol might impair peripheral sensory sensitivity. Neill et al. (1990) showed that alcohol might affect processing of motion in depth. Roquelaure et al. (1995) measured visual contrast sensitivity for all spatial frequencies. The reduction was 2.40 dB below the level of the controls. Avant (1990) presented evidence indicating that alcohol influences precortical visual processing and that the influence of alcohol was greater in females than males. More direct evidence of visuo-motor effects was presented by Katoh (1988) who registered the effect of alcohol on latency and saccadic velocity of eye movements in two types of task complexity. The results showed a slowing effect of alcohol on latency ranging from 8.4 to 16.8%, corresponding to the task complexity. The impairment of the saccadic velocity ranged from 27.4 to 25.5%. Post et al. (1994) examined the effect of alcohol on compensatory eye movements and perceptual stability and found that alcohol influenced both the vestibulo-ocular reflex (VOR) and apparent concomitant motions (ACM). Bauer (1993) studied smooth pursuit (SPEM) and saccadic (SEM) eye movements comparing cocaine-dependents, alcoholics, and controls. Eye movement data indicated that alcohol-dependent subjects exhibited slower saccadic onset latencies than the other two groups. The above authors also found that pursuit movements tracking accuracy was higher among the cocaine-dependents than among the alcoholics or the controls.

Although a number of studies have attended to the influence of alcohol upon vision and perception, the influence of alcohol on eye movement parameters relating to reading has been scarcely investigated. Acknowledging on the one hand that modern occupations are heavily dependent upon reading skills and on the other hand that consumption of moderate amounts of alcohol is very common in everyday life style in most Western countries (Edwards et al., 1994), this issue is worthy of close attention.

Contrary to common-sense belief, eye movements during reading are not smooth or continuous across a text. Broadly speaking, the movement patterns consist of a series of 'jumps' and 'stops'. The movements are predominantly generated in the brain stem, but the neural output also seems to be influenced by higher levels in the brain (Kandel et al., 1991). For several years now, eye movements have been studied mainly with the help of corneal reflection technology. The patterns disclosed, often denoted saccadic patterns, are quite
complex (see, e.g., Rayner, 1978; Evans and Drasdo, 1990). A typical saccadic pattern registered with a corneal reflection system is illustrated in Fig. 1. A saccade is normally defined as a rapid eye movement to redirect the line of sight. Occasionally the eye moves backwards to refixate a word. This corrective backward movement is called a regression. Skilled readers have few and long saccades, few or no regressions and short fixations. Long fixation time, frequent fixations and regressions are characteristics of disturbed reading skills (Evans and Drasdo, 1990; Birnbaum, 1993).

The present study focuses on the effect of three blood alcohol concentrations (BACs; 0, 50 and 100 mg/dl) on five eye movement components relevant for reading, namely saccadic length, number of fixations per time unit, number of regressions, duration of fixations (i.e. the time the eye stops before the next saccade) and reading speed.

**MATERIALS AND METHODS**

**Subjects and experimental design**

After obtaining their informed consent, 18 males (mean age 26.2 years) volunteered to participate in an investigation on the effect of intake of alcohol on vision. All subjects were in good health and were moderate social drinkers. Furthermore, in order to avoid possible interactions between alcohol and visual, refractional or binocular anomalies, the subjects were selected according to three criteria: (1) they were not to wear glasses or contact lenses; (2) they should not have manifest strabismus or show distant eso- or exo-phorias >4 PD (Keystone View Ophthalmic Telebinocular); (3) binocular Snellen acuity should be 1.0 or better.

We used a within-subject design with three repeated measurements: (1) the placebo condition; (2) the 0.05% (50 mg/dl) condition; (3) the 0.1% (100 mg/dl) condition. The subjects were assigned to the treatment conditions according to an alternating procedure as follows: in the first session, the first subject received a placebo drink, the second subject received a 0.05% alcoholic drink and the third subject received a 0.1% alcoholic drink etc. In the second session the first subject received a 0.05% drink, the second subject a 0.1% drink and the third subject a placebo drink etc. This assignment procedure was repeated across all three conditions for all 18 subjects.

**Measurement of eye movements and reading parameters**

Eye movements were recorded using an infrared (IR) corneal reflection monitoring system using the OBER 2 UNO-Parallel system (Permobil Meditech AB, Timrå, Sweden). This apparatus does not monitor the position of the eyes continuously, but uses short pulses of the IR light at chosen frequencies. The intermittent sampling allows the use of short flashes of low intensity IR. The OBER-2 system measures eye movements in both eyes in a horizontal and vertical direction, digitizing the movements with a frequency of 100 Hz. Thus, several functional components relevant to the reading process may be assessed (Ygge et al., 1993).

Saccadic length (or recognition span) is operationalized as the number of words covered during one saccade. Number of fixations per time unit is calculated as the arithmetic sum of fixations per 100 words read. The number of regressions is calculated as the arithmetic sum of backward
movements (i.e. refixations) per 100 words read. Duration of fixation, i.e. the length of time the eye stops before the next saccade, is measured in real-time in milliseconds.

There were no direct sources of light or lamps into the goggles, to the side of the subjects or behind the subjects, which could induce reflections that could interfere with the presented stimuli. The subjects were seated in a comfortable chair allowing them to place their feet firmly on the ground. A head and chin rest was used to stabilize the subject’s head during the recording session.

The text was part of a short novel and was placed at the level of the subjects’ eyes in the centre of the subjects’ visual field. Each text line was 13 cm, reading time (i.e. the time allowed to read the passage) was limited to 20 s and the reading distance was 40 cm. The equipment was calibrated at the start of the experiment following the recommended guidelines (OBER-2 Manual, 1991).

**Inducing BACs**

Three BACs were induced, 0, 50 and 100 mg/dl, respectively (i.e. comparable with the US units 0 g% w/v, 0.05 g% w/v and 0.1 g% w/v, respectively. Many readers with moderate knowledge of clinical chemistry are probably more familiar with the BAC units of 0.0%, BAC 0.05% and BAC 0.1%. These units will therefore be used in the following). In order to reach BACs of 0.05% and 0.1%, we administered absolute ethanol doses of 0.65 and 1.3 ml/kg body weight using a commercially available alcoholic drink (vodka) of 40% ethanol mixed with lemon-tasting mineral water to a v/v concentration of ~10% ethanol. The mean (SD) body weight of the subjects was 75.4 (11.2) kg. The total amount consumed in each experimental session was ~1 litre. To avoid a steep rise in BACs, the pre-experiment drinking period was 1 h. An assistant (blind for type of drink) served a drink (250 ml) to the subjects at ~15 min intervals. The placebo drink consisted of the lemon-tasting mineral water with an added vodka-taste. In order to control further for olfactory stimuli, the rim of each glass was smeared with vodka just before they had the placebo drink.

**Measurements of BAC**

The BAC was estimated indirectly by measuring the end-expired breath alcohol content (BrAC) by use of the Lion Alcometer SD-2. This is a pocket model convenient for clinical and experimental research purposes, since no technical procedures for collection of whole blood specimens (normally from the antecubital vein) are required. Also, due to a number of factors affecting distribution of alcohol in the human body, venous blood samples might result in falsely low ethanol concentrations (Yi, 1991). In general, instruments for measuring ethanol in the breath had shown acceptable accuracy, linearity and reliability (Jones, 1978), although for some of these instruments (most notably the Intoxilyzer 5000) a constant analytical bias has been noted yielding slightly lower BrAC values than BAC values (Jones et al., 1992). For experimental purposes, however, linearity and reliability are most important. For the SD-2, these values are very good [linearity = 0.998; reliability = repeated measurements showed <0.03% measurement errors compared to a reference signal (Swedish Testing Institute, 1984)]. BrAC was measured 15 min after the last drink when the subjects were at the beginning of the flattening phase of the blood alcohol curve. The Alcometer was calibrated at the beginning of each day.

**Ethics**

The study was approved by the Regional Ethics Committee for Medical Sciences of Health Region II in Norway.

**RESULTS**

The distributions of the eye movement factors were rather skewed. We therefore employed the nonparametric Friedman test for k-related samples (Siegel and Castellan, 1988). Figure 2 shows the number of eye fixations (i.e. saccadic pauses) and intake of alcohol for the three experimental conditions. The subjects made significantly more saccadic pauses per 100 words read in the alcohol conditions, compared to the placebo condition ($\chi^2 = 5.80, df = 2, P < 0.05$).

The effects of alcohol consumption at the >2 dose levels were examined for reading speed and regressions and also saccadic length. There was a reduction of reading speed across the conditions, but the individual variations were so considerable that the mean differences were not significant.
Fig. 2. The figure shows the number of fixations for the placebo condition (light grey bar), the 0.05% BrAC condition (dark grey bar) and the 0.1% BrAC condition (black bar). Fixations are calculated as the number of fixations (i.e. saccadic pauses) per 100 words read. Error bars show the standard deviations; n = 18.

Fig. 3. The figure shows the duration of fixation in milliseconds for the placebo condition (light grey bar), the BrAC 0.05% condition (dark grey bar), and the BrAC 0.1% condition (black bar). Error bars show the standard deviations; n = 18.

There were also no significant differences in number of regressions or saccadic length between the two alcohol conditions and the placebo control.

Figure 3 presents the results of duration of fixations (i.e. saccadic pauses). The length of the saccadic pauses increased with increasing BAC levels ($\chi^2 = 15.9$, df = 2, $P < 0.001$).

**DISCUSSION**

The subjects in this study were skilled readers. The average number of words read per minute (342 words in the placebo condition) indicates a reading speed that is somewhat higher than average for Norwegian high schools (Hunstad and Hagtvedt, 1984). In all three conditions, the subjects exhibited few regressions, another indicator of well-developed reading skills.

Moderate alcohol dosages (i.e. BAC 0.05% and 0.1%) clearly have some detrimental effects upon the functional integrity of the oculomotor system, effects which also influence the reading process. The number of fixations (saccadic pauses) (Fig. 2) and duration of fixation (Fig. 3) increased as a function of alcohol dosages, while regressions and number of words read were not particularly affected. Duration of fixation factor reflects the time the eye stops between each saccadic 'jump'. It is during this time interval that the eye 'sees' written text; the eye is perceptually blind during each saccadic movement (Sekuler and Blake, 1985). Normally, reading speed is related to duration of fixation (and several other parameters) and we would therefore expect a slowing of reading speed with increased fixation time. Although there was a tendency towards fewer words read with increased BAC, this reduction was not significant at the 0.05 level, probably due to the low number of subjects and the large individual variations in reading speed. In addition, a factor related to the text should also explain the relative insensitivity of reading speed. The reading task had the same basic content for all three sessions and the text was also quite easy in terms of syntax and semantics. Some transfer of learning might therefore have occurred across the sessions, probably counterbalancing some of the effects of alcohol. For new texts or more complex reading materials, the effect of alcohol could perhaps be more marked.

Slowing of fixation time and increased number of saccadic pauses indicate that two basic components of the reading process are disturbed, even by moderate doses of alcohol. The practical implications of these effects are difficult to pinpoint in detail, but disturbances at the sensory level, such as eye movements, might also disturb higher cognitive processes such as the encoding and storage of visual information (Stephens et al., 1992). However, this issue should be further investigated.

The increased duration of fixation as a function of elevated BAC levels is probably the result of the depressant effects of alcohol on the brain stem. This effect has been well documented in several studies (Hogan and Gilmartin, 1985; Yi, 1991; Tabakoff and Hoffmann, 1992). A number of
nuclear complexes involved in perceptual and oculomotor control reside in this integrating part of the central nervous system and adjacent areas such as the rostral vestibular complex, the pontine and parvo-cellular parts of the reticular formation, the prepositus hypoglossi nucleus, the nucleus intertrigeminus, the nucleus supratrigeminus and the subnucleus caudalis. From the nuclei, there are several possible interactions with other reticular areas, the extra-pyramidal nuclei, the cerebellum, the medulla and several parts of the thalamus (Ruggiero et al., 1982; Lie and Watten, 1987). The higher cortical control over saccadic movements seems to be exerted through the superior colliculus. For instance, recordings have shown that neurons in the intermediate layer of superior colliculus fire before contralateral saccades of specific size and direction. This output drive seems to excite the long-lead burst cells of the paramedian pontine reticular formation. Furthermore, these cells have rather large movement fields (Kandel et al., 1991, p. 674). Thus, the increased duration of fixation might be explained by the alcohol-induced slowing of the processing time of the saccadic eye movements generated by the reticular formation in the brain stem. It is also documented in several investigations that the saccadic generators in the brain stem are influenced by the cerebral cortex, most notably the frontal eye field and the posterior parietal cortex. The frontal eye field probably exerts control over saccades through the superior colliculus in two ways: (1) movement neurons project directly to the intermediate layer of the superior colliculus and excite the motor output neurons; (2) projections to the caudate nucleus excite the neurons which inhibit the substantia nigra, which again releases the inhibition of the superior colliculus. Thus, alcohol might influence the oculomotor control centres in the brain stem through several paths.

REFERENCES


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