Increased Distractibility by Task-Irrelevant Sound Changes in Abstinent Alcoholics

Jyrki Ahveninen, Iiro P. Jääskeläinen, Eero Pekkonen, Anja Hallberg, Marja Hietanen, Risto Näätänen, Erich Schröger, and Pekka Sillanaukke

Background: Chronic alcoholism is accompanied by “frontal” neuropsychological deficits that include an inability to maintain focus of attention. This might be associated with pronounced involuntary attention shifting to task-irrelevant stimulus changes and, thereafter, an impaired reorienting to the relevant task. The neural abnormalities that underlie such deficits in alcoholics were explored with event-related potential (ERP) components that disclosed different phases of detection and orienting to stimulus changes.

Methods: Twenty consecutive abstinent male alcoholics (DSM-IV) and 20 age-matched male controls (healthy social drinkers) were instructed to discriminate equiprobable 100 and 200 msec tones in a reaction-time task (RT) and to ignore occasional, either slight (7%) or wide (70%), frequency changes (hypothesized to increase RT) during an ERP measurement.

Results: In the alcoholics, we found pronounced distractibility, evidenced by a RT lag (p < 0.01) caused by deviants, that correlated (Spearman $\rho = 0.5$) with a significantly enhanced (p < 0.01) amplitude of mismatch negativity (MMN) to deviants. Significantly increased RT lag for trials subsequent to deviants (slight $p < 0.001$, wide $p < 0.05$) in the alcoholics suggested impaired reorienting to the relevant task. The MMN enhancement also predicted poorer hit rates in the alcoholics (Spearman $p = 0.6–0.7$). Both the MMN enhancement and pronounced distractibility correlated (Spearman $\rho = 0.4$) with an early onset of alcoholism.

Conclusions: Attentional deficits in the abstinent alcoholics were indicated by the increased distractibility by irrelevant sound changes. The MMN enhancement suggested that this reflects impaired neural inhibition of involuntary attention shifting, being most pronounced in early-onset alcoholics.

Key Words: Alcoholism, EEG, Auditory Event-Related Potentials, Involuntary Attention Shifting, Mismatch Negativity (MMN).
Impaired control of involuntary attention shifting could cause pronounced distractibility and inability to modify responses to external stimuli, which often are evident in alcoholics on clinical observation.

Deviations in tone frequency, occurring in an unattended series of repetitive “standard” tones, impair both speed and accuracy of reaction time (RT) task performance (Jääskeläinen et al., 1999; Schröger and Wolff, 1998a,b). This involuntary attention shifting to task-irrelevant deviants can be indexed by mismatch negativity (MMN), a preattentive ERP component that discloses different phases of detection and orienting to a stimulus change (Alho, 1995; Näätänen, 1992; Näätänen et al., 1978; Tiitinen et al., 1994). The supratemporal MMN subcomponent reflects initial detection of difference between the deviant and an auditory-cortical representation of standard tone (Näätänen, 1992; Näätänen et al., 1978), which further triggers involuntary attention shifting and elicits the frontal MMN subcomponent. Previous MMN studies on alcoholism, which generally show no impairment in automatic change-detection, have indicated that MMN might be even slightly enhanced or accelerated during the first weeks of abstinence (Ahveninen et al., 1999; Pekkonen et al., 1998). Whether this phenomenon is associated with pronounced involuntary attention shifting has not yet been studied. Therefore, we studied possible abnormalities in involuntary attention shifting and their cerebral basis by using a recently developed combined ERP and behavioral RT paradigm in abstinent chronic alcoholics (Jääskeläinen et al., 1999; Schröger and Wolff, 1998b). Results were correlated with subjects’ demographic variables, such as onset age of alcoholism.

MATERIALS AND METHODS

Twenty abstinent male alcoholic inpatients (19–55 years old, mean 40 years) who met DSM-IV criteria for alcohol dependence were recruited consecutively from a routine treatment program. Twenty education-matched male controls (21–59 years old, mean 37 years) were healthy social drinkers with self-reported ethanol consumption of 97 g/week (12–216 g) who abstained from alcohol and other drugs at least 48 hr before the measurement. The patients’ exclusion criteria were acute withdrawal symptoms (Clinical Institute Withdrawal Assessment for Alcohol; Sullivan et al., 1989), hearing deficits, heart diseases, liver diseases, diabetes mellitus, current drug abuse, history of dependence on a drug other than alcohol, head trauma, Korsakoff’s syndrome, or the presence of psychiatric and neurological diseases unrelated to alcoholism. The mean demographic variables of the alcoholics were as follows: abstinence duration 20 days (7–45 days), self-reported onset of drinking 29 years (14–50 years), years of excessive drinking 11 years (1–35 years), and weekly ethanol consumption 1213 g (336–2520 g). The Alcohol Use Disorders Identification Test (Seppä et al., 1995) was used. A written informed consent was obtained after the procedures had been explained fully to the patients. The Ethics Committee of the A-Clinic Foundation, Helsinki, Finland, approved the study. Six alcoholics (three with early and three with late onset) used antidepressants or other central nervous system medication but showed similar behavioral and ERP responses as the 14 unmedicated alcoholics.

In the forced-choice RT task, the subjects were presented randomly with binaural pure tones (5 msec rise and fall times) that equiprobably

Fig. 1. (a) The grand-average event-related potentials (ERP) to the slightly deviant stimuli (750 Hz) superimposed on that of the standard stimuli (700 Hz) in both groups. The 32 electrode positions are illustrated schematically according to the extended 10–20 system. (b) The deviant minus standard-stimulus difference waves of controls superimposed on those of alcoholics. (c) The grand-average scalp distribution of mismatch negativity (MMN; the distance-weighted least-squares method; McLain, 1974) for slight deviants during the earlier (140–190 msec) and later (190–240 msec) time windows in the controls and alcoholics.
aged standard-tone ERP, MMN, P3a, and reorienting negativity (RON; Schröger and Wolff, 1998a) were analyzed from difference waveforms for the slight and wide deviants (ERP for slight deviants minus standard ERP, ERP for wide deviants minus standard ERP, respectively). The MMN amplitude for deviants was determined as an average response during two consecutive 50 msec periods. For the slight deviants, 140 to 190 msec (early) and 190 to 240 msec (later) poststimulus periods were used, and because the MMN latency decreases as the magnitude of stimulus deviance increases (Tiitinen et al., 1994), the respective periods were 80 to 129 msec (earlier) and 130 to 179 msec (later) for the wide deviants. In addition to the peak amplitudes, average P3a amplitudes at 250 to 399 msec and average RON amplitudes at 480 to 549 msec (Schröger and Wolff, 1998a) were analyzed from difference waveforms for the MMN. The MMN peak latency was not significantly delayed in the alcoholics. Other differences were not observed in P3a or in RON for slight deviants, or in any ERP components elicited by the wide deviants, or in the P1, N1, and P2 to standards (Fig. 1a).

RESULTS

RT lag caused by task-irrelevant frequency changes was significantly pronounced in the alcoholics (Fig. 2, Table 1), as shown by significant group main effects in ANOVAs for slight \( F(1,38) = 11.3, p < 0.01 \) and wide deviants \( F(1,38) = 4.17, p < 0.05 \). A priori contrasts indicated that RT lag by slight deviants \( F(1,38) = 6.81, p < 0.05 \), RT lag in standards after slight deviants \( F(1,38) = 13.2, p < 0.001 \), and RT lag in standards after wide deviants \( F(1,38) = 5.57, p < 0.05 \) were significantly larger in the alcoholics than controls (Fig. 2, Table 1). No differences emerged in other behavioral variables.

Table 1. Mean ± SD of Reaction Time (RT) and Hit Rate (HR) Variables in the Controls and Alcoholics

<table>
<thead>
<tr>
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<th>Controls (n = 20)</th>
<th>Alaskaics (n = 20)</th>
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<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
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<td>RT (msec)</td>
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<td>Standards</td>
<td>501</td>
<td>63.3</td>
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<td>RT lag (msec)</td>
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<td>to slight deviants</td>
<td>20</td>
<td>21.2</td>
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<tr>
<td>Wide deviants</td>
<td>39</td>
<td>39.3</td>
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<td>RT lag (msec) to standards</td>
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<tr>
<td>After slight deviants</td>
<td>7</td>
<td>17.5</td>
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<tr>
<td>After wide deviants</td>
<td>44</td>
<td>35.4</td>
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<tr>
<td>HR (%) Standards</td>
<td>80</td>
<td>23.5</td>
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<tr>
<td>Slight deviants</td>
<td>79</td>
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<tr>
<td>Wide deviants</td>
<td>76</td>
<td>23.2</td>
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<tr>
<td>HR (%) to standards</td>
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<tr>
<td>After slight deviants</td>
<td>80</td>
<td>23.8</td>
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<td>After wide deviants</td>
<td>76</td>
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* \( p < 0.05 \); *** \( p < 0.001 \).

The behavioral distractibility caused by the involuntary attention shifting to task-irrelevant frequency change, indicated by the reaction time (RT) lag in deviants and first standards after deviants. 

\[ F(1,38) = 8.12, p < 0.01 \] for the MMN to slight deviants. The amplitude of 190 to 240 msec MMN to the slight deviants was significantly larger (alcoholics, mean 0.50 \( \mu \)V; controls, mean 0.06 \( \mu \)V) in the alcoholics than controls \( F(1,38) = 6.95, p < 0.05 \), but the earlier MMN amplitudes (140–190 msec) indicated no group differences (Fig. 1b and c). The MMN peak latency was not significantly delayed in the alcoholics. Other differences were not observed in P3a or in RON for slight deviants, or in any ERP components elicited by the wide deviants, or in the P1, N1, and P2 to standards (Fig. 1a).

In the alcoholics, significant correlation was observed between MMN enhancement and behavioral distractibility. The increased later MMN to slight deviants correlated significantly with increased RT lag (Spearman \( \rho = -0.50, p < 0.05 \)) and poorer HR (\( \rho = 0.67, p < 0.01 \)) caused by slight deviants, and with poorer HR to standards after slight deviants (\( \rho = 0.61, p < 0.01 \)). In the alcoholics, later MMN and poor HR for wide deviants also correlated significantly (\( \rho = 0.44, p < 0.05 \)). No such correlations emerged in the controls, but generally the trend was similar, for instance, between the RT lag in standards after wide deviants and earlier MMN for wide deviants (\( \rho = -0.43, p = 0.06 \)). However, the RT to standard stimuli correlated with the age of the control subjects (\( \rho = 0.65, p < 0.01 \)), but no such correlation was observed in the alcoholics. In the alcoholics, the self-reported onset age of alcoholism correlated significantly (\( \rho = 0.45, p < 0.05 \)) with amplitude of the later MMN to slight deviants. There was a trend toward correlation between RT lag in slight deviants and onset age (\( \rho = -0.4 \)) and between the number of previous withdrawal
treatments and RT lag in slight deviants ($\rho = 0.43$, $p < 0.06$).

**DISCUSSION**

Pronounced involuntary attention shifting to task-irrelevant frequency changes in the alcoholics, which interfered with their duration-discrimination performance, was shown by the significantly pronounced RT lag in frequency deviants and in standards presented immediately after them. The abnormality in the neural processes that underlie involuntary detection and orienting to stimulus changes (Näätänen, 1992; Schröger and Wolff, 1998a) was shown further by the augmented later phase of the MMN to slight deviants in the alcoholics. The association between the behavioral dysfunction and electrophysiological changes in the alcoholics was shown by the significant correlation that emerged between the MMN augmentation and pronounced RT lag and poorer HR to slight deviants, and between the MMN augmentation and poorer HR to the first standards after deviants.

Detection of an unattended sound change is believed to elicit the supratemporal MMN subcomponent, and this process presumably triggers involuntary attention-shifting that is reflected by the frontal MMN subcomponent (Alho, 1995; Näätänen, 1992). The frontal MMN sources recently were shown to be activated a few milliseconds later than the temporal generators (Rinne et al., 2000). The later phase of MMN that was enhanced in alcoholics thus might reflect predominantly frontal MMN sources, notably also shown to be particularly sensitive to acute alcohol effects (Jääskeläinen et al., 1996). Together, the present results support studies that showed attenuated P3b during Go/No-Go trials in alcoholics, interpreted to reflect impaired inhibition of task-irrelevant processing (Cohen et al., 1997). Analogically, the enhancement of the later phase of MMN in alcoholics might reflect an inability to inhibit the frontal-temporal neural network involuntarily activated by the frequency changes. The loss of this task-relevant inhibition could explain the pronounced distractibility by task-irrelevant sound changes in alcoholics. The increased RT lag for trials that succeeded frequency deviants, in turn, suggests an impaired reorientation to the relevant task in alcoholics.

The significant correlation between later MMN to slight deviants and self-reported onset age of alcoholism suggests that the abnormalities in involuntary attention may be most pronounced in the alcoholics with early onset of dependence. Many of the alcoholics had begun pathological drinking in their teens, which might have interfered with their frontal lobe maturation, known to occur in early adulthood. Yet, a greater share of neurotoxic changes in these alcoholics is probably produced by ethanol consumption after the maturation of their frontal lobes. Precipitating trait factors, proposed to be associated with an impulsive and aggressive subtype of alcoholism with an early onset of drinking (Cloninger et al., 1988; Mann et al., 1998), also might have affected the present results. At the same time, a number of cerebral problems other than alcoholism might impair the control of involuntary attention shifting. Hence, more detailed studies, with larger subject samples, are needed on the deficits in involuntary attention and alcoholism.

The present results support earlier findings of MMN enhancement in abstinent alcoholics (Ahveninen et al., 1999; Pekkonen et al., 1998). Despite the lack of correlation between the abstinence duration and pronounced involuntary attention shifting, the present result also could have been affected by the postwithdrawal central nervous system hyperexcitability (Buck et al., 1991). This phenomenon is caused partially by increased excitability mediated by N-methyl-D-aspartate receptor channels (Buck and Harris, 1991; Tsai and Coyle, 1998) that also are proposed to be crucial for MMN generation (Javitt et al., 1996).

In sum, the attentional deficits in alcoholism were demonstrated by reduced ability to inhibit involuntary attention shifting to task-irrelevant sound changes and impaired re-orienting to the relevant task after distraction. The neural abnormality that underlies this behavioral distractibility in alcoholics, which correlates with early onset of excessive drinking, was indicated by the enhancement of later phase of MMN, which presumably reflected frontal MMN subcomponent. These findings might provide new insights into the origins of cognitive deficits and psychopathology in alcoholism.

**ACKNOWLEDGMENT**

We thank T. Rinne for technical support.

**REFERENCES**


