Acute Effects of Alcohol on Auditory Thresholds and Distortion Product Otoacoustic Emissions in Humans

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INTRODUCTION

Acute alcohol (ethanol) challenge is known to induce various cognitive disturbances. Alcohol can also affect hearing by acting on the human central nervous system. Significant reductions in the amplitude of auditory cortical potentials have been reported after alcohol ingestion (1). Subcortical alterations of audiobility under the influence of alcohol in humans have also been studied. Several investigators showed that acute and chronic ethanol ingestion could alter auditory brainstem potentials and middle latency responses (2, 3). Studies of the effects of alcohol on human auditory thresholds yielded varying results. Murata et al. (4) reported that drinking even very small amounts of alcohol induces a temporary reduction in auditory threshold. It was also reported that moderate alcohol consumption (> 140 g/week) could protect against hearing loss in older adults (5).

A few animal studies have been carried out to investigate the effects of acute and chronic alcohol consumption on peripheral and central auditory systems. In an animal model of fetal alcohol syndrome, alcohol-exposed animals were found to have central auditory processing disorders characterized by prolonged transmission of neural potentials along the brainstem portion of the auditory pathway. Other animals also had peripheral auditory disorder in the form of congenital sensorineural hearing loss (6). In 1981, Morizono and Sikora (7) reported that topical application of ethanol induced a reduction in all bioelectric functions of the inner ear, including cochlear microphonics, endocochlear potentials and whole nerve action potentials, in the guinea pig. However, in another experiment, exposure to ethanol alone was found to have no effects on auditory sensitivity and did not cause any toxicity to outer hair cells (OHCs) (8). These conflicting results may be due to differences in the dosage, method or duration of alcohol exposure. In this study, we employed distortion product otoacoustic emissions (DPOAEs) as a tool for investigating the acute effects of alcohol ingestion on the function of human OHCs.

MATERIAL AND METHODS

Eight young adults (3 males, 5 females; mean age 25.6 years; range 22–29 years) volunteered to participate in this study. None of them had any history of ear disease or alcohol abuse. All subjects initially underwent pure-tone audiometry (tested frequency range 250–8000 Hz) and DPOAE recordings. They were then asked to drink various amounts of whisky (40% alcohol) based on their weight (3 ml/kg) together with their breakfast over a 30-min period. After ingestion of alcohol, all subjects reached the stage of clinical intoxication, as distinguished by slight slurring of speech, dizziness and slight ataxia. Blood alcohol levels were estimated based on a recent study by the Police Department of Taiwan (9). According to that study, the blood alcohol concentration increased quickly within 45 min after alcohol ingestion and declined greatly after 120 min. Fig. 1. Pure-tone thresholds (PTs) and DPOAEs were recorded serially at 30 min and 1, 2 and 3 h after alcohol consumption.

DPOAEs were recorded using a GSI 60 DPOAE system (GSI Inc., USA). Emissions were elicited by means of two continuous, pure primary tones at frequencies f1 and f2 (f2/f1 was fixed at 1:2) generated by two separate transducers, which also contained a

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Effects of alcohol on DPOAEs

Changes in DPOAE amplitudes at different tested frequencies in all subjects before and after alcohol ingestion are shown in Table I and the trends are depicted in Fig. 2. At frequencies of < 4375 Hz, there were no obvious amplitude changes in all subjects during the test period. At frequencies of > 5500 Hz, a tendency for the DPOAE amplitude to decrease was observed at 30 min and 1 h after alcohol ingestion. At 5500 Hz, the mean change in amplitude was $-2.3 \pm 2.43 \text{ dB}$ at 30 min (paired t-test: $p < 0.05$) and $-2.4 \pm 1.85 \text{ dB}$ at 1 h (paired t-test: $p < 0.05$). At 6562 Hz, the mean change in amplitude was $-3.5 \pm 1.60 \text{ dB}$ at 30 min (paired t-test: $p < 0.05$) and $3.0 \pm 2.00 \text{ dB}$ at 1 h (paired t-test: $p < 0.05$). However, the DPOAE amplitude returned to the normal baseline at 2 and 3 h after ingestion. There was no significant change in noise level during the entire course of DPOAE measurements.

The DPOAE amplitudes varied from person to person even when no alcohol was consumed. The mean DPOAE amplitudes at various times before and after alcohol intake did not differ significantly at each frequency (Table II; one-way ANOVA: $p > 0.05$). This clearly shows the individual variations in DPOAE amplitudes.

DISCUSSION

A variety of factors, including amounts of food and alcohol consumed, speed of intake and individual absorption and metabolic rates, have been found to affect blood alcohol concentration in different individuals. This may partially explain the large individual

### Table I. Changes in DPOAE amplitude after alcohol intake

<table>
<thead>
<tr>
<th>Geometric mean of $f_1$ and $f_2$ (Hz)</th>
<th>Mean (±SD) change in DPOAE amplitude (dB)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>30 min</td>
</tr>
<tr>
<td>6562</td>
<td>$-3.5 \pm 1.60^*$</td>
</tr>
<tr>
<td>5500</td>
<td>$-2.3 \pm 2.43^*$</td>
</tr>
<tr>
<td>4575</td>
<td>$-0.9 \pm 3.00$</td>
</tr>
<tr>
<td>3500</td>
<td>$-1.1 \pm 2.03$</td>
</tr>
<tr>
<td>2750</td>
<td>$-0.5 \pm 1.60$</td>
</tr>
<tr>
<td>2187</td>
<td>$-1.1 \pm 1.73$</td>
</tr>
<tr>
<td>1687</td>
<td>$0.8 \pm 1.04$</td>
</tr>
<tr>
<td>1375</td>
<td>$-0.1 \pm 1.25$</td>
</tr>
<tr>
<td>1062</td>
<td>$0.4 \pm 1.60$</td>
</tr>
<tr>
<td>812</td>
<td>$-0.5 \pm 1.93$</td>
</tr>
<tr>
<td>687</td>
<td>$-0.5 \pm 1.77$</td>
</tr>
</tbody>
</table>

$^* p < 0.05$ (paired t-test).
Fig. 2. Changes in DPOAE amplitude at various times after alcohol ingestion at each geometric mean frequency.

differences in DPOAE amplitude changes after alcohol consumption. However, our results demonstrate a significant decrease in the DPOAE amplitudes at high frequencies after moderate alcohol consumption (1.2 g/kg). The time course of the changes in DPOAEs closely matches the changes in blood alcohol levels of Taiwanese subjects described in the report of Tsai (9). This strongly suggests that the changes in DPOAEs are caused by alcohol and are concentration-dependent. In our study there was no change in the auditory threshold, which is in accordance with the results obtained by Loquet et al. (8). Therefore, we have demonstrated that changes in DPOAEs precede changes in the auditory threshold. In other words, DPOAE measurements are more sensitive for detecting early and minor changes in the OHCs than conventional audiometry.

The reductions in DPOAE amplitudes at higher frequencies reflect impairment in OHC functions in the basal turn of the cochlea. The exact mechanisms by which alcohol affects the OHCs remain unknown. However, there are several possibilities. First, the
alcohol itself may be ototoxic to the OHCs. In general, the OHCs in the basal turn are most vulnerable to ototoxic injuries. Morizono and Sikora (7) reported ototoxicity after local application of alcohol to the inner ear of guinea pigs. In their experiment, a reduction in cochlear microphonics was observed, indicating that the OHCs were damaged. A disturbance in the endocochlear environment by alcohol and its metabolites may also result in abnormal outer cell motility. Second, alcohol may influence neurotransmission in the inner ear. It has been suggested that alcohol suppresses the central nervous system by inhibiting excitatory transmission via N-methyl-D-aspartate receptors and enhancing inhibitory transmission via γ-aminobutyric acid subtype A (GABA-A) receptors (11). A number of studies have confirmed the presence of cholinergic and GABAergic efferents on OHCs in animals (12) and so alcohol may suppress the OHCs via these efferent pathways. Third, middle ear impedance or muscle tonic activity after alcohol ingestion may explain the decreased DPOAE amplitudes.

In summary, we have demonstrated that consumption of moderate amounts of alcohol induces reductions in DPOAE amplitudes at high frequencies in humans. These amplitude changes are completely reversible. This suggests that alcohol not only affects hearing via the central nervous system, but also influences the function of OHCs. However, the effects of chronic alcohol consumption on the central and peripheral auditory systems are still unclear and require further investigation.

REFERENCES


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