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The effect of alcohol on cervical and ocular vestibular evoked myogenic potentials in healthy volunteers

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HIGHLIGHTS

- Consumption of alcohol up to a maximum BrAC of 1.5‰ (mean 0.97‰) had a selective dampening effect on oVEMP amplitude, while there was no effect of alcohol on oVEMP latency or cVEMP amplitude or latency.
- Optokinetic stimulation also reduced oVEMP amplitude by decreasing the mean level of gaze and inferior oblique muscle activity throughout the recording.
- cVEMPs and oVEMPs can be reliably recorded in subjects who are under the influence of alcohol or have nystagmus, providing that they can cooperate and achieve a reasonable mean level of up-gaze during the recording.

ABSTRACT

Objective: We investigated the effect of alcohol on the cervical and ocular vestibular evoked myogenic potentials (cVEMPs and oVEMPs). As alcohol produces gaze-evoked nystagmus (GEN), we also tested the effect of nystagmus independent of alcohol by recording oVEMPs during optokinetic stimulation (OKS).
Methods: The effect of alcohol was tested in 14 subjects over multiple rounds of alcohol consumption up to a maximum breath alcohol concentration (BrAC) of 1.5‰ (mean 0.97‰). The effect of OKS was tested in 11 subjects at 5, 10 and 15 deg/sec.
Results: oVEMP amplitude decreased from baseline to the highest BrAC level by 27% (range 5–50%, $P < 0.001$), but there was no significant effect on oVEMP latency or cVEMP amplitude or latency. There was a significant negative effect of OKS on oVEMP amplitude (16%, $P = 0.006$).
Conclusions: We found a selective effect of alcohol on oVEMP amplitude, but no effect on the cVEMP. Vertical nystagmus elicited by OKS reduced oVEMP amplitude.
Significance: Alcohol selectively affects oVEMP amplitude. Despite the effects of alcohol and nystagmus, both reflexes were reliably recorded in all subjects and conditions. An absent response in a patient affected by alcohol or nystagmus indicates a vestibular deficit.

1. Introduction

Alcohol has been shown to have significant and widespread effects on the central nervous system. As acute alcohol intoxication commonly produces vertigo and imbalance, the effect of alcohol on vestibular function has been the focus of many studies. For example, research has demonstrated impairment of standing balance in healthy human volunteers after consumption of alcohol (e.g. Kubo et al., 1989; Modig et al., 2012a,b; Savolainen et al., 1980). The greatest deficits typically occur on tasks thought to rely on vestibular input, i.e. in which visual and proprioceptive cues are absent or misleading, such as standing with eyes closed on an unstable surface (Goebel et al., 1995; Ledin and Ödqvist, 1991; Tianwu et al., 1995; Woollacott, 1983). The clearest evidence of a direct effect of alcohol on the peripheral vestibular organs is the occurrence of positional alcohol nystagmus (PAN), in which alcohol

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is thought to change the specific gravity of the cupula, causing the semicircular canals to become sensitive to gravity and evoking nystagmus when the head is tilted laterally (Aschan and Bergstedt, 1975; Fetter et al., 1999).

To investigate whether alcohol affects the detection or transmission of vestibular sensory information, studies have measured its effect on vestibular reflexes, predominantly the vestibulo-ocular reflex (VOR). Alcohol has been shown to significantly diminish the nystagmus evoked by both caloric irrigation and horizontal angular rotation (Berthoz et al., 1977; Bochenek and Ormerod, 1962; Chiang and Young, 2007; Post et al., 1994; Tianwu et al., 1995). Similarly, alcohol decreases dynamic visual acuity during vertical translations, though the gain of the translational VOR does not appear to be affected (Schmäl et al., 2000, 2003). Studies of ocular counterrolling have shown a small decrease in torsion after alcohol consumption (Diamond and Markham, 2008; Markham and Diamond, 2006). In contrast, there does not appear to be an effect of alcohol on the perception of subjective visual vertical (SVV) either during standard test administration (Zingler et al., 2003) or during eccentric rotation (Lindgren et al., 1998), though there tends to be an alcohol-related increase in visual field dependence on the rod and frame test (Hafstrom et al., 2007).

While these studies point to significant effects of alcohol on the vestibular system, it is not clear whether the effects occur in the peripheral vestibular organs or along central vestibular pathways, such as in the brainstem or cerebellum. Alcohol has well-documented, detrimental effects on the central systems involved in vestibular and oculomotor function. With higher doses of alcohol saccades have slower velocity and longer latency, while smooth pursuit has decreased gain and becomes increasingly saccadic (Baloh et al., 1979; Barnes, 1984; Bittencourt et al., 1980; Fransson et al., 2010; Holdstock and de Wit, 1999; Wilkinson et al., 1974). The gain and slow phase velocity of optokinetic nystagmus also tends to be decreased with alcohol (Baloh et al., 1979; Tianwu et al., 1995) and the ability to suppress the VOR during head motion is diminished (Barnes, 1984; Harder and Reker, 1995). One of the best-documented oculomotor effects of alcohol consumption is gaze-evoked nystagmus (GEN), resulting from a deficit in gaze-holding and the neural integrator (e.g. Booker, 2001; Goding and Dobie, 1986).

It is also not clear whether there is a difference in the sensitivity of the semicircular canals or otolith organs to alcohol. On tests specific to the otoliths, such as ocular counterrolling, SVV and the translational VOR, alcohol effects have typically been small or non-significant. We therefore wished to investigate the effect of alcohol on two otolith-dependent vestibular reflexes, the cervical and ocular vestibular evoked myogenic potentials (cVEMPs and oVEMPs). VEMPs are short-latency muscle reflexes recorded from the neck and extraocular muscles in response to vestibular stimulation with brief bursts of sound or vibration (see Rosengren et al., 2010 for review). The cVEMP is recorded with surface electrodes from the sternocleidomastoid (SCM) muscle ipsilateral to the stimulated ear and consists of a biphasic positive-negative waveform with peak latencies at approximately 13 and 23 ms (i.e. p13-n23). In contrast, the oVEMP is recorded predominantly from the inferior oblique extraocular muscle contralateral to the stimulated ear from electrodes placed beneath the eyes. It consists of a biphasic negative-positive peak with latencies of approx. 10 and 15 ms (n10-p15). Recordings from single motor units in these muscles have shown that the cVEMP is produced by a short-latency inhibition of the SCM and the oVEMP by an excitation of the inferior oblique muscle (Colebatch and Rothwell, 2004; Weber et al., 2012). As sound and vibration have been shown to preferentially activate the otoliths, with the semicircular canals activated to a lesser extent (Curthoys et al., 2006; Murofushi and Curthoys, 1997; Zhu et al., 2011), VEMPs are thought to reflect the integrity of the otolith organs. The particular otolith organ responsible for each type of VEMP evoked by different stimuli is still controversial (e.g. Papathanasiou, 2012). Studies in patients with differential dysfunction of one vestibular nerve bundle suggest that the air-conducted sound-evoked cVEMP is a test mainly of inferior vestibular nerve afferents (Rosengren and Kingma, 2013). Similar research suggests that the skull vibration-evoked oVEMP is a test mainly of superior nerve afferents (Rosengren and Kingma, 2013), but this might depend upon the particular vibration stimulus used. Combined, this evidence suggests that the sound-cVEMP might originate predominantly in the saccule and, depending on the stimulus, the vibration-oVEMP mainly in the utricle. The reflexes also test two different pathways: the vestibulo-collic (VCR) and vestibulo-ocular (VOR) reflex pathways. We tested the effect of alcohol on cVEMPs evoked by air-conducted sound and oVEMPs evoked by skull vibration. We chose these stimuli as they produce the most robust responses for each reflex. Only one study has previously investigated the effect of alcohol on the cVEMP (Chiang and Young, 2007). The authors tested normal volunteers before and after consumption of a dose of alcohol designed to bring subjects close to the local legal limit for driving. They found no effect of alcohol on sound-evoked cVEMP amplitude, but a small elongation of p13 latency. To extend these findings, we aimed to measure both cVEMPs and oVEMPs at multiple breath alcohol concentration (BrAC) levels up to a higher maximum of 1.5 ‰ (per mil).

As alcohol is known to produce GEN, we also considered whether the presence of nystagmus per se might affect oVEMPs independent of the effect of alcohol. oVEMPs are also very sensitive to the angle of vertical gaze: they are typically recorded from beneath the eyes with gaze directed upwards (Govender et al., 2009). The n10 oVEMP peak is largest in this position because it originates in the inferior oblique muscle, which is active and close to the recording electrodes during up-gaze (Rosengren et al., 2013; Weber et al., 2012). With increasing alcohol consumption the incidence and strength of GEN increase, changing the average direction of gaze and possibly altering oVEMP amplitude. We sought to mimic the effect of GEN in sober volunteers by recording oVEMPs during optokinetic stimulation (OKS).

2. Methods

2.1. Participants

Fourteen healthy volunteers participated in Experiment 1: the effect of alcohol on cVEMPs and oVEMPs (mean age 29 years, range 24–38 years; 10 males, 4 females). Eleven different volunteers participated in Experiment 2: the effect of OKS on oVEMPs (mean age 33 years, range 26–46 years; 4 males, 7 females). In both cases the participants had no history of vestibular dysfunction, neurological disease or alcohol dependence. The participants gave informed written consent according to the Declaration of Helsinki and the study was approved by the local ethics committee (Kantonale Ethik–Kommission Zurich, 2010-0468).

2.2. Measurements

2.2.1. cVEMP recording

The cVEMP stimulus was an unshaped burst of sound (500 Hz, 4 ms), delivered at 126 dB peak SPL using headphones and a custom amplifier (TDH 39, Telephonics Corp.). The stimuli were generated with customized software using a laboratory interface (micro1401, Cambridge Electronic Design (CED)) and delivered at a rate of 7.5 Hz for 200 repetitions per trial. Subjects reclined to ~30 deg above horizontal and lifted and turned their heads away from the side of the stimulus for the duration of each trial. SCM