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Effects of muscle contraction on cervical vestibular evoked myogenic potentials in normal subjects

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HIGHLIGHTS

- The effect of sternocleidomastoid (SCM) contraction on cVEMP amplitude is strong and linear.
- Small muscle contraction differences have the same effect on cVEMP amplitude as 5–9 dB stimulus intensity changes.
- Minimum contraction levels are required for accurate interpretation of cVEMPs.

ABSTRACT

Objective: Cervical vestibular evoked myogenic potentials (cVEMPs) are vestibular-dependent muscle reflexes recorded from the sternocleidomastoid (SCM) muscles in humans. cVEMP amplitude is modulated by stimulus intensity and SCM muscle contraction strength, but the effect of muscle contraction is less well-documented. The effects of intensity and contraction were therefore compared in 25 normal subjects over a wide range of contractions.
Methods: cVEMPs were recorded at different contraction levels while holding stimulus intensity constant and at different intensities while holding SCM contraction constant.
Results: The effect of muscle contraction on cVEMP amplitude was linear for most of the range of muscle contractions in the majority of subjects (mean $R^2 = 0.93$), although there were some nonlinearities when the contraction was either very weak or very strong. Very weak contractions were associated with absent responses, incomplete morphology and prolonged p13 latencies. Normalization of amplitudes, by dividing the p13–n23 amplitude by the muscle contraction estimate, reduced the effect of muscle contraction, but tended to underestimate the amplitude with weak contractions.
Conclusions: Minimum contraction levels are required for accurate interpretation of cVEMPs.
Significance: These data highlight the importance of measuring SCM contraction strength when recording cVEMPs.

1. Introduction

Cervical vestibular evoked myogenic potentials (cVEMPs) are muscle reflexes produced by stimulation of the vestibular system with bursts of sound, vibration or galvanic stimulation. They are a type of vestibulo-collic reflex: mediated by the vestibular organs, vestibular nerve and nucleus, vestibulospinal tract, and accessory nucleus and nerve (Uchino and Kushiro, 2011). They are most commonly recorded from the sternocleidomastoid (SCM) neck muscles in response to stimulation with loud bursts of air-conducted (AC) sound (see Rosengren et al., 2010 for review). The reflex can be recorded from an active surface electrode placed near the middle of the SCM muscle belly and a reference over the medial clavicle, and appears as a short-latency, biphasic positive–negative potential with peak latencies of approximately 13 and 23 ms (i.e. p13–n23). cVEMPs evoked by AC sound are thought to originate predominantly in the ipsilateral saccule and are therefore used in neuro-otology settings as a test of saccular function (Rosengren et al., 2010). Left–right amplitude symmetry is typically the most important response metric, however amplitude is affected by two main factors in addition to the integrity of the sacculo-collic pathway: stimulus intensity and SCM contraction strength.

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Colebatch and Rothwell (2004) demonstrated that the p13–n23 potential is produced by an inhibition of the SCM muscle. In order to detect a reduction in muscle activity the muscle needs to be tonically active. cVEMP amplitude is greater during stronger muscle contractions as more tonically active units can be inhibited by the vestibular stimulus. The effect of contraction strength is common to many reflexes and is thought to be a general property of the motor unit pool, allowing reflexes to scale with contractions in order to maintain appropriate sensitivity (Matthews, 1986). The effect of muscle contraction on the cVEMP was noted in the first detailed report of the reflex (Colebatch et al., 1994), but has been systematically studied only rarely. Several studies have shown that the effect is mostly linear (Akin et al., 2004; Akin and Murnane, 2001; Colebatch et al., 1994; Lim et al., 1995; Watson and Colebatch, 1998), although there are some data suggesting a possible saturation effect with strong contractions (Colebatch et al., 1994; McCaslin et al., 2014). It has also recently been suggested that the relationship might be significantly nonlinear in some subjects (Bogle et al., 2013). However, there has been little detailed analysis of very strong or very weak contractions to date.

As the muscle contraction has a significant effect on cVEMP amplitude, electromyogram (EMG) monitoring and measurement are important. The most important goal is to ensure that the contraction is sufficient, so that a response is not missed due to a weak contraction. Previous authors have proposed minimum levels of contraction (e.g. 40–50 μV, Rosengren et al., 2010), though these values are typically based on clinical experience rather than experimental data. The second goal of monitoring the muscle contraction is to ensure a fair comparison across trials or between the left and right sides. If the SCM contraction is asymmetric, a patient may erroneously appear to have an asymmetric cVEMP.

Measurement of EMG allows the contraction strength to be matched across trials or sides, or to be used to correct for any contraction asymmetry. In the latter scenario, the EMG estimate is used to express the raw p13–n23 cVEMP amplitude as a ratio (or proportion) of the background muscle contraction, thus normalizing the amplitude measure (sometimes called a ‘corrected value’). If the muscle contraction effect is indeed linear, normalization procedures should cancel it (Colebatch et al., 1994; Lim et al., 1995). This was recently demonstrated at a group level for moderate to strong contractions (McCaslin et al., 2014). Data from normal subjects have also shown that normalized amplitudes are less variable (van Tilburg et al., 2014) and more symmetric (McCaslin et al., 2013; Welgampola and Colebatch, 2001). However, it is not clear if normalization is successful across a wide range of muscle contraction strengths in individual subjects. For example, if cVEMP amplitude saturates during very strong contractions the ratio would be expected to be underestimated for those trials (e.g. McCaslin et al., 2014).

Different forms of amplitude correction have been used for some time (Basta et al., 2005; Brantberg et al., 2003; Tseng et al., 2013; Vanspauwen et al., 2009; Welgampola and Colebatch, 2001). However, it is not known if the normalization technique is always successful. One problem is that individual measures or ranges of muscle contraction strength are often not reported in the literature, even in studies specifically examining the effects of muscle contraction. Data are also sometimes averaged over multiple trials or sides, obscuring the natural variability of muscle contractions, and it is important to know the maximum effect that muscle contraction strength or asymmetry can have in a single patient or trial, as individuals are the focus of clinical testing. The primary objective of the current study was therefore to investigate the effect of muscle contraction on cVEMP parameters over a wide range of SCM contraction strengths. Particular emphasis was placed on the range of values obtained, to record the extremes that can occur under different test conditions.

The second objective was to compare the magnitude of the muscle contraction and stimulus intensity effects. The question was: how much is cVEMP amplitude altered by small changes in muscle contraction and what degree of stimulus intensity change would produce the same effect? This enabled the muscle contraction effect to be expressed in terms of equivalent sound intensity units (dB), which are typically more familiar to clinicians than EMG units (e.g. mean rectified EMG in μV), to highlight the magnitude of the background contraction effect.

2. Methods

2.1. Subjects

Twenty-five normal volunteers with no history of vestibular dysfunction or neurological disease were tested (mean age 35 years, range 22–62 years; 6 males, 19 females). The participants gave informed written consent according to the Declaration of Helsinki and the study was approved by the local ethics committee (X13-0270 & HREC/13/RPAH/354).

2.2. Stimulation and recording parameters

Subjects were stimulated in one ear (12 right, 13 left, selected pseudo randomly). As the effects of two experimental factors were investigated, muscle contraction and stimulus intensity, only one ear was tested to minimise the duration of the experiment. The stimulus was an AC tone burst (500 Hz, 2 ms plateau, 0 ms rise/fall) delivered using headphones and a custom amplifier (TDH 39, Telephonics Corp., Farmingdale, USA). The stimuli were generated with Signal software and a laboratory interface (micro1401, both from Cambridge Electronic Design Ltd [CED], Cambridge, United Kingdom) and delivered at a rate of 7.5 Hz for 200 repetitions per trial. Stimulus intensities ranged from 112 to 136 dB peak sound pressure level (SPL, 84–108 dB L_{Aeq}, integrated A-weighted intensity at equivalent sound level). SCM muscle activity was recorded bilaterally (in 21 subjects and ipsilaterally only in 4) from surface electrodes (Clear trace, Conmed Corp., Utica New York, USA) placed over the SCM muscle belly (active, inverting) and medial clavicle (reference). An earth electrode was placed on the sternum. EMG was sampled at 10 kHz from 20 ms before to 80 ms after stimulus onset, amplified and bandpass filtered (5 Hz–2 kHz), using 1902 amplifiers (CED) and the same micro1401 data acquisition interface and Signal software as described above. Negative potentials at the active electrodes were displayed as upward deflections.

2.3. Procedure

The effect of stimulus intensity was tested while keeping the SCM muscle contraction relatively constant and the effect of muscle contraction was tested while keeping the stimulus intensity constant. Subjects first reclined to approx. 20° above horizontal, lifted their head off the bed, faced forwards and held their head against gravity. In this position, the test ear was stimulated at maximal intensity (136 dB peak SPL) and EMG from both sides of the neck was measured to assess contraction symmetry ($N = 21$ subjects). The intensity was then systematically decreased in steps of 3 dB until threshold was reached ($N = 25$). Trials were repeated near threshold to confirm the presence of a response. The experimenter aimed to keep the muscle contraction on each trial as close as possible to the contraction measured on the first trial. To achieve this, rectified EMG was monitored in real time and subjects were instructed to turn their head slightly during the head lift when required.