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A source analysis of short-latency vestibular evoked potentials produced by air- and bone-conducted sound

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Abstract

Objective: To map short-latency vestibular evoked potentials (VsEPs) using air- (AC) and bone-conducted (BC) sound and to perform source analysis to determine their origin.
Methods: Ten normal volunteers, chosen to have low-normal thresholds for acoustic vestibular activation, participated. In the first part, the subjects' individual thresholds for vestibular activation (V_T) were established using vestibular evoked myogenic potentials (VEMPs) recorded from the sternocleidomastoid muscles. AC sound was delivered with headphones and BC sound with a commercial B71 bone vibrator. In the second part, VsEPs were recorded using Ag/AgCl scalp electrodes in a 10–20 montage supplemented by infra-ocular, mastoid and cerebellar electrodes. Stimuli were 2 ms pips, consisting of a single cycle of 500 Hz, presented at +18 dB re V_T ("vestibular" condition) and −3 dB re V_T (control condition).
Results: Following the control stimulus, auditory mid-latency responses (MLRs) were observed. In the vestibular condition, two dominant groups of non-MLR potentials of presumed vestibular origin appeared (vestibular evoked potentials, or VsEPs), which consisted of a P10–N17 complex maximal at Pz, and an N15–P21 complex maximal at Fpz. Large potentials were also recorded from the infra-ocular electrodes at similar latencies. Source analysis indicated that the two complexes were largely accounted for by a combination of ocular vestibular evoked myogenic potentials (OVEMPs) and sub-cortical sources (possibly vestibular cerebellum), with a smaller contribution from anterior cortical and other myogenic sources.
Conclusions: Both the N15 and P10 potentials appear to receive an ocular myogenic contribution but both appear also to receive a contribution from other central structures.
Significance: The P10 and N15 complexes appear to represent the activity of otolith-dependent projections.

Keywords: Vestibular; Evoked potentials; Source mapping; Otoliths

1. Introduction

In humans, projections from canal and otolith afferents are likely to underlie the vestibular contribution to our ability to, for example, distinguish between self-motion and motion of the environment, conditions under which visual input may be identical (Benson, 1982). Evoked potentials are a well-established method of investigating afferent projections to the cerebral cortex and can be applied to any situation in which it is possible to evoke a synchronous volley of afferent activity. This is difficult, however, for vestibular afferents using their natural stimulus – rotational and linear movement – because synchronous activation is hard to achieve using imposed movements and the results are prone to stimulus artefact. Nevertheless, very rapid imposed head rotations have been reported to evoke short-latency potentials (Hood and Kayan, 1985; Sohmer et al., 1999). Problems also arise with the use of caloric or surface galvanic

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stimulation which co-activate somatosensory and pain receptors in the aural or mastoid region. de Waele et al. (2001) used direct intraoperative vestibular nerve stimulation to achieve a synchronous vestibular afferent volley in anaesthetised patients and evoked a series of potentials in scalp electrodes, in particular, in frontal locations. However, this is not a procedure which can be routinely used in normal subjects or in other patient groups.

An alternate method for synchronously activating the vestibular system is by means of acoustic stimulation, in conjunction with the use of suitable auditory controls. This technique exploits a conserved vestibular sensitivity to sound (e.g. Todd and Merker, 2004), and has been developed and validated using vestibular evoked myogenic potentials (VEMPs) recorded from the sternocleidomastoid (SCM) muscles (Colebatch et al., 1994; Welgampola et al., 2003). Todd et al. (2003) used acoustic vestibular stimulation with a limited electrode array to demonstrate the existence of short-latency vestibular evoked potentials (VsEPs) produced by bone-conducted sound. These had some similarities with the potentials observed by de Waele et al. (2001), most notably in terms of latency and in showing a strong frontal distribution, but differed in other respects, particularly in terms of scalp distribution. Two potentials, in particular, were observed, which were considered likely to be vestibular and not auditory. These were a P10 which was maximal at Cz and an N15 which was maximal at Fpz. More recently, Rosengren and Colebatch (2006) confirmed the vestibular dependence of these potentials, by recording them in a group of patients with severe to profound bilateral hearing loss.

On the basis of scalp distribution Todd et al. (2003) argued that the P10 was likely to be cortical in origin, whereas the N15 was most likely ocular. Subsequent studies have demonstrated that the N15 has an ocular myogenic origin (Rosengren et al., 2005; Todd et al., 2007). By means of a bipolar surface electrode montage Todd et al. (2007) characterised ocular vestibular evoked myogenic potentials (OVEMPs), consisting of positive and negative potentials, the size and polarity of which depended on the electrode location as well as the stimulus modality (air or bone-conducted). The OVEMPs peaked at short-latency (~10–13 ms) and preceded any sound evoked eye movement. Whilst a cortical origin for the P10 complex was presumed, on the basis of the limited electrode array used, it was not possible to speculate beyond a source consisting of a bilateral pair of dipoles or a single midline dipole. Thus, the principal aim of this study was to map the P10 complex using a higher resolution 10–20 montage and to perform source analysis. Both air-conducted (AC) and bone-conducted (BC) acoustic stimuli were used: AC stimuli to explore the effects of unilateral saccular activation (McCue and Guinan, 1994; Murofushi and Curthoys, 1997) and BC to explore the effects of activation of both otolith receptors bilaterally (Curthoys et al., 2006).

2. Methods

2.1. Subjects

Ten normal subjects with no hearing or vestibular deficits were recruited from staff and students at the Prince of Wales Hospital, Royal Prince Alfred Hospital and University of Western Sydney (4 males, 6 females; mean age 32 yrs, range 24–43 yrs). Subjects were selected from an initial population of 13 for low, but normal, VEMP thresholds to sound. A patient suffering from Meniere's disease (male, 63 yrs of age) was also tested 1 month before and 6 months after left vestibular neurectomy. All subjects gave informed consent according to the Declaration of Helsinki. The methods were approved by the Local Ethics Committees.

2.2. Stimuli

The experimental stimuli employed for obtaining VEMPs and VsEPs were AC and BC 2 ms, 500 Hz, single cycle tone pips. As any VsEPs would be potentially mixed with auditory evoked potentials (auditory brainstem responses [ABR] and mid-latency responses [MLR]), an auditory control was used, consisting of the same waveform but at low intensity. The intensity of the stimuli was measured in terms of peak-to-peak amplitudes. For AC sound, the maximum output within the acceptable linear range was a drive of 10 V peak to peak (pp), equivalent to a peak SPL of 141.4 dB re 20 μPa (as measured by the L_{Lpk} parameter with linear frequency weighting) and a RMS SPL of 121.2 dB re 20 μPa (measured by the L_{AI} parameter, with A-frequency weighting and impulse time weighting). For the bone-conductor, this drive was 20 V pp, equivalent at 500 Hz to a peak force level of 135 dB (re 1 μN), a peak acceleration level of 27 dB re 1 g and an RMS acceleration level of 12.5 dB re 1 g (using the same weightings as above). Intensities are expressed in dB relative to these maximum input values. Calibrations were carried out using a 2260 Investigator (Brüel and Kjaer, Naerum, Denmark) with a pressure-field microphone (Model 4134) and artificial ear (Model 4153) for AC sound and an artificial mastoid (Model 4930) for BC sound. The stimuli were generated with alternating polarity by means of customised software, using a laboratory interface (1401plus, Cambridge Electronic Design, Cambridge, UK) and custom amplifier. They were delivered by headphones (TDH 49, Telephonics Corp., Farmingdale, NY, USA) or by a commercial bone-conductor (B71, Radioear Corp., New Eagle, PA, USA). The patient with Meniere's disease was stimulated with AC clicks of 0.1 ms duration at 141 dB re 20 μPa.

2.3. Experimental procedure

Before the experiment the subjects were informed of the procedure and asked to sign a consent form. Screening