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Frequency characteristics of cortical activity associated with perturbations to upright stability

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ABSTRACT

Cortical evoked potentials are evident in the control of whole-body balance reactions in response to transient instability. The focus of this work is to continue to advance understanding of the potential cortical contributions to bipedal balance control. Temporally unpredictable postural perturbations evoke a negative potential (N1), which has drawn parallels to error-related negativity (ERN) as well as visual and auditory evoked N1 responses. The mechanism underlying the generation of event-related potentials (ERPs) has been a matter of debate for the past few decades. While the evoked model proposes that ERPs are generated by the addition of fixed latency and fixed polarity responses, the phase reorganization model suggests that ERPs are the result of stimulus-induced phase reorganization of the ongoing oscillations. Previous studies have suggested phase reorganization as a possible mechanism in auditory N1, visual N1 and error-related negativity (ERN). The purpose of the current study was to explore the frequency characteristics of the cortical responses to whole-body balance perturbations. Perturbations were evoked using a lean and release protocol. The results revealed a significant power increase and phase-locking of delta, theta, alpha, and beta band activity during perturbation-evoked N1. This may suggest that the stimulus-induced phase reorganization of the ongoing electroencephalographic (EEG) activity could account for the features of cortical ERPs in response to perturbation of upright stability.

1. Introduction

Cortical activity evoked by external stimuli or events provide important insight into the underlying spatial and temporal aspects of nervous system control of behavior. We have a specific interest in understanding the potential role of such cortical evoked potentials that are evident in the control of whole-body balance reactions in response to transient instability. Numerous studies have shown that temporally unpredictable postural perturbations such as platform translations, chair tilt, and standing lean and release elicit a negative potential (N1), which is a pronounced and consistent feature of the perturbation-evoked cortical response (see [15,16] for review). The peak of the N1 response occurs approximately 100–200 ms after the perturbation onset in the fronto-central region of the cerebral cortex with peak amplitude at FCz [17–20]. It has been suggested that the N1 represents the sensory processing of balance disturbances [18]. In addition, it has been speculated that the N1 may represent an error-related negativity (ERN) that may be associated with a specific cortical area (anterior cingulate) [20,21]. Understanding of the cortical role in balance control may be found in parallel to event related potentials evoked by other stimuli. As noted, there are proposed links between the perturbation-evoked N1 and the ERN [18,20,21]. In addition, there are stimulus-evoked N1 responses to visual or auditory stimuli that share some characteristics with perturbation evoked potentials [1–4]. One intriguing characteristic is that there is a temporal synchronization of the evoked frequency components.

For the past few decades there has been ongoing debate regarding the genesis of event-related potentials (ERP) [1–14]. A conventional view is that ERPs are generated by superposition of fixed latency and fixed polarity responses on to the ongoing EEG activity (evoked or additive model). Alternatively, the cortical potentials may instead reflect event-related phase reorganization of the ongoing EEG oscillations (phase resetting or reorganization model) (see [13,14] for review of both models). The distinction

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between these different explanations for the genesis of the ERP is important as they may have a significant impact on the interpretation of the associated cortical events. EEG phase reorganization has already been reported in response to auditory and visual stimuli [1–4]. In addition, the ERN associated with incorrect motor responses has been suggested to be the result of the partial phase-locking of theta band (4–7 Hz) EEG activity [5]. The intracranial EEG recordings also showed phase resetting of ongoing oscillations during a working memory task [8]. Such work leads to the view that the characteristics of the frequency responses to such stimulus-evoked responses may have the potential to yield insight into underlying control.

Unlike stimulus-evoked responses associated with visual or auditory stimuli, there has been little attention paid to the frequency characteristics of the perturbation-evoked N1 response. The purpose of the current study was to explore the frequency characteristics of the cortical responses to whole-body balance perturbations. This will allow a comparison of cortical activity associated with the control of balance reactions to other stimulus-evoked negativities which may, in turn, provide some insight into the genesis of the perturbation-evoked N1 response.

2. Materials and methods

Adults ($n = 14$, 5 females) aged 26.6 ± 4.4 years voluntarily consented to participate. The study protocol was approved by the Office of Research Ethics at the University of Waterloo. Perturbations were induced using a custom-made lean and release cable system [22]. The participants stood on a force plate (AMTI, Watertown, MA) [23] and leaned forward at load on a horizontal cable of 5–7% of body weight. Temporally unpredictable perturbations were triggered by manually releasing the cable (Supplementary Fig. 1). The lean angle was selected to evoke a feet-in-place reaction to recover balance (no stepping or grasping). A total of 40 trials were collected from each participant (interstimulus interval ranged 1–15 s).

Electromyography (EMG) (bilateral medial gastrocnemius and tibialis anterior), center of pressure (COP), and cable force were collected along with synchronized 64-channel EEG referenced to linked mastoids (Neuroscan, El Paso, TX). All the data were sampled at a rate of 1000 Hz. Electrooculographic signals were also recorded using four electrodes. Impedance of all electrodes was kept less than 5 kΩ throughout the experiment. EEG signals were recorded, amplified, and filtered (DC-300 Hz) online using a SynAmps2 amplifier and SCAN 4.3 (Neuroscan, El Paso, TX). EMG, COP, and cable force were filtered and analyzed for the measurement of reaction time and any evidence of pre-perturbation activity. Cable force was used to determine the onset of perturbation [22].

EEG signals were preprocessed using Neuroscan EDIT 4.3. Data were band pass filtered (1–30 Hz), epoched around the perturbation onset (−600 ms to +500 ms), and baseline corrected (baseline period: −600 ms to −500 ms). Post-processing was performed using EEGLAB [24]. Independent component analysis (ICA) was performed on EEG data to remove ocular, muscular, cardiac, and line noise artifacts. ICA-pruned EEG data was once again visually inspected to further remove epochs contaminated by gross movements that were not removed by ICA noise reduction. ICA was also used to identify the brain source that generated the perturbation-evoked N1. ERP scalp maps were plotted to visualize the scalp topography of the perturbation-evoked cortical response. To visualize the phase-locking across trials, phase-sorted ERP-images (a two-dimensional image with color-coded single-trials sorted in order of phase and stacked) were plotted [24]. Power spectral analyses using the Matlab pwelch function were performed for averaged ERP and unaveraged EEG epochs at specific frequencies. This computed the mean log power spectrum of data epochs and plotted the scalp distribution of power at discrete frequencies (2, 6, 8, 10, 12, 14, 20 Hz). The similar topography of EEG and ERP power indicates that ERPs are generated by the synchronization of ongoing EEG oscillations [3,12]. Event-related spectral perturbations (ERSP), which measure the mean event-related changes in spectral power over time, were computed at the FCz electrode site using Fast Fourier Transform (FFT) and the corresponding significant ($p < 0.01$) time-frequency maps were plotted. Event-related inter-trial coherence (ITC; also called 'phase-locking factor') was computed to verify significant ($p < 0.01$) phase-locking across trials with respect to perturbation onset [3,24].

3. Results

Compensatory balance reactions were consistently evoked as reflected by medial gastrocnemius EMG activation and associated COP excursion. On average, the onset of balance reactions was 182.4 ms (SD 10.3) and 184.1 ms (SD 7.3) for the left and right medial gastrocnemius, respectively. The peak of the perturbation-evoked N1 response at FCz had a mean latency of 107.9 (SD 8.3) ms and mean amplitude of 30.85 (SD 10.95) μV (Fig. 2C, bottom panel). The scalp topography showing N1 concentrated in fronto-central areas (Fig. 1A) is consistent with previous studies [15–20]. The power spectral maps of unaveraged EEG (Fig. 1B) and averaged ERP (Fig. 1C) data during the 20–180 ms post-stimulus interval showed similar scalp topography. The scalp distribution of power in the delta, theta, alpha, and beta frequencies showed power increases concentrated at fronto-central midline electrode sites. There was considerable inter-subject variability in the N1 latency as seen in the ERP-image at the FCz electrode site (Fig. 2A). The ERSP plot (Fig. 2B), which gives the total power without regard to the phase of the signal, showed a significant increase ($p < 0.01$) in delta, theta, alpha, and beta power during the N1 period. The event-related ITC plot (Fig. 2B), which gives the degree of event-related phase consistency across trials, revealed significant ($p < 0.01$) phase-locking of 1–20 Hz activity during the N1 period. Power increase and phase coherence in the lower frequency bands appear to occur before the perturbation onset, but this may be an artifact associated with the poor time resolution of windowed FFT in lower frequency bands. Phase-sorted ERP-image plots (Supplementary Fig. 2) also showed significant synchronization of delta, theta, alpha, and beta activity during N1. The variability in the phases across trials seen in the phase-sorted ERP-images is likely due to the difference in N1 latencies between subjects. ICA analysis revealed that a fronto-central component (FC) mainly accounted for the generation of N1 (Supplementary Fig. 3). The scalp topography (Fig. 3A) of FC showed fronto-central concentration and, indeed, the component ERP-image (Fig. 3B) and average component ERP (Fig. 3D, bottom panel) of the FC closely resembled those of the FCz electrode site. The ERSP plot (Fig. 3C) and ITC plot (Fig. 3D) of the FC showed significant ($p < 0.01$) power modulation and phase consistency across trials, respectively, during the N1 period.

4. Discussion

This study is the first to examine the frequency modulations associated with the cortical N1 response evoked by unpredictable whole-body balance perturbations. The present results reveal evidence of phase synchronization associated with the perturbation-evoked N1 response. This observation is consistent with the previous findings for visual N1, auditory N1, ERN, and ERP during a working memory task [1–8]. In the present study, phase-locking of ongoing oscillations is most clearly revealed by the uniform phase distribution across trials in the phase-sorted ERP-image and event-related ITC plots. It is interesting to note that the