Neuroscience Letters 599 (2015) 75–80

Contents lists available at ScienceDirect

Neuroscience Letters

journal homepage: www.elsevier.com/locate/neulet

Research article

Temporal and spatial organization of gait-related electrocortical potentials

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HIGHLIGHTS

- A gait-related cortical potential (GRCP) occurs over the motor cortex during walking.
- The temporal pattern of the GRCP is phase-locked to specific events in the gait cycle.
- A widely distributed cortical network is involved in gait control.

ARTICLE INFO

Article history:
Received 6 January 2015
Received in revised form 15 May 2015
Accepted 19 May 2015
Available online 21 May 2015

Keywords:
Electroencephalography
Gait
Movement-related cortical potential
Source localization

ABSTRACT

To advance gait rehabilitation research it is of great importance to understand the supraspinal control of walking. In this study, the temporal and spatial characteristics of averaged electrocortical activity during treadmill walking in healthy subjects was assessed. Electroencephalography data were recorded from 32 scalp locations, averaged across trials, and related to phases of the gait cycle based on the detection of left heel strike. A characteristic temporal pattern of positive and negative potentials, similar to movement-related cortical potentials, and related to the gait cycle was observed over the cortical leg representation area. Source localization analysis revealed that mainly the primary somatosensory, somatosensory association, primary motor and cingulate cortex were activated during walking. The negative peaks of the gait-related cortical potential were associated with activity predominantly in the cingulate and prefrontal cortex, while the primary motor, primary somatosensory and somatosensory association cortex were mainly active during the positive peaks. This study identified gait-related cortical potentials during walking. The results indicate a widely distributed cortical network involved in gait control.

1. Introduction

Bipedal locomotion is a fundamental everyday-life skill that allows independent mobility and is, therefore, a key determinant for the quality of life. It is generally accepted that walking involves a complex interaction between supraspinal centers, central pattern generators (CPGs) and multi-sensory peripheral sources [9]. However, the exact neurophysiological mechanisms are still unclear and further research is necessary to advance neurological gait rehabilitation.

Providing insight into the spatial organization of the supraspinal control of walking has been difficult, as most neuroimaging techniques are not adequate for recordings in dynamic situations [3]. The first attempts were made by means of single-photon emission computed tomography and functional near-infrared spectroscopy. These studies found that mainly the sensorimotor areas (primary somatosensory cortex (S1), somatosensory association cortex (SA), primary motor cortex (M1), pre-(PMC) & supplementary motor cortex (SMA)) [10,20], the cingulate cortex (CC) [10], the prefrontal cortex (PFC), the cerebellum and basal ganglia [10] are active during walking. More recently, positron emission tomography (PET) [15], functional magnetic resonance imaging (fMRI) [17] and electroencephalography (EEG) [11,33,34] have found similar brain areas.

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http://dx.doi.org/10.1016/j.neulet.2015.05.036
0304-3940/

Furthermore, fMRI and PET studies also identified the parahippocampal gyri [15], the thalamus and parts of the insular cortex [17] as contributors to the supraspinal control of walking.

An important advantage of EEG is its usability in dynamic situations and its high temporal resolution enabling researchers to link brain potentials to the timing of lower limb movements [3]. One of the earliest and very consistent findings with regard to EEG and voluntary movement is the so-called movement-related cortical potential (MRCP), see [31] for a review. The MRCP is a small (i.e., <10 μV) and slow brain potential, starting 2 s to 400 ms prior to the onset of a voluntary movement over the sensorimotor cortices. It is characterized by a slow negative slope (i.e., readiness potential), followed by a plateau tendency (i.e., motor potential) and a quick change from a negative to a positive potential immediately after the movement (i.e., reafferent potential) [1,31]. Recently, two studies reported on MRCPs during complex movements. Wieser et al. found a characteristic pattern of positive and negative brain potentials that was closely related to automated stepping movements on a vertical tilt table [34], while Jain et al. revealed slow cortical, alternating field potentials in relationship to the phases of the pedaling cycle, a locomotor-like task [12]. Yet until now no study explored the spatial and temporal characteristics of an MRCP during actual walking. Only two studies have measured EEG during real walking, but mainly looked at characteristics of the frequency spectra [11,30], see [3] for a review. Therefore, the purpose of this study was to investigate (1) whether an averaged electrocortical potential could be identified during walking, (2) whether this potential was in temporal relation to the gait cycle and (3) which sources were underlying this potential. It was hypothesized that a characteristic waveform, similar to an MRCP, would be present, closely related to the phases of the gait cycle and that this waveform would be prominent at electrodes overlying the cortical leg representation area.

2. Materials and methods

Ten healthy subjects (3 men, 7 women; mean age 28.2 SD 4.1 years) gave their written informed consent to participate in this study. All experimental procedures were performed according to the standards set by the declaration of Helsinki and approved by the local medical ethics committee. First, the subjects' preferred walking speed on the treadmill was determined. This was followed by the experimental protocol consisting of 5 min of seated rest, 5 min of quiet stance and 20 min of treadmill walking. Participants were given instructions to keep their natural arm swing, relax their muscles, avoid head movements and look at a point in front of them.

Continuous EEG was recorded from 32 active electrodes placed according to the international 10–20 system [13] (Fp1/2, F3/4/7/8/z, FC1/2/5/6, T7/8, C3/4/z, TP9/10, CP1/2/5/6, P3/4/7/8/z, PO9/10, O1/2/z) (Brain Products GmbH, Germany). Sampling rate was 1000 Hz and electrode impedance was <5 kOhm. A force sensing resistor placed on the subjects' left heel detected left heel strike (LHS) and was synchronized with the EEG data. The other events in the gait cycle were estimated based on the moment of L HS. Data were processed using EEGLAB [6]: filtered (0.5 Hz high-pass, 30 Hz low-pass, 50 Hz notch filter), segmented based on L HS and interpolated as such that the length of each gait cycle was normalized to 1000 ms. Subsequently, artifactual epochs were removed in three steps. First, an automatic artifact detection procedure was applied, removing epochs with values (a) ≥ ± 100 μV, (b) ≥ 5 SDs of the mean kurtosis, (c) ≥ 5 SDs of the mean probability distribution, (d) drifts ≥ 50 μV/epoch and R-square limit ≤ 0.3 and (e) spectra deviating from the mean by ±50 dB in the 0–2 Hz and by +25 or −100 dB in the 20–30 Hz frequency window. Second, remaining epochs were visually inspected for artifacts. Third, independent component analysis was used to parse EEG signals into spatially static, maximally independent components (ICs) [16]. The DIPFIT function of EEGLAB [24] was used to compute an equivalent current dipole model that best explained the scalp topography of each IC. ICs were removed if the projection of the equivalent current dipole model to the scalp accounted for less than 75% of the scalp map variance, or if the topography, time-course and power spectrum of the IC was reflective of artifacts [14,18]. The remaining ICs were projected back onto the scalp channels to produce artifact-corrected epochs. Data were then re-referenced to an average of all electrodes and all epochs were averaged to one ensemble-averaged electrocortical potential around L HS for each electrode and each subject. For EEG data recorded during rest and stance, the same procedure was followed, except that data were randomly segmented to epochs of 1000 ms. From the averaged electrocortical potential during walking, the latency and amplitude of the most important peaks at Cz, compared to rest and stance, were identified (Fig. 1A). The peak-to-peak amplitude of the ensemble-averaged potentials at all electrodes was calculated for the three conditions. Thereafter, distributions were checked for normality and a Wilcoxon signed ranks test with the level of significance fixed at $p < .05$ was performed to compare the peak-to-peak at Cz amplitude between conditions (IBM SPSS Statistics, version 22, Chicago).

Next, exact low resolution brain electromagnetic tomography (eLORETA) was used to compute the cortical 3D distribution of current density ($\mu V^2/mm^4$) for walking compared to rest and stance [25,26]. Current source densities in each voxel were compared by permutation tests (i.e., log of ratio of averages) with 5000 random permutations which accounts for the multiple comparisons that are inherent to the voxel-by-voxel hypothesis testing [21]. As a result a critical threshold (i.e., t-critical) is determined at a significance level of $p < .01$ (Table 1). Voxels with statistical values exceeding this threshold have their null hypothesis, i.e., no difference in current source densities, rejected. In Table 1, data are presented as the percentage of significant active voxels per functional brain area, relative to the total amount of voxels in that brain area, for walking compared to rest (i.e., entire time window 1–1000 ms) and for walking compared to standing (i.e., entire time window and the most important peaks: peak 1 = 4–54 ms, peak 2 = 116–166 ms, peak 3 = 487–537 ms, peak 4 = 611–661 ms). Fig. 1C displays the voxel-by-voxel *t*-values for walking compared to standing at the 4 peaks in Talairach space as statistical non-parametric maps (SnPMs), while Supplementary Fig. 2 displays the exact current density maxima during walking around the 4 peaks.

3. Results

3.1. Gait-related electrocortical potentials

The averaged electrocortical potentials during walking show alternate positive and negative potentials occurring once or twice per gait cycle at most electrodes. At the L and R hemisphere, a negative peak around the moment of ipsilateral HS (i.e., contralateral push-off) and a positive peak around ipsilateral push-off (i.e., contralateral HS) can be distinguished. At central electrodes (i.e., Fz, Cz), two positive and two negative peaks occurred: Fig. 1A shows that the positive peaks at Cz occurred at latencies 141 and 636 ms, close to L and R toe off, respectively. The negative peaks occurred at latencies 29 and 512 ms, close to R and L HS, respectively. The peak-to-peak amplitudes of the waveforms during walking (mean = 4.371, SD = 2.079 μV) were significantly higher ($p < .05$) compared to rest (mean = 1.719, SD = 0.501 μV) and stance (mean = 1.740, SD = 0.625 μV) for all electrodes.