Posture Alters Human Resting State

Robert T. Thibault, Michael Lifshitz, Jennifer M. Jones & Amir Raz


doi: 10.1016/j.cortex.2014.06.014

Copyright 2014 by the authors. This Open Access post print is distributed under the terms of the Creative Commons Attribution Non-Commercial, No Derivatives License.

http://creativecommons.org/licenses/by-nc-nd/2.0/ca
Posture alters human resting-state

Robert T. Thibault a, Michael Lifshitz a, Jennifer M. Jones a, & Amir Raz a,b,*

a McGill University, 3775 University, Montreal, QC, H3A 2B4, Canada.
b The Lady Davis Institute for Medical Research & Institute for Family and Community Psychiatry, Jewish General Hospital, 4333 Cote Ste. Catherine, Montreal, QC, H3T 1E4, Canada

*Please address correspondence to:
Professor Amir Raz, 4333 Cote Ste. Catherine, Montreal, QC, H3T 1E4, Canada
amir.raz@mcgill.ca
Tel: 1-514-340-8210; Fax: 1-514-340-8124
ABSTRACT

Neuroimaging is ubiquitous; however, neuroimagers seldom investigate the putative impact of posture on brain activity. Whereas participants in most psychological experiments sit upright, many prominent neuroimaging techniques (e.g., functional magnetic resonance imaging (fMRI)) require participants to lie supine. Such postural discrepancies may hold important implications for brain function in general and for fMRI in particular. We directly investigated the effect of posture on spontaneous brain dynamics by recording scalp electrical activity in four orthostatic conditions (lying supine, inclined at 45°, sitting upright, and standing erect). Here we show that upright versus supine posture increases widespread high-frequency oscillatory activity. Our electroencephalographic findings highlight the importance of posture as a determinant in neuroimaging. When generalizing supine imaging results to ecological human cognition, therefore, cognitive neuroscientists would benefit from considering the influence of posture on brain dynamics.

Keywords

EEG, fMRI, neuroimaging, posture, supine position.
1. INTRODUCTION

Neuroimagers typically assume that body-position scantily affects neural activity (Raz et al., 2005). Here we challenge this tacit assumption by demonstrating that posture rapidly changes oscillatory dynamics of the resting brain as measured by electroencephalography (EEG). Sparse findings show that orthostatic variations (e.g., sitting upright, lying supine, standing erect) modulate specific cognitive processes and sensory thresholds; for example, body-position alters visual perception (Goodenough et al., 1981), problem solving (Lipnicki & Byrne, 2005), anticipatory anxiety (Lipnicki & Byrne, 2008), pain sensitivity (Spironelli & Angrilli, 2011), and odor discrimination (Lundström et al., 2008). Comparing postures using a stance-adjustable positron emission tomography (PET) gantry, moreover, studies reported signal differences across postures in a wide range of cortical and subcortical regions (Ouchi et al., 2001; Ouchi et al., 1999). These collective findings propose posture as a modulator of neural activity. Although a few studies have found changes in EEG as a function of posture (Chang et al., 2011; Cole, 1989; Rice et al., 2013), these efforts shied away from directly testing and addressing how posture may influence brain activity in canonical imaging contexts such as those common to fMRI and EEG. The present account addresses this lacuna.

2. MATERIALS AND METHODS

2.1 Participants

Nineteen participants provided written informed consent in accordance with the Research Ethics Board at McGill University and in compliance with the Code of Ethics of the World Medical Association – Declaration of Helsinki – before the experiment. We excluded data from seven participants because at least one of their recordings contained fewer than four two-second
epochs without blinking artifacts. Although we excluded only 4% (13/304) of all 30-second trials, adhering to a fully factorial design required we exclude 37% (7/19) of participants. All twelve participants whose data we included (mean age = 20.5 ± 2.0 years; nine females) reported having consumed no nicotine and no more than one caffeinated beverage on the day of testing.

2.2 Experimental procedure

Participants randomly transitioned among four postures (supine, 45° incline, sitting, and standing; see Fig. 1.1). For each posture, participants underwent a 30-second adaptation followed by a 30-second EEG recording in four counterbalanced conditions: eyes closed with mental counting task, eyes closed with no task, eyes open with mental counting task, and eyes open with no task. To avoid electrode contact with the table and artifacts produced by neck muscles, participants used neck-support throughout the experiment.

2.3 Electroencephalography

We collected high-density EEG data from 128 pin-type active electrodes using an ActiveTwo system (BioSemi, Amsterdam, The Netherlands) acquiring data using ActiView (BioSemi) at a sampling rate of 2048 Hz. We set filters to 0.5-70 Hz with a 60-Hz notch filter using 2-Hz width to eliminate electrical noise. Electrode impedances measured below 20 kOhms before each recording and neither drifted during the experiment nor changed as a function of specific postures. Throughout data acquisition and in line with the standard in the field, BioSemi equipment references electrodes to a signal formed by a Common Mode Sense active electrode and a Driven Right Leg passive electrode, located slightly occipitally from Cz (Metting van Rijn, Peper, & Grimbergen, 1990, 1991). Before analyzing the data, the Brain Electrical Source Analysis (BESA®) package re-referenced each electrode to the average of all EEG electrodes.

2.4 Setting
We partitioned an area of our laboratory measuring 3 x 2 x 2 m with grey-blue monochromatic sheets to control for visual stimuli across positions. We used squares of white tape measuring 3 x 3 cm as fixation points for conditions with open eyes. Depending on the specific posture, participants lay on a tilt table, sat upright in a chair, or stood flush with a wall in the middle of the testing area. The testing room was quiet throughout.

2.5 Data analysis

We manually scanned and labeled data with irregular high amplitude delta waveforms recorded by frontal electrodes as artifacts due to eye movement. We replaced electrode channels containing other ectopic waveforms with interpolated waveforms from surrounding electrodes using the BESA® package. We then fast-Fourier transformed all artifact-free 2-s epochs and calculated the average absolute power at each bandwidth using the $FFT_{average}$ function in BESA®. This function applies a cosine square window to the first and last 10% of each epoch to attenuate the amplitudes at the ends to zero. Using Statistical Analysis Software (SAS®), we performed a full-factorial three-way analysis of variance (ANOVA) (Posture x Task x Eye condition) on the logarithm of the absolute power (measured in $\mu V^2$) at each electrode for each bandwidth (delta ($\delta$) 0.5-4 Hz; theta ($\theta$) 4-8 Hz; alpha ($\alpha$) 8-14 Hz; beta ($\beta$) 14-30 Hz; and gamma ($\gamma$) 30-50 Hz). To account for multiple comparisons, we calculated an adjusted p-value for each dimension of the ANOVA at each bandwidth using positive false discovery rate (Storey, 2002). We corrected all pairwise comparisons using Tukey’s Honest Significant Difference Test. Using SAS® we confirmed normality and homogeneity of variance in each analysis. We generated color (heat) and electrode maps using MATLAB 7.11 (Mathworks, Natick, MA) and EEGLAB (Delorme and Makeig, 2004).
We performed a secondary analysis using a dipole source montage from BESA®. This montage employs spatial filtering to transform signals from recorded surface channels into fifteen virtual source channels inside the brain. These interpolated regions represent a single source with three single dipoles at the same location with orthogonal orientations. Finally, we corroborated our primary and secondary analysis on the main effect of posture using the power-spectrum density analysis function in Brainstorm 3.1 (Tadel et al., 2011). This function applies Welch’s method to obtain power spectra, and then performs Fourier transforms on the power spectra to obtain the average absolute power at each bandwidth.

2.6 Electromyography

To test whether muscle artifact contributed to our results we ran a control experiment on an additional six participants (mean age = 26 ± 10.7 years; three females) using flat-type active electrodes especially designed for recording electromyograms (EMGs). We placed six EMG electrodes at the following locations: the superior region of the left sternocleidomastoid; the superior region of the right trapezius; anterior to the earlobe on the left masseter; above the left eye; below it; and lateral to its temporal canthus (Fig. 2A). The EMG placed on the trapezius touched the neck support and received different amounts of pressure as a function of posture. To test whether postural effects on EEG signals are transient or long-lasting, we recorded two eight-minute runs with a one-minute break between runs. These participants either lay horizontally (supine) on a tilt table, lay at 45° (supine), or sat upright in a chair with eyes open and no task (Fig. 2C). Using BESA®, we removed vertical and horizontal electrooculogram (VEOG and HEOG) artifacts at a minimum of 250µV and 150µV, respectively, from the EEG electrodes only. We statistically analyzed both EMGs and EEGs using a two-way ANOVA (Posture x Run) using SAS®. All other aspects of the experiment (participants, experimental procedure,
electroencephalogram, setting, data analysis) for these six participants matched the above-mentioned procedures (i.e., sections 2.1 through 2.5).

3. RESULTS

Our primary analysis revealed a main effect of posture in the β and γ ranges (Fig. 1.1) and a main effect of eye condition across all waveforms except γ (Fig. 1.2). We found a main effect of posture on β-band activity over rostral frontal cortex as well as over medial and right occipital cortex (Fig. 1.1). Beta activity increased over frontal and occipital areas when inclined at 45° compared to supine (Fig. 1.1A), and increased over occipital regions when sitting upright compared to 45° incline (Fig. 1.1B). We also found a main effect of posture on γ-band activity distributed widely over the scalp. Gamma activity increased over lateral frontal regions when at 45° incline compared to supine (Fig. 1.1C), and increased over medial and right occipital regions when sitting compared to at 45° incline (Fig. 1.1D). Moreover, we observed widespread γ increases when sitting upright compared to lying supine (Fig. 1.1E). Both β and γ activity increased over frontal areas when at 45° incline compared to supine, and increased over occipital regions in sitting and standing erect positions compared to 45° incline. Across postures, eye closure instigated widespread increases in δ, θ, and α activity, as well as increases in β activity over dorsofrontal, parietal, and occipital regions (Fig. 1.2).

Our secondary analysis revealed a main effect of posture, eye condition, and task on the electrical activity of interpolated cortical areas (Fig. 1.3). Upright postures featured increased γ activity for all 15 brain regions and with increased β activity in all brain regions excluding central and left parietal areas (Fig. 1.3A). Eye closure featured increased δ, θ, and α activity in all cortical areas and with increased β activity in dorsofrontal, parietal, and occipital brain regions.
When performing a task, $\delta$, $\theta$, and $\beta$ activity decreased throughout the cortex, $\alpha$ activity decreased in frontal areas, and $\gamma$ activity decreased in midline and right parietal regions (Fig. 1.3C).

Our investigation of potential muscle artifact largely ruled out the involvement of muscular activity in the effects we report herein. Whereas posture altered EEG data in the gamma range congruent with experimental data from 12 participants (Fig. 2B), posture did not influence muscle activity based on recordings from EMG electrodes placed on the sternocleidomastoid muscle, on the masseter muscle, or around the eye ($p > 0.1$; Fig. 2A). EMG of the upper trapezius recorded an increase in gamma power in the sitting posture compared to lying down flat or inclining at an angle ($p < .05$; Fig. 2A). Postural effects were sustainable and comparable between runs ($p > .05$) at all EEG and EMG sensors.

4. DISCUSSION

Our findings indicate that orthostatic condition rapidly influences high-frequency cortical activity. The most prominent alterations occurred over occipital and frontal brain regions. $\beta$ and $\gamma$ activity increased from laying supine to inclining at $45^\circ$ and increased further when sitting upright. These changes manifested regardless of whether participants engaged in a cognitive task and irrespective of whether their eyes were open or closed. Changes appeared within 30 s and persisted for at least 16 minutes. Thus, our findings suggest a difference in baseline activity rather than transient event-related synchronizations or desynchronizations.

We obtained postural effects even for a small additional sample comprising the EEG data from six participants with EMGs. The EMG activity recorded from the trapezius changed across postures; both differential pressure on the electrode and increased neck tension across postures
may account for this difference. However, muscle activity alone is unlikely to account for the present EEG findings: 1. While posture altered baseline gamma activity in the posterior of the neck (trapezius), all other measured muscles – lateral neck and superior jaw muscles (sternocleidomastoid and masseter) as well as muscles superior, lateral, and inferior to the eye (frontalis and orbicularis oculi) – remained unchanged; 2. We observed scalp-wide postural EEG effects; 3. Another muscle, the temporalis, located on the scalp superior to the ear, might also produce muscle artifact. While our EMGs did not measure temporalis activity, the EEG sensors did. However, many of the EEG electrodes located over the temporalis showed no differences across postures (Fig. 1.1 and Fig. 2B).

If the trapezius were responsible for the observed changes, we would have expected a gradient of postural EEG effects – greatest around occipital regions and diminishing further away, weakest towards frontal areas. However, our data are inconsistent with this pattern (Fig. 1.1). Moreover, moderate lateralization typifies our present findings of changes in gamma oscillations – a result difficult to attribute to neck tension because participants faced symmetrically forward in all postures. Thus, cortical activity appears primarily responsible for our results.

Two physiological mechanisms likely contribute to the influence of posture on electrical scalp activity: 1) alterations in cerebrospinal fluid (CSF) thickness and 2) changes in noradrenergic output. First, because CSF is highly conductive, minute shifts in CSF concentration can cause substantial alterations in EEG signals (Ramon et al., 2006; Ramon et al., 2004; Wendel et al., 2008). Using upright and recumbent MRI scanners, findings demonstrated that intracranial CSF concentration decreased when sitting up compared with lying down (Alperin et al., 2005). Thus, CSF scattering may influence the propagation and recording of high
frequency cortical activity (Rice et al., 2013). Second, multiple reports suggest that altered noradrenergic output modulates EEG activity (Cole, 1989; Lipnicki, 2009; Schneider et al., 2008). When supine, gravity stimulates cardiopulmonary and arterial baroreceptors, reducing sympathetic system activation (Mohrman & Heller, 2003). This process decreases noradrenergic output from the locus coeruleus (Berridge & Waterhouse, 2003) and in turn dampens down cortical excitability (Rau & Elbert, 2001). Postural influences on EEG recordings, therefore, putatively involve alterations in both CSF thickness and noradrenergic output.

Head-direction neurons may also contribute to the observed changes between postures; however, this explanation is unlikely because head-direction cells are relatively insensitive to changes in the vertical planes (pitch and roll) and rely heavily on visual markers (Taube, 2007) which were absent in our visually uniform environment. Nonetheless, changes in vestibular inputs to head-direction cells (Yoder & Taube, 2014) may play some role in altering the recorded EEG signal. Future research relying on source localization would further elucidate the neural origin of posture-mediated EEG changes.

Triangulating data from converging methodologies would serve to illuminate the influence of posture on brain dynamics. Magnetoencephalography (MEG), for example, permits recording while sitting upright, reclining at a 0-90° angle, or laying supine – an advantageous feature for further characterizing neural patterns associated with body-position. MEG can complement other imaging modalities; for example, posture-induced changes in high-frequency cortical activity may confound fMRI data when investigating higher brain functions associated with β and γ oscillations (Siegel et al., 2012). Although upright MRI scanners for humans exist, they tend to employ low magnetic fields, which preclude fMRI sequences. Whereas posture may play an especially prominent role in regulating brain function in atypical populations such as the
elderly (Edlow et al., 2010) and specific patient groups (Ouchi et al., 2005; Thompson et al., 2005), unraveling the effects of posture on the typical human brain has at least three broad implications: 1) Overcoming orthostatic caveats associated with distinct scanning environments; 2) Developing compensatory computational models to improve the specificity and generalizability of brain imaging; and 3) Providing insights into brain states that rarely lend themselves to imaging postures (e.g., in contemplative practices (Brewer et al., 2011; Tang et al., 2012)). Regardless of whether cortical sources, muscle artifacts, or other parameters influence changes in brain activity, our findings highlight the importance of considering posture when unraveling oscillatory dynamics in the human brain. Unlocking the influence of posture on neural processing would pave the road to a more scientific understanding of this pervasive, albeit little acknowledged, ecological nuance.
AKNOWLEDGEMENTS

Dr. Amir Raz acknowledges funding from the Canada Research Chair program, Discovery and Discovery Acceleration Supplement grants from the Natural Sciences and Engineering Research Council of Canada (NSERC), Canadian Institutes of Health Research, and the Volkswagen Foundation (VolkswagenStiftung). Michael Lifshitz acknowledges a Francisco J. Varela Research Award from the Mind and Life Institute and a Vanier Canada Graduate Scholarship from NSERC.

AUTHOR CONTRIBUTIONS

A.R. conceived the idea. All authors contributed to experimental design. R.T.T. and J.M.J. collected data. R.T.T. and M.L. analyzed data. R.T.T. wrote a draft of the manuscript. M.L. and A.R. wrote the final version of the paper. All authors discussed the results and implications and commented on the manuscript at all stages.
FIGURES

Figure 1.1 β and γ activity differences among postures.

Dots represent electrodes where three-way ANOVAs yielded a significant main effect for posture (black: p < .05; red: p < .005). Color maps represent the average power at each electrode for that particular posture and bandwidth. Small electrode maps show significant Tukey-corrected pairwise comparisons between select postures. Dots represent an increase in power when moving toward upright postures. ANOVAs for δ, Θ, and α bandwidths were not significant.
Figure 1.2 Resting-state changes associated with eye closure.

Color maps represent the average power across postures and tasks at each electrode with eyes closed and eyes open. Electrode map dots display sensors where three-way ANOVAs yielded a significant main effect of eye condition (black: p < .05; red: p < .005). Eye closure increased δ, Θ, α, and β power, but had no significant effect on gamma activity.
Figure 1.3 EEG changes in interpolated cortical regions.

Dots represent underlying cortical regions where activity differed between conditions of posture, eye closure, and task (black: $p < .05$; red: $p < .005$). Topographic maps display regional sources interpolated via a 3D dipole head model. The brain regions presented consist of ten lateral regions (i.e., temporal-anterior, temporal-posterior, frontal-lateral, central-lateral, and parietal-lateral) and five midline regions (i.e., pre-frontal, frontal, central, parietal, and occipital).
Figure 2 EMG and EEG related changes.

Dots represent sensors. Black and red dots show an increase in power when moving toward upright postures. **A.** Schematic depiction of the dense-array electrodes (light blue with a dark center) and six EMG electrodes where two-way ANOVAs yielded statistically significant (red: \( p < .05 \)) and non-significant changes (green: \( p > .1 \)) between postures in the gamma bandwidth. **B.** Same ANOVAs across EEG scalp electrodes (red: \( p < .05 \) and black: \( p < .1 \)). **C.** Color maps represent the average power at each electrode for that particular posture. Small electrode maps show Tukey-corrected pairwise comparisons between select postures. ANOVAs for \( \delta \), \( \Theta \), \( \alpha \), and \( \beta \) bandwidths were not significant.
REFERENCES


