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Effects of attentional dispersion on sensory-motor processing of anticipatory postural control during unilateral arm abduction

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Highlights

1. This is the first study that systematically investigated the effects of attentional dispersion on sensory-motor processing of anticipatory postural control during unilateral arm abduction with several event-related potentials.

2. Attentional dispersion clearly affected important neural processing for the execution of unilateral arm abduction in our visuospatial cuing task, and the dispersion function was related to the onset time of postural muscle activation.

3. The results of this study can be effectively applied to elderly subjects or be used in clinical investigations for patients with sensory-motor impairments.

Abstract

Objective: We investigated effects of attentional dispersion on sensory-motor processing of anticipatory postural control during unilateral arm abduction.

***Methods*:** Thirteen adults performed arm abduction under two types of attentional dispersion conditions. A target stimulus was presented with 30% probability in two- or three-positions. By cue signal presentation, subjects either focused their attention on one position or divided attention for two or three positions and abducted right arm for target stimulus. Event-related potentials and onset time of postural muscles were measured.

Results: P1-N1 and N2 amplitudes decreased with attentional dispersion in both conditions, but P3 did not change. With attentional dispersion to three-positions, N2 latency increased and start of late CNV was delayed, and also the onset time of gluteus medius was late in correlation to the late CNV changings, with no changings in two-positions.

Conclusions: With attentional dispersion, brain activation decreased in the area related to the sensory processing and especially in the stimulus discrimination area. With increasing attentional dispersion, the delay in motor preparation or anticipatory attention to target stimuli was related to the delay in stimulus discrimination and onset time of postural muscle activation.

Significance: Effects of attentional dispersion on sensory-motor processing of anticipatory postural control were experimentally demonstrated.

1. Introduction

Many studies have shown that, with rapid arm movement while standing, postural muscles of the legs and trunk that control standing posture are activated before arm's focal muscles, to moderate postural disturbance caused by the arm movement (Belen'kiĭ et al., 1967; El'ner, 1973; Friedli et al., 1984; Horak et al., 1984). One of the factors affecting the dynamic postural control is the ability to disperse attention in response to environmental and individual conditions. Using simultaneously conducted cognitive and postural control tasks (i.e., dual task), the effects of attentional dispersion on these tasks have been investigated (Woollacott and Shumway-Cook, 2002; Maki and McIlroy, 2007). In dual task, the effects of attentional dispersion are evaluated as the decrease in performance of one or both tasks, which reflects just the result of total processing until motor output. Generally to perform reaction task to the sensory stimulation, neural processing includes the sensory, perceptual, and cognitive processing of stimuli, and the motor processing of preparation, selection, and execution of the responses. Both processing are presumably executed in parallel but integrated through an organizing system (Goodin and Aminoff, 1998). Also, it is reported that attentional allocation changes according to the significance of the processing (Lavie, 1995). However, for the studies concerned about the postural control, attention allocated to each neural processing has not been investigated until the present moment.

One well-known paradigm to manipulate attentional allocation is the visuospatial

cuing paradigm (Posner et al., 1980). In this paradigm, a cue signal is presented before the imperative stimulus for response and indicates the position where the stimulus will be presented. The number of possible positions presenting the imperative stimulus determines the attentional allocation. Using this paradigm, Tomita and Fujiwara (2008) investigated the relationship between attention-related modulations of visual-sensory processing and the activation pattern of postural muscles during unilateral arm abduction. They analyzed P1 and N1 components of event-related potentials (ERPs) from occipital electrodes contralateral to the visual stimuli. These components are reported to reflect the visual-sensory processing activity in extrastriate visual cortex (Hopfinger et al., 2001; Di Russo et al., 2003). Their results indicated that with attentional dispersion (only two positions), P1-N1 amplitude decreased and the onset time of postural muscle activation was late, and these changings were correlated. However, no studies have experimentally investigated the effects of attentional dispersion using ERPs on neural processing after visual-sensory processing, such as discrimination and cognition of stimuli, and motor preparation. Moreover, the relationship between these changings in neural processing and activation of postural muscles also remains unclear.

N2 component of ERP is reported to reflect the discrimination of the task-relevant visual features (Simson et al., 1977; O'Donnell et al., 1997). N2 is the posterior negativity with a latency of approximately 200 ms after the target stimulus in

discrimination tasks, and may indicate activation of the visual pathway that processes the target-defining feature (O'Donnell et al., 1997). N2 amplitude is reportedly larger for attended stimuli than for non-attended ones (Eimer, 1993; Woldorff et al., 2002).

Cognitive processing during arm movement has been evaluated using P3 component (Shen et al., 2009). This component is the parietocentral positivity with a latency of approximately 300 ms after the target stimulus. P3 is thought to indicate the cognitive processing that occurs after sensory stimulus evaluation, such as context closure (Desmedt and Debecker, 1979; Verleger, 1988) and/or context updating (Donchin and Coles, 1988). Many previous studies suggested that multiple regions including the parietal lobe are involved in P3 generation (Halgren et al., 1995; Knight and Scabini, 1998; Bledowski et al., 2004). Although P3 amplitude is reported to be sensitive to the amount of attentional allocation engaged in the cognitive processing (Polich, 2004), no significant difference in P3 amplitude has been found between attentional focusing and dispersion in the cuing paradigm (Wright et al., 1995).

Motor preparation during arm movement has been evaluated using contingent negative variation (CNV) (Maeda and Fujiwara, 2007; Fujiwara et al., 2009b). CNV is recorded by averaging electroencephalogram (EEG) waveforms between warning (S1) and imperative stimuli (S2) (Walter et al., 1964). This reportedly represents activation of the supplementary motor and premotor areas (Ikeda et al., 1999). The late component of CNV just before S2 is believed to reflect the motor preparation processing (Rohrbaugh

et al., 1976), including postural preparation (Maeda and Fujiwara, 2007; Fujiwara et al., 2009b, 2011) and anticipatory attention directed to S2 (Brunia and van Boxtel, 2001). The late CNV potential was reportedly larger when the cue signal (i.e., S1) indicated a location of the imperative stimulus (Wright et al., 1995).

It was our original proposal that the simultaneous measurement of these ERPs will allow us to systematically investigate the effects of attentional dispersion during unilateral arm abduction on visual-sensory, perceptual, cognitive and motor preparation processing. Furthermore, it will be possible to demonstrate the relationships between these changings in neural processing with attentional dispersion and anticipatory activation of postural muscles. To control attentional dispersion, we increased number of positions where attention will be divided and attentional dispersion will be larger. According to these considerations, we have suggested the following working hypotheses:

(1) With increasing attentional dispersion, P1-N1 and N2 amplitudes and late CNV potentials should decrease.

(2) The activation pattern of postural muscles should change in correlation with the changings of visual-sensory, perceptual, cognitive and motor preparation processing due to attentional dispersion.

2. Methods

2.1 Subjects

Subjects were 6 men and 7 women, with a mean age of 24.0 years (standard deviation (SD) = 4.0), all were right-handed. Their mean height, weight, and foot length were 163.1 cm (SD = 8.5), 56.6 kg (SD = 8.6), and 24.3 cm (SD = 1.6), respectively. All subjects had normal or corrected-to-normal vision. No subject had any history of neurological or orthopedic impairment. Informed consent was obtained in accordance with the Declaration of Helsinki from all subjects following an explanation of the experimental protocols, which were approved by our Institutional Ethics Committee.

2.2 Apparatus

A force platform (FPA34, Electro-design, Japan) was used to measure the positions of the center of foot pressure in the mediolateral and anteroposterior directions (CoPx and CoPy, respectively). To control the initial CoPx and CoPy positions, these were displayed on an oscilloscope (DS6612, IWATSU, Japan) and monitored by an experimenter, as onset time of postural muscles is influenced by CoP position just before arm movement (Fujiwara et al., 2003).

Arm acceleration was recorded using a miniature unidirectional accelerometer (AS-5GB, KYOWA, Japan), which was taped to the dorsal surface of the right wrist so that the axis of sensitivity was along the frontal plane. The position of the right wrist was recorded using a position sensor system (C1373, Hamamatsu Photonics, Japan).

A screen was placed 63 cm in front of subjects and carefully positioned so that the stimuli were delivered straight ahead on the subject's horizontal line of sight. Visual cue signals and imperative stimuli were presented on the screen using the Multi Trigger System (MTS0410, Medical Try Systems, Japan). This system emits analog trigger outputs at the same time as the presentation of cue signals and imperative stimuli.

Silver-silver chloride (Ag-AgCl) cup electrodes (diameter, 8 mm) for recording of the EEGs were affixed to the scalp using a paste at Fz, Cz, Pz, and Oz in accordance with the international 10-20 system, and at OL and OR (located halfway between O1 and T5, and O2 and T6, respectively). All electrodes were referred to the linked earlobe. A ground electrode was placed at Fpz. The horizontal and vertical electrooculogram (h-EOG and v-EOG) were recorded in bipolar fashion from electrodes on the outer canthi of both eyes and electrodes above and below the left eye, respectively.

Surface electrodes (P-00-S, Ambu, Denmark) were used in bipolar derivation to record surface electromyographic (EMG) activity of the following muscles: right middle deltoid (MD) as a focal muscle of abduction of the arm; left erector spinae (ES) at the level of the iliac crest and left gluteus medius (GM) at the point 2.5 cm distal to the iliac crest as postural muscles. These postural muscles are reported consistently to be activated in advance of MD during unilateral arm abduction, in 27 cm stance width (Fujiwara et al., 2009a). Furthermore, with arm movements in this stance, postural stability is high and the individual differences in patterns of postural movement and

postural muscles activation are the smallest. Therefore, we selected these three muscles and the 27 cm stance width for the present study. For each muscle, electrodes were fixed after shaving and cleaning the skin with alcohol. The electrodes were aligned along the long axis of the muscle with an inter-electrode distance of about 3 cm.

Electrode input impedance was reduced to below 5 kΩ. Signals from electrodes were amplified (EEG: ×40000; EOG and EMG: ×4000) and band-pass-filtered (EEG: 0.05-60 Hz; EOG: 0.05-30 Hz; EMG: 5-500 Hz) using an amplifier (Biotop-6R12, NEC-Sanei, Japan). All electrical signals were sent to two separate computers (Dimension E521, Dell Japan, Japan; Bostro 200, Dell Japan, Japan) via A/D converters (ADA16-32/2(CB)F, CONTEC, Japan) with a 1000-Hz sampling rate and 16-bit resolution.

2.3 Visual stimuli and protocol for presentation

Fig. 1 shows the protocol for presentation of visual stimuli. The fixation point was a centrally located square subtending a visual angle of 1×1°. A visual cue signal was presented for 100 ms, and a visual imperative stimulus was presented for 150 ms from 1 s after cue signal onset. Interval between the cue signals was 3 s. Cue signals were triangles presented around the fixation point. The imperative stimuli were checkerboards subtending a visual angle of 6×6°. A vertically or horizontally oriented rectangle on the checkerboard indicated that the stimulus was target (probability of

presentation: 30%) or non-target (70%), respectively.

In order to investigate the effects of increasing of attentional dispersion, two types of position conditions were conducted separately: The imperative stimulus was presented at 9° to the left or right from the fixation point (two-position condition), or to the left, right or center position (three-position condition). In the two-position condition, three types of cue signals were presented: either to the left side or to the right side (probability of presentation: 25% for each cue), and both sides (neutral cue, 50%). In the three-position condition, four types of cue signals were presented: to the left, right or to the center (16.7% for each cue), and all these positions together (neutral cue, 50%). After the presentation of the cue signal, an imperative stimulus was presented in the directed position. If it was a neutral cue, the imperative stimulus was presented unpredictably in one of the two or three positions according to each condition, with the equal probability. As results, subjects covertly focused attention on the position (attentional focusing) or divided attention for two or three positions (attentional dispersion). The order of the cue signals and imperative stimuli and the presentation position of the imperative stimuli were random. Each condition was conducted on separate days, and the order of conditions was randomized for each subject.

2.4 Procedure

All measurements were taken while subjects were standing barefoot with feet 27 cm

apart and parallel on the force platform. Subjects were instructed to maintain stable binocular fixation on the fixation point during the measurements. Initially, CoPx and CoPy positions were measured for 10 s while subjects maintained a quiet standing posture (QSP) with their arms by their sides. A total of five measurements were taken, with a 30-s period of seated rest between them. The means of the five measurements were used as the subject's representative CoPx and CoPy positions during QSP.

After subjects abducted the arm 10 times as a practice, the arm movement trials commenced. The subjects maintained the CoPx and CoPy position within the QSP ± 1 cm for at least 3 s, and then the presentation of visual stimuli began. In response to the target stimuli (vertically oriented rectangle), subjects abducted their right arm in a maximum speed, stopped voluntarily in a horizontal position, and maintained this position for about 1 s before returning to the starting position. Subjects were told to respond to the stimuli as quickly and accurately as possible. One experimental block lasted for 100 s and consisted of 33 stimuli (target or non-target). Subjects had a standing rest period of 30 s between each block and a seated rest period of 3 min between every two blocks. EEG waveforms for target or non-target stimuli were averaged on-line using a computer. The experimental blocks were repeated until the acceptable number of trials was obtained for each attentional state (attentional focusing and dispersion) in both position conditions: 25 trials of right target for N2 and P3 analyses and 60 trials of right non-target for P1 and N1 analyses. Attentional effects on

P1 and N1 components are clearly elicited by the stimuli in the right visual field (Proverbio and Mangun, 1994; Tomita et al., 2007). Furthermore, as the direction of the arm response was to the right side, the neural control for the response to the right stimuli was simpler. Therefore, we mainly focused on the analyses of ERPs for the stimuli presented in right position. The criteria for acceptable trials were the following: (1) no eye movement (< 0.5°, judged from h-EOG using the method reported by Mangun and Hillyard (1991)), (2) no eye blink (voltage at v-EOG not exceeding ±100 μV), and (3) no excessive muscle-related potential (voltage at any EEG electrodes not exceeding ±100 μV) during the time period from 100 ms before the imperative stimulus onset to 800 ms after onset, as well as (4) the CoP positions within QSP ± 1 cm just before arm abduction trials.

2.5 Data analysis

Data analyses were performed using two types of software (EEG: EPLYZER II, Kissei Comtec, Japan; EMG and CoP: BIMUTAS II, Kissei Comtec, Japan). Trials where subjects did not respond correctly to the target or non-target stimulus were defined as error trials, and were eliminated from the analysis. The percentage of error trials among all trials was calculated as the error rate.

2.5.1 ERPs

Each ERP was averaged separately for all combinations of attentional state (focusing or dispersion) and condition (two- or three-position).

We analyzed P1 and N1 components recorded from the OL electrode on the left side, elicited by non-target stimuli only, in order to avoid contamination by ERPs related to motor responses, with reference to a previous study (Di Russo et al., 2003). The averaged epochs for P1 and N1 began 100 ms before non-target stimulus onset and continued for 900 ms. Mean amplitude of the 100 ms pre-stimulus period was defined as the baseline for averaging. Peaks within the following time windows with respect to non-target onset were identified: P1 (80-130 ms) and N1 (140-220 ms). P1 and N1 latencies and P1-N1 amplitude were calculated as the time from stimulus onset to the peaks and the peak-to-peak amplitude from P1 peak to N1 peak, respectively.

N2 and P3 components are elicited by target stimuli in a discrimination task (Donchin and Coles, 1988; Goodin and Aminoff, 1998). For visual target stimuli, N2 and P3 have maximum amplitude in the occipital and parietal areas, respectively (Simson et al., 1977). Therefore, we analyzed N2 and P3 components recorded from Oz and Pz, elicited by target stimuli. The averaged epochs for N2 and P3 began 100 ms before target stimulus onset and continued for 900 ms. Mean amplitude of the 100 ms pre-stimulus period was defined as the baseline for averaging. The averaged waveform was smoothed using a 30-Hz low-pass filter. The largest negative peak between 200 ms and 350 ms and the largest positive peak between 250 ms and 600 ms after the

presentation of the target stimulus were defined as N2 and P3, respectively. Latencies and amplitudes of the peaks were calculated as the time from stimulus onset to the peaks and the voltage difference between the peaks and baseline.

Since late CNV was consistently maximal at Cz, waveforms recorded from Cz were analyzed. The mean amplitude for the 100-ms period before the cue signal was defined as the baseline. Waveforms of EEGs from 100 ms before the onset of cue signal to the onset of the right imperative (both target and non-target) stimulus were then averaged off-line. Trials with large eye movements, eye blinks or excessive muscle-related potentials during this time epoch were excluded. The lowest acceptable trial number for CNV averaging was 12 (Tecce, 1972). The averaged waveform was smoothed using a 4-Hz low-pass filter. Since CNV potentials at the S2 presentation point are reportedly equal in the cases of inter-stimulus intervals (ISIs) between 1 and 3 s (Ruchkin et al., 1986), the late CNV component could be measured with a 1-s ISI (Eimer, 1993; Wright et al., 1995). In order to analyze changing patterns in late CNV, mean potential was calculated for every 100 ms from 500 ms to 0 ms before the onset of the imperative stimulus.

2.5.2 EMGs

In order to exclude electrocardiographic and movement artifacts, all EMGs were 40-Hz high-pass-filtered using the seventh-order Butterworth method and then

full-wave-rectified. Fig. 2 shows representative EMG data. The time course of EMG bursts of the focal and postural muscles in each trial was analyzed as described below with reference to a previous study (Shen et al., 2009). The burst onset of MD was identified by visual inspection of the EMG trace on a computer screen, since background activity of MD before burst onset was extremely small. The time difference between the target stimulus onset and burst onset of MD was defined as the MD reaction time. The back ground activity of postural muscle was defined as the EMG activity over the period from -300 to -150 ms with respect to the burst onset of MD. The burst activation of each postural muscle was identified when its onset was within -150 ms to +100 ms with respect to burst onset of MD, and the envelop line of the burst activity deviated more than the mean + 2SDs of the back ground activity for at least 50 ms. The burst onset was defined as the time point where the above deviation began in the EMG wave included in the envelope line. The onset time of postural muscles was defined as time difference between burst onset of postural muscles and MD, and presented as a negative value when burst onset of postural muscles preceded MD.

To analyze the activity level of each postural muscle, EMG waveforms in the period from -100 to +200 ms with respect to burst onset of the muscle was then averaged for each attentional state in both position conditions. The averaged EMG waveforms were smoothed using a 40-Hz low-pass filter. The EMG peak amplitude and its latency with respect to burst onset were measured.

2.5.3 CoP

Mean CoPx and CoPy positions were calculated for the periods from -300 ms to -150 ms with respect to the burst onset of MD (before arm movement) and from 0 to +150 ms with respect to the endpoint of arm movement. The endpoint was defined as the end of the second burst activity of MD, where the burst envelope line first deviated lower than the mean + 2SDs for 500 ms just before arm lowering, with reference to the curves of the wrist position and arm acceleration. The differences between these mean positions were considered as CoPx and CoPy displacements, respectively.

2.6 Statistical analysis

Tables 1, 2 and 3 show the list of parameters that were compared for the statistical analysis. All data were analyzed using Shapiro-Wilk tests for normality. Normally distributed data (N1 latency, P1-N1 and P3 amplitudes, CNV mean potentials, MD reaction time, onset time of ES, peak amplitude of ES and peak latency of GM) were analyzed using parametric tests, while skewed data (P1, N2 and P3 latencies, N2 amplitude, error rate, onset time of GM, peak amplitude of GM, peak latency of ES, mean CoPx and CoPy positions before arm movement, and CoPx and CoPy displacements) were analyzed using non-parametric tests. The data with attentional focusing for each parameter showed no significant differences between two- and

three-position conditions. These position conditions were conducted on separate days. Therefore, we regarded the data with attentional focusing as the standards and the effects of attentional dispersion were evaluated as follows. First, the paired *t*-test or Wilcoxon test was used to compare data for each parameter between attentional focusing and dispersion in each position condition. Next, differences for each parameter between attentional focusing and dispersion were calculated for each position condition. These differences were compared between position conditions by the paired *t*-test or Wilcoxon test in order to investigate the effects of attentional dispersion to two and three positions. Pearson correlation or Spearman's correlation was used to evaluate the magnitude of correlation between the differences in each parameter. Alpha level was set at $p < 0.05$. All statistical analyses were performed using SPSS 14.0J (SPSS Japan, Japan).

3. Results

P1-N1 and N2 amplitudes were significantly smaller with attentional dispersion than with attentional focusing in both position conditions ($p < 0.05$) (Fig. 3A, B, Table 1). No significant differences were found in the decreases of P1-N1 amplitude with attentional dispersion between two- and three-position conditions. The decrease in N2 amplitude with attentional dispersion tended to be larger in three-position condition than two-position condition ($p = 0.1$). Only in the three-position condition, N2 latency was

significantly longer with attentional dispersion than with attentional focusing ($p < 0.01$) (Fig. 3B, Table 1). In P1 and N1 latencies and P3 amplitude and latency, no significant differences were observed between attentional dispersion and focusing in either position condition (Fig. 3A, C, Table 1).

Only in the three-position condition, CNV mean potentials of the two periods from -300 to -100 ms were significantly smaller with attentional dispersion than with attentional focusing ($p < 0.05$) (Fig. 4, Table 2). In the mean potentials of the three periods from -500 to -300 ms and from -100 to 0 ms, no significant differences were recognized between attentional dispersion and focusing in either position condition.

No significant differences in error rates were found between attentional dispersion and focusing in either position condition. MD reaction times were significantly longer with attentional dispersion than with attentional focusing in both position conditions ($p < 0.01$) (Table 3). The increase in reaction time with attentional dispersion was significantly larger in three-position condition than two-position condition ($p < 0.01$). Only in the three-position condition, the onset time of GM was significantly later with attentional dispersion than with attentional focusing ($p < 0.05$) (Table 3). No significant differences in onset time of ES, peak amplitude and latency of GM and ES, mean CoPx and CoPy positions before arm movement, and CoPx and CoPy displacements were found between attentional dispersion and focusing in either position condition (Table 3).

As regards correlations between ERPs, a significant correlation was found only in

changings with attentional dispersion between N2 latency and CNV mean potential in the period from -300 to -200 ms (r = -0.54, p < 0.01). As regards correlations between ERPs and EMGs, changing in onset time of GM was significantly correlated with changings in CNV mean potentials from -300 to -100 ms (CNV potential from -300 to -200 ms: r = -0.51; CNV potential from -200 to -100 ms: r = -0.63, p < 0.01). No significant correlations were found in changings of other parameters between ERPs and EMGs.

4. Discussion

Generally to perform reaction task to the sensory stimulation, neural processing includes the sensory, perceptual, and cognitive processing of stimuli, and the motor processing of preparation, selection, and execution of the responses. Attetional allocation changes according to the significance of the processing (Lavie, 1995). In this research, error rates did not differ significantly between attentional dispersion and focusing, while MD reaction time changed according to attentional dispersion, suggesting that the subjects would place a priority to respond accurately rather than rapidly. Therefore, we consider that attention will be directed to the essential processing for the accurate response, and the effects of attentional dispersion would be clearly presented on that processing. With these viewpoints, we will discuss the hypotheses proposed in Introduction.

P1-N1 component is considered to reflect the activities of visual-sensory processing in extrastriate visual cortex (Hopfinger et al., 2001; Di Russo et al., 2003). P1-N1 amplitudes were significantly smaller with attentional dispersion than with attentional focusing. No significant effect of the increasing of attentional dispersion was found in P1-N1 amplitude. These results suggest that the activation in exrastriate visual cortex related to visual-sensory processing would decrease with attentional dispersion. This processing would be relatively easy, and therefore it would not be necessary to allocate more attention to this processing.

N2 component is reported to reflect the discrimination of the task-relevant visual features (Simson et al., 1977; O'Donnell et al., 1997). N2 amplitudes were significantly smaller with attentional dispersion than with attentional focusing. With increasing attentional dispersion, N2 amplitude tended to decrease further and N2 latency became significantly longer. The reason why the effects of attentional dispersion were clearly reflected in N2 would be that discrimination of the target stimulus after visual-sensory processing was important for an accurate response, and therefore, more attention was allocated for stimulus discrimination.

P3 component had no effects of attentional dispersion. P3 amplitude is reported to be sensitive to the amount of attentional allocation engaged in the context closure and updating of cognitive processing and its latency reflects processing time (Polich, 2004). To identify P3, an oddball task, in which subjects execute a reaction movement to

infrequently appearing target stimuli, has been used widely. The task using in the present study is considered to be an oddball-like task, where the context updating would mean switching cognitive processing from the target stimulus to the next cue signal. P3 amplitude and latency are reportedly changed by the difficulty of discriminating stimuli (Polich, 1987; Maguire et al., 2009). Since the cue signals in the present study were simple triangles at the center of the display, with sufficient time for detection (100 ms) (Hartmann et al., 1979; Luczak and Sobolewski, 2005) and predictable timing of reappearance (2 s after the imperative stimulus) (Sato et al., 1973), it would be easy to discriminate each cue signal. Therefore, the context closure or updating processing would not be affected by attentional dispersion.

CNV is reportedly related to motor preparation (Rohrbaugh et al., 1976) including postural preparation (Maeda and Fujiwara, 2007; Fujiwara et al., 2009b, 2011) and anticipatory attention directed to the imperative stimuli (Brunia and van Boxtel, 2001). It reportedly represents activation in the frontal lobe such as the supplementary motor and premotor areas (Ikeda et al., 1999). In our results, CNV mean potentials decreased in the periods from -300 to -100 ms before the onset of imperative stimulus with attentional dispersion to three positions, but not two positions. These results suggest that in case of the three-position, the start of attentional allocation to the motor preparation or anticipation of the imperative stimuli would be delayed due to the larger attentional dispersion. With attentional dispersion to two positions, it would not be necessary to

allocate all attention to visual-sensory processing or stimulus discrimination, therefore, attention could be allocated sufficiently to motor preparation or anticipation.

A significant correlation was found between the CNV time course and N2 latency. It is thought that the neural circuit related to the organizing system would consist of the frontal and parietal cortices responsible for attentional control (Corbetta and Shulman, 2002). Hagmann et al. (2008) has demonstrated that frontal and posterior areas are reciprocally connected. Functional connections between frontal areas including the premotor area and dorsolateral prefrontal cortex related to CNV generation and posterior perceptual areas including V1, V2 and V4 related to N2 generation are needed to perform target detection (discrimination) (McIntosh et al., 1994; Smith and Jonides, 1999). From all described above, we consider that with increasing attentional dispersion, processing of anticipatory attention directed to the imperative stimuli would be late, causing a delay in stimulus discrimination.

Next, the motor output aspects will be discussed. The reaction time of MD as a focal muscle of arm abduction, which reflects processing time from stimulus input to motor output, became longer with attentional dispersion. This is consistent with the findings of previous studies (Posner et al., 1980; Tomita and Fujiwara, 2008). The difference between attentional dispersion to three positions and attentional focusing was 35 ms in MD reaction time, and 23 ms in N2 latency. This suggests that with attentional dispersion to three positions, processing related to motor output after stimulus

discrimination such as motor selection, starting a motor program or output of muscle activation would be late for 12 ms. No significant correlations were found in changings between MD reaction time and each ERP component. Therefore, the degree of decrease in activation with attentional dispersion in each processing would be different among individuals.

For activation of postural muscles, onset time of GM with respect to the burst onset of MD was late by 2.7 ms with attentional dispersion to three positions, and this delay is extremely smaller than MD reaction time changing. No attentional effects were found on activity levels of postural muscles. Previously it has been suggested that the programs of arm movement and postural control are synergistic rather than completely independent, however this synergy is reported to be not fixed but has plasticity; i.e., the postural set changes according to the internal and external conditions (Cordo and Nashner, 1982; Massion, 1992; De Wolf et al., 1998). Our results suggest that the program of focal and postural muscles would be synergistic and the start of the program would be delayed with attentional dispersion. However, activation timing of the postural muscle would be slightly affected by attentional dispersion. The changing in onset time of GM with attentional dispersion was correlated with the changing of CNV, but not with N2 latency, suggesting the late onset of postural muscle activation would be correlated with the delay of motor preparation or anticipatory attention directed to the imperative stimuli. In addition, because the delay of GM onset time was small, CoP

displacement was not affected by attentional dispersion.

In conclusion, effects of attentional dispersion on brain activation were different in each stage of sensory-motor processing. Activation in brain areas related to visual-sensory processing (P1-N1) and stimulus discrimination (N2) decreased with attentional dispersion, with a remarkable decrease in latter one. With increasing attentional dispersion, the delay in motor preparation or anticipatory attention to imperative stimuli (CNV) was related to the delay in stimulus discrimination and onset time of postural muscle activation. The present study helps to systematically evaluate the effects of attentional dispersion on visual-sensory, perceptual, cognitive and motor preparation processing, and postural control during voluntary arm movement. The results of this study can be effectively applied to elderly subjects or be used in clinical investigations for patients with sensory-motor impairments.

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Legends

Fig. 1. Visual stimuli and protocol for presentation. An imperative stimulus was presented at 9° to the left or right of the fixation point in the two-position condition and to the left, right or center position in the three-position condition. Probabilities of presentation were 30% for target stimulus and 70% for non-target stimulus. The order of the cue signals and imperative stimuli and the presentation position of the imperative stimuli were random.

Fig. 2. Representative electromyographic data during right arm abduction. MD: right middle deltoid, ES: left electro spinae, GM: left gluteus medius. The solid and dashed lines indicate the target stimulus onset and the burst onset of MD, respectively. Arrows indicate onset of burst activation of postural muscles.

Fig. 3. Grand average waveforms of P1 and N1 for right non-target stimuli (A), and N2 (B) and P3 (C) for right target stimuli.

Fig. 4. Grand average waveforms of contingent negative variation (CNV) between cue signal and right imperative (target and non-target) stimuli.

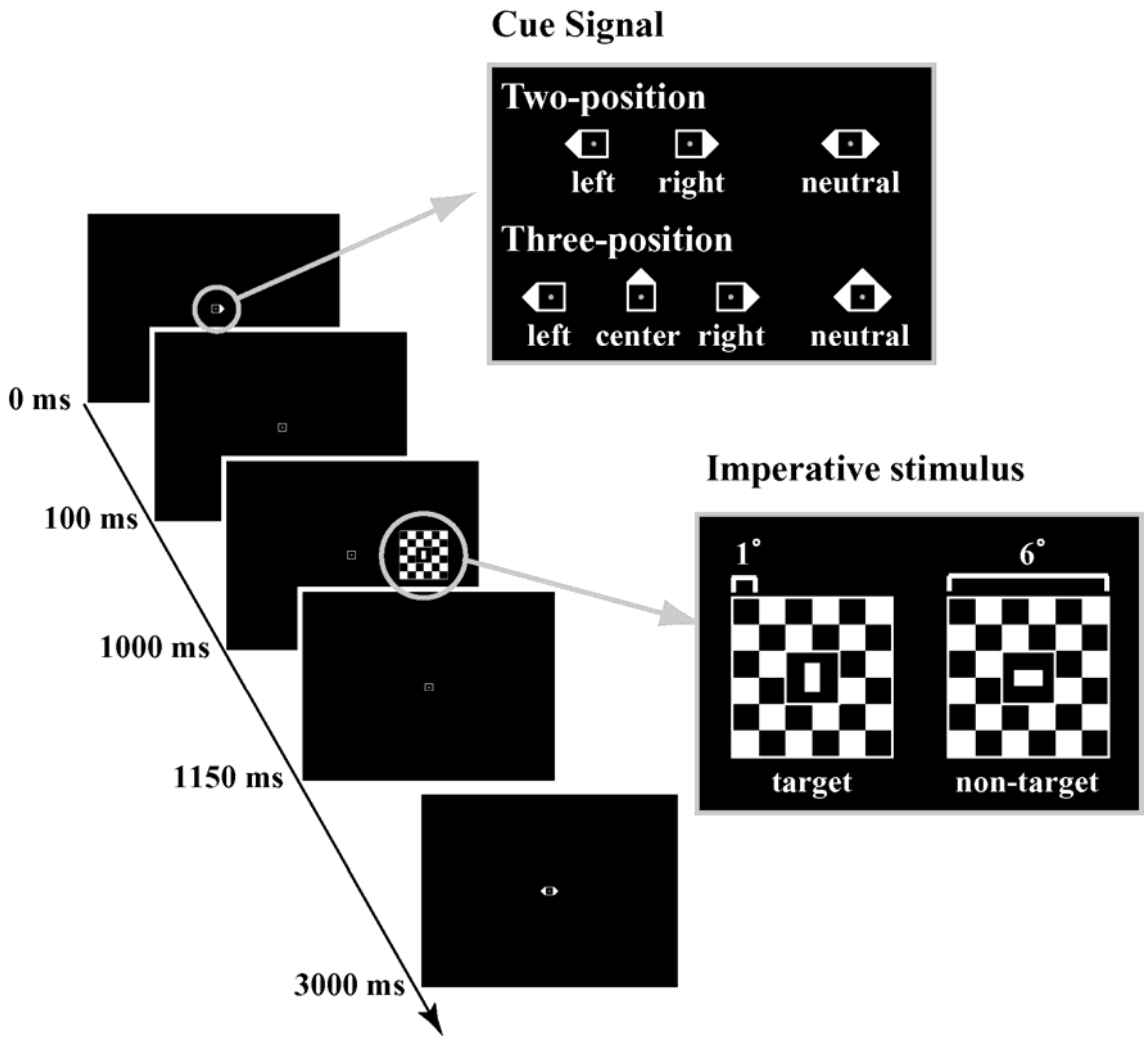
Cue Signal
Two-position
left
right
neutral
Three-position
left
center
right
neutral
0 ms
100 ms
1000 ms
1150 ms
3000 ms
Imperative stimulus
1°
6°
target
non-target

Fig. 1

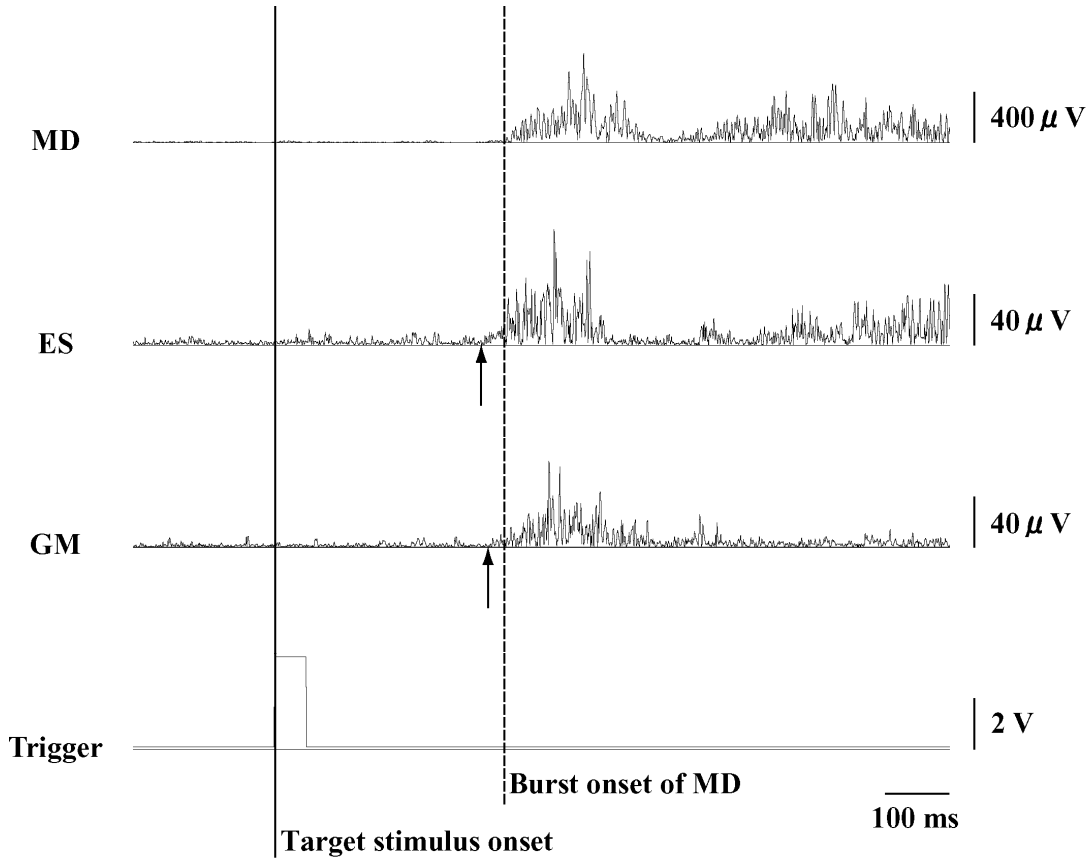
MD
ES
GM
Trigger
400 μ V
40 μ V
40 μ V
2 V
Burst onset of MD
Target stimulus onset
100 ms

Fig. 2

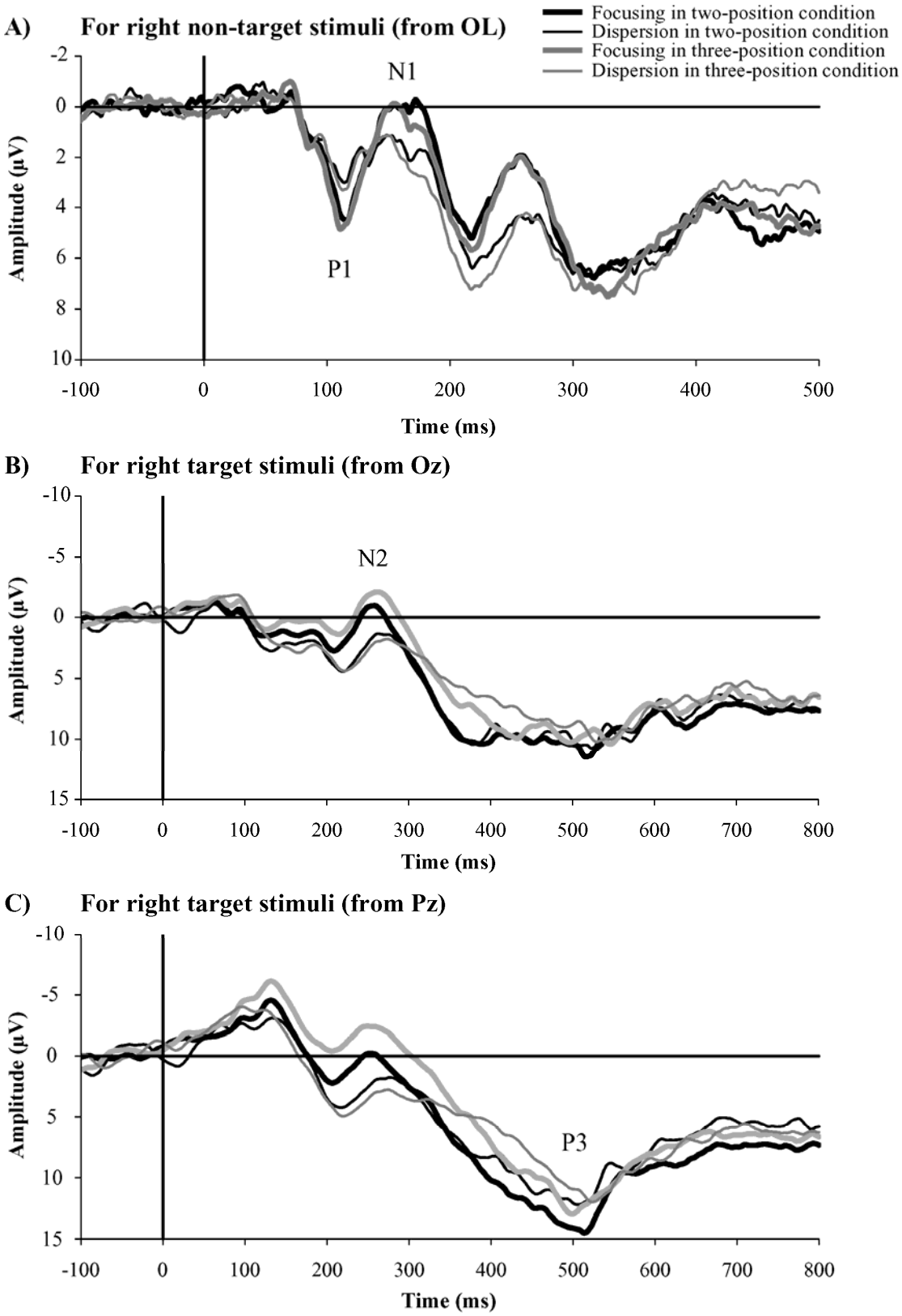
A) For right non-target stimuli (from OL)
Focusing in two-position condition
Dispersion in two-position condition
Focusing in three-position condition
Dispersion in three-position condition
N1
P1
Amplitude (µV)
Time (ms)
B) For right target stimuli (from Oz)
N2
Amplitude (µV)
Time (ms)
C) For right target stimuli (from Pz)
P3
Amplitude (µV)
Time (ms)

Fig. 3

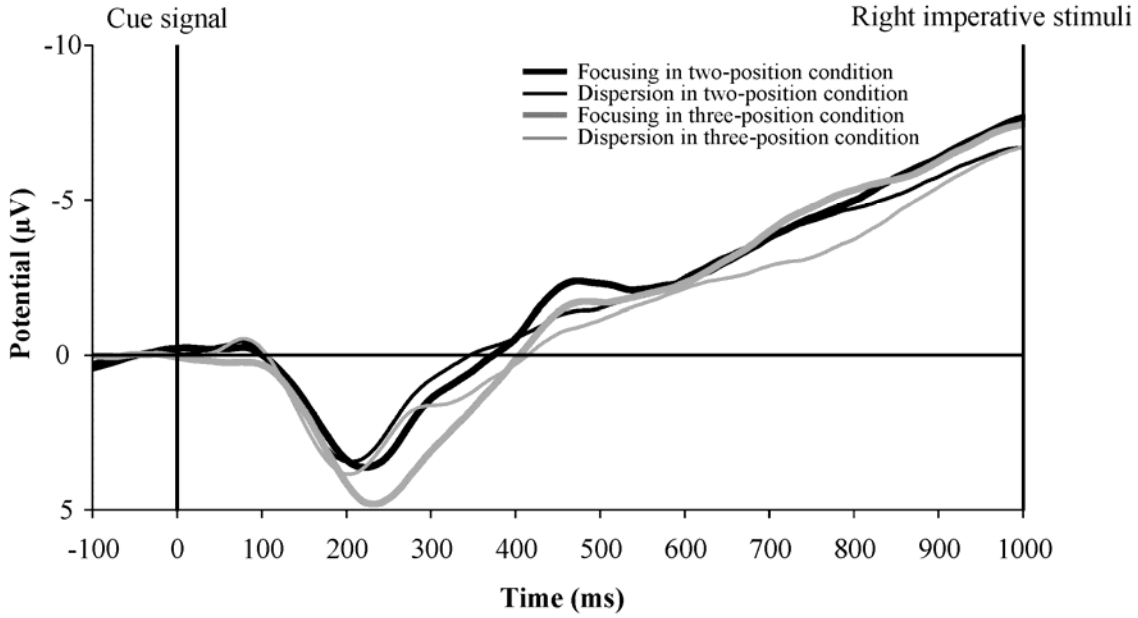
Cue signal
Right imperative stimuli
Focusing in two-position condition
Dispersion in two-position condition
Focusing in three-position condition
Dispersion in three-position condition
Potential (μV)
-10
-5
0
5
-100
0
100
200
300
400
500
600
700
800
900
1000
Time (ms)

Fig.4

Table 1

Means and standard deviations of P1, N1, N2 and P3 components. t : tested with paired t-test; T: tested with Wilcoxon test.

Dependent variables		Two-position condition	Statistical values	Significance	Three-position condition	Statistical values	Significance		Difference between attentional focusing and dispersion	Statistical values	Significance
P1 latency (ms)	focusing	115.2 ± 8.3	$T = 44$	N.S	115.2 ± 9.6	$T = 31$	N.S	Two-position	-0.2 ± 8.8	$T = 29$	N.S
	dispersion	115.0 ± 14.0			120.1 ± 22.3			Three-position	4.8 ± 14.8		
N1 latency (ms)	focusing	164.5 ± 17.1	$t_{12} = 0.2$	N.S	163.5 ± 19.1	$t_{12} = 0.0$	N.S	Two-position	0.5 ± 8.2	$t_{12} = 0.1$	N.S
	dispersion	165.0 ± 21.6			163.5 ± 23.1			Three-position	0.0 ± 12.6		
P1-N1 amplitude (μV)	focusing	8.0 ± 3.6	$t_{12} = 3.2$	$p < 0.01$	8.2 ± 4.0	$t_{12} = 4.0$	$p < 0.01$	Two-position	-1.8 ± 2.1	$t_{12} = 0.7$	N.S
	dispersion	6.1 ± 3.6			5.9 ± 3.7			Three-position	-2.2 ± 2.0		
N2 latency (ms)	focusing	263.2 ± 20.7	$T = 33$	N.S	262.8 ± 14.3	$T = 0$	$p < 0.01$	Two-position	8.9 ± 26.2	$T = 24$	N.S
	dispersion	272.2 ± 21.9			285.8 ± 23.9			Three-position	23.1 ± 17.7		
N2 amplitude (μV)	focusing	1.5 ± 4.1	$T = 13$	$p < 0.05$	3.0 ± 4.9	$T = 7$	$p < 0.01$	Two-position	-1.8 ± 2.5	$T = 21$	$p = 0.1$
	dispersion	-0.4 ± 4.3			-0.5 ± 4.9			Three-position	-3.5 ± 3.5		
P3 latency (ms)	focusing	453.5 ± 68.8	$T = 32.5$	N.S	444.8 ± 70.5	$T = 25$	N.S	Two-position	7.2 ± 26.2	$T = 41$	N.S
	dispersion	460.7 ± 73.0			462.2 ± 77.8			Three-position	17.4 ± 41.3		
P3 amplitude (μV)	focusing	15.2 ± 7.4	$t_{12} = 2.1$	N.S	13.3 ± 9.2	$t_{12} = 0.5$	N.S	Two-position	-1.9 ± 3.2	$t_{12} = 0.9$	N.S
	dispersion	13.3 ± 7.2			12.6 ± 9.0			Three-position	-0.7 ± 4.5		

Table 2
Means and standard deviations of contingent negative variation (CNV) mean potentials for every 100-ms period from 500 to 0 ms before onset of right imperative (target and non-target) stimuli.

Dependent variables		Two-position condition	Statistical values	Significance	Three-position condition	Statistical values	Significance		Difference between attentional focusing and dispersion	Statistical values	Significance
CNV -500 to -400 ms (μV)	focusing	2.2 ± 2.2	$t_{12} = 0.6$	N.S	1.9 ± 2.5	$t_{12} = 0.8$	N.S	Two-position	-0.3 ± 1.5	$t_{12} = 0.1$	N.S
	dispersion	1.9 ± 2.0			1.6 ± 2.8			Three-position	-0.3 ± 1.4		
CNV -400 to -300 ms (μV)	focusing	3.0 ± 2.1	$t_{12} = 0.4$	N.S	3.1 ± 2.5	$t_{12} = 1.3$	N.S	Two-position	0.2 ± 1.7	$t_{12} = 1.8$	N.S
	dispersion	3.2 ± 2.1			2.5 ± 2.5			Three-position	-0.6 ± 1.6		
CNV -300 to -200 ms (μV)	focusing	4.4 ± 1.9	$t_{12} = 0.2$	N.S	4.7 ± 2.3	$t_{12} = 3.5$	$p < 0.01$	Two-position	-0.1 ± 1.3	$t_{12} = 2.7$	$p < 0.05$
	dispersion	4.3 ± 2.0			3.2 ± 2.1			Three-position	-1.5 ± 1.6		
CNV -200 to -100 ms (μV)	focusing	5.7 ± 2.4	$t_{12} = 1.1$	N.S	5.7 ± 2.4	$t_{12} = 2.8$	$p < 0.05$	Two-position	-0.5 ± 1.6	$t_{12} = 1.4$	N.S
	dispersion	5.2 ± 2.8			4.6 ± 2.5			Three-position	-1.2 ± 1.5		
CNV -100 to 0 ms (μV)	focusing	7.1 ± 3.1	$t_{12} = 1.7$	N.S	6.9 ± 2.9	$t_{12} = 1.4$	N.S	Two-position	-0.7 ± 1.6	$t_{12} = 0.1$	N.S
	dispersion	6.3 ± 3.4			6.2 ± 3.1			Three-position	-0.8 ± 2.0		

Table 3

Means and standard deviations of error rate, reaction time of right middle deltoid (MD), onset time, peak amplitude and peak latency of postural muscles, mean CoPx and CoPy positions before arm movement and CoPx and CoPy displacements (ES: left electro spinae, GM: left gluteus medius). t : tested with paired t-test; T: tested with Wilcoxon test.

Dependent variables		Two-position condition	Statistical values	Significance	Three-position condition	Statistical values	Significance		Difference between attentional focusing and dispersion	Statistical values	Significance
Error rate (%)	focusing	4.0 ± 5.4	$T = 38$	N.S	6.6 ± 8.1	$T = 40$	N.S	Two-position	0.3 ± 2.0	$T = 34$	N.S
	dispersion	4.3 ± 4.3			6.1 ± 5.7			Three-position	-0.5 ± 3.5		
MD reaction time (ms)	focusing	339.0 ± 47.4	$t_{12} = 4.3$	$p < 0.01$	348.8 ± 41.2	$t_{12} = 8.6$	$p < 0.001$	Two-position	16.1 ± 13.4	$t_{12} = 4.4$	$p < 0.01$
	dispersion	355.2 ± 45.0			383.8 ± 44.3			Three-position	35.0 ± 14.7		
Onset time of ES (ms)	focusing	-23.8 ± 21.3	$t_{12} = 0.7$	N.S	-22.9 ± 16.1	$t_{12} = 0.5$	N.S	Two-position	0.8 ± 3.7	$t_{12} = 0.1$	N.S
	dispersion	-23.0 ± 20.0			-22.3 ± 18.2			Three-position	0.6 ± 4.8		
Onset time of GM (ms)	focusing	-13.6 ± 15.4	$T = 29$	N.S	-15.2 ± 16.1	$T = 16$	$p < 0.05$	Two-position	1.3 ± 3.5	$T = 31$	N.S
	dispersion	-12.2 ± 16.4			-12.5 ± 14.4			Three-position	2.6 ± 4.0		
Peak amplitude of ES (μV)	focusing	27.0 ± 13.1	$t_{12} = 1.1$	N.S	24.5 ± 9.5	$t_{12} = 0.3$	N.S	Two-position	-0.8 ± 2.7	$t_{12} = 0.5$	N.S
	dispersion	26.2 ± 12.0			24.3 ± 9.2			Three-position	-0.2 ± 3.2		
Peak amplitude of GM (μV)	focusing	20.2 ± 15.8	$T = 30$	N.S	18.9 ± 12.8	$T = 40$	N.S	Two-position	0.7 ± 1.9	$T = 37$	N.S
	dispersion	20.9 ± 16.9			19.6 ± 13.7			Three-position	0.7 ± 2.8		
Peak latency of ES (ms)	focusing	86.4 ± 24.9	$T = 30.5$	N.S	86.8 ± 27.7	$T = 22$	N.S	Two-position	5.8 ± 17.1	$T = 41$	N.S
	dispersion	92.2 ± 25.3			92.6 ± 20.6			Three-position	5.8 ± 18.0		
Peak latency of GM (ms)	focusing	105.3 ± 27.2	$t_{12} = 0.9$	N.S	107.1 ± 34.4	$t_{12} = 1.3$	N.S	Two-position	-3.4 ± 13.8	$t_{12} = 0.5$	N.S
	dispersion	101.9 ± 25.1			100.2 ± 27.5			Three-position	-6.9 ± 18.8		
Mean CoPx position before arm movement (cm)	focusing	0.12 ± 0.61	$T = 28$	N.S	0.04 ± 0.67	$T = 28$	N.S	Two-position	-0.05 ± 0.13	$T = 32$	N.S
	dispersion	0.06 ± 0.59			0.04 ± 0.65			Three-position	0.00 ± 0.10		
Mean CoPy position before arm movement (cm)	focusing	10.55 ± 2.03	$T = 35$	N.S	10.67 ± 2.22	$T = 43$	N.S	Two-position	-0.04 ± 0.13	$T = 39$	N.S
	dispersion	10.51 ± 2.02			10.66 ± 2.19			Three-position	-0.02 ± 0.19		
CoPx displacement (cm)	focusing	0.78 ± 0.44	$T = 41$	N.S	0.78 ± 0.41	$T = 38$	N.S	Two-position	-0.01 ± 0.11	$T = 29$	N.S
	dispersion	0.77 ± 0.49			0.80 ± 0.46			Three-position	0.03 ± 0.14		
CoPy displacement (cm)	focusing	0.19 ± 0.11	$T = 45$	N.S	0.19 ± 0.21	$T = 35$	N.S	Two-position	0.00 ± 0.07	$T = 38$	N.S
	dispersion	0.19 ± 0.10			0.20 ± 0.20			Three-position	0.01 ± 0.07		