

An EEG/MEG study of anticipatory postural adjustment in a bimanual load-lifting task

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Abstract

Anticipatory postural adjustments (APA) are necessary for counteracting destabilizing forces induced by prime movements. The present study examined APA in a bimanual load-lifting task using simultaneous electroencephalography (EEG) and magnetoencephalography (MEG). A 1-kg weight placed on the left arm was either lifted by an experimenter (imposed condition) or by the subject (voluntary condition). In the imposed condition, inhibition of electromyographic (EMG) activity in the load-bearing muscle (biceps brachii, BB) only occurred after the onset of unloading and was accompanied by event-related desynchronisation (ERD) of cortical activity in the beta frequency band (15-30 Hz). This ERD was localized to the primary sensory cortex using beamforming analyses. In the voluntary condition, EMG inhibition and beta ERD occurred prior to the onset of lifting and beta ERD was localized to the primary motor cortex. This anticipatory cortical and muscle activity ties in well with the feedforward property of APA. This paradigm will be used to study problems of neuro-motor coordination in autism spectrum disorders (ASD).

Keywords: Event-related desynchronisation; Anticipatory postural adjustment; Bimanual coordination; Autism spectrum disorder

Introduction

Movement of body segments disturbs postural equilibrium. In order to minimize this disturbance, APAs are made to preemptively counteract the reactive forces brought about by volitional movements. For example, Belenkiy, Gurfinkel, & Paltsev (1967) showed that during arm-raising in standing, anticipatory EMG activity in a leg muscle preceded the onset of EMG activity in the arm muscle thereby compensating for the postural disturbance induced by the arm movement. This predictive response in the lower limbs is also present in functional arm-raising movements such as reach-to-grasp.

APAs during goal-directed movements have also been reported in locomotion (Taga, 1998; McFadyen, Malouin, & Dumas, 2001) and catching (Lacquaniti & Maioli, 1989; Morton, Lang, & Bastian, 2001). However, reliable quantification of APAs within such complex movement patterns is complicated by the involvement of multiple muscles, often located in close proximity (Hirschfeld & Forssberg, 1991). This inherent complexity makes the recording of EMG activity from specific muscles less reliable due to electrical cross-talk between adjacent muscles (Kilner, Baker, & Lemon, 2002). The bimanual load-lifting task developed by Hugon, Massion, & Wiesendanger (1982) avoids these problems. In this paradigm, the subject supports a weight placed on the left arm and maintains the elbow joint at a desired angle before unloading it with the right hand. The clear-cut compartmentalization of the flexion and extension musculature in the arm means that EMG from the prime flexor (biceps brachii) and the prime extensor (triceps brachii) of the elbow can be reliably isolated. Furthermore, the muscles involved in the supporting and lifting components are lateralized in a task-specific manner meaning that load-lifting is a good model for studying APA both in normal and clinical populations.

ASD including autism and Asperger syndrome are characterized by pervasive abnormalities of social interactions, communication, and stereotyped behaviours (American Psychiatric Association, 1994; World Health Organization, 1992). 'Clumsiness' or poverty of motor coordination, albeit not a diagnostic criterion for the disorders, is a common feature among autistic people (Gillberg & Coleman, 1992; Bauman, 1992). The motor-related deficit may be among the earliest symptoms of the disorders (Kanner, 1943), but very little is known about the neurological bases of the problem. Research in the area has been to quantify the deficits in motor function (Ghaziuddin

& Butler, 1998; Pan, Tsai, & Chu, 2009; Nikolic & Ilic-Stosovic, In Press) and qualify aspects of motor-related processes that might account for sub-optimal motor performance (Martineau et al., 2004; Rinehart, Bradshaw, Brereton, & Tonge, 2001; Schmitz et al., 2003; Haswell, Izawa, Dowell, Mostofsky, & Shadmehr, 2009). Findings from these studies converged on a central deficit of neuro-motor coordination, a case of neural and muscular dysfunction. To this end, the bimanual load-lifting task involving high-level limb coordination might be a good model for studying motor-related problems in ASD.

The neural mechanisms of APA involve a distributed network of cortical and subcortical structures. Studies of patients with primary and supplementary motor area lesions showed that APA was either degraded or abolished in a bimanual coordination task (Viallet, Massion, Massarino, & Khalil, 1992). Impaired APA was also reported in primates with motor-related cortical ablation (Brinkman, 1981, 1984). Apart from cortical lesions, pathology of subcortical structures including basal ganglia also affects postural adjustment in goal-directed movements. In two studies of patients with Parkinson's disease, the APA was partially preserved in half the patient group and abolished in the rest (Traub, Rothwell, & Marsden, 1980; Viallet, Massion, Massarino, & Khalil, 1987). In another study of patients with cerebellar pathology, APA was found to be largely intact, albeit with an onset significantly earlier relative to normal controls (Diedrichsen, Verstynen, Lehman, & Ivry, 2005).

Martineau, Schmitz, Assaiante, Blanc, & Barthelemy, (2004) investigated APA in autistic children during bimanual load-lifting using EEG. Results from that study showed ERD in electrodes overlying right primary motor cortex preceding APA when the weight was voluntarily lifted with the right hand. The authors speculated that ERD in contralateral motor cortex was related to the inhibition of EMG activity in the load-bearing muscle that minimized arm deflection (see Schmitz, Martineau, Barthelemy, & Assaiante, 2003). However, precautions should be taken when interpreting the results. First, the number of subjects (n=7) and trials per condition (n=10) were small in the study. Previous studies that investigated cortical dynamics of self-paced movements typically involved 60-80 trials (Pfurtscheller & Aranibar, 1979; Cheyne, Bakhtazad, & Gaetz, 2006). Second, inter-electrode spacing on EEG caps coupled with smearing of potentials by poor volume-conducting medium and the skull mean that electrode topographies do not necessarily reflect cortical sources (Nunez & Westdorp, 1994). The current study addresses these issues. We measured brain activity with simultaneous EEG and MEG and co-registered these data with structural magnetic resonance imaging (MRI) scans to improve source model solutions. Here we report representative results from one subject.

Methods

Subject and general procedures

One right-handed subject (female, age = 24.8 years) participated in the pilot study that was carried out at KIT-Macquarie Brain Research Laboratory (Macquarie University). The subject gave informed consent to the procedures, which were approved by the university's ethics committee.

The bimanual load-lifting task was performed while lying on a bed in a supine position in a magnetically shielded room (MSR). The subject's left arm was positioned adjacent to the body so that elbow flexion could be performed comfortably. In order to minimize movements at the left shoulder joint, which potentially result in movement artifacts, the arm was strapped with a Velcro tape to a cushion placed just above the elbow joint. The left hand rested on a cushion placed near the wrist joint such that the forearm was inclined about 20 degrees from the horizontal plane. The subject's right arm rested on the abdomen. The experimenters communicated with the subject via audio earphones. Visual displays were projected via a mirror onto a screen, which was directly in the subject's line of sight.

At the start of each trial, the subject was instructed to raise the left arm such that it was no longer resting on the cushion. A 1-kg weight was then placed over a photo-detector that was secured to a 5 x 5 cm platform on the left arm. During a voluntary trial, as indicated by a visual cue on the screen, the subject lifted the weight with her right hand voluntarily. During an imposed trial, an experimenter in the MSR lifted the weight unexpectedly (see Figure 1; not the actual subject). The photo-detector triggered a registration pulse that was sent to the data acquisition computer at the onset of unloading in both conditions. Inter-trial intervals were in the order of 8 to 12 sec depending on the time of unloading after the visual cue. A total of 160 trials (80 voluntary; 80 imposed) were performed in a pseudo-random order. After 40 trials, there was a rest interval of approximately 20 seconds.



Figure 1: Subject's position in MSR during bimanual load-lifting task. During a voluntary trial, the subject lifted the 1-kg weight using the right hand (*left*). During an imposed trial, an experimenter lifted the weight unexpectedly (*right*).

Data acquisition

High-resolution anatomical images were obtained using a 3.0 Tesla MRI scanner (Philips) at Saint Vincent's Hospital, NSW, Australia.

Brain activity was recorded with simultaneous whole-head MEG (160-channel coaxial first-order gradiometer system, Kanazawa Institute of Technology, Kanazawa, Japan) and MEG-compatible EEG (Brain Products; 64-channel Silver/Silver Chloride ring electrodes). Data was sampled at 1 kHz, online filtered between 0.03 and 200Hz.

EMG activity was sampled at 1 kHz using MEG-compatible surface electrodes (Brain Products). The recorded signals were amplified and band-pass filtered between 20 and 450 Hz. During the trials, EMG activity was recorded from 3 muscles, 2 contributing to the elbow joint torque of the loaded (left) arm: BB and triceps long head (TLH) and 1 contributing to the elbow joint torque of the arm lifting the weight: brachioradialis (BR). The subject had to perform a series of brisk test movements to confirm the site of electrode placement. These movements were: elbow flexion for BB, elbow extension for TLH, and elbow flexion for BR while the right hand was in a semi-pronated position.

Data preprocessing and analyses

The photo-detector initiated trigger was used as the onset of lifting ($t=0$) to align brain and muscle activity to the unloading response. EMG data 400 ms preceding and 400 ms following unloading was grouped by condition, rectified, and averaged across trials.

EEG and MEG signals within an epoch of 2 sec preceding and 3 sec following the trigger were analyzed using Brain Electrical Source Analysis (BESA 5.2) software. Time-frequency representation (TFR) plots were calculated by expressing cortical oscillatory frequency as a percentage of that during a baseline period from 3000 to 4000 ms post-unloading. EEG and MEG data were averaged across trials to yield the evoked waveform for each condition. Loci of source activity were localised using a beamformer algorithm implemented in BESA 5.2 (Robinson, 1999) and reported in the Talairach coordinate system. Source images were superimposed on subject's anatomical MRI scan.

Results

EMG data

To examine EMG correlates of APA in the load-bearing arm, amplitude of EMG activity in the biceps brachii was plotted as a function of time (Figure 2). Averaged across trials, anticipatory inhibition of EMG activity in the biceps brachii preceded voluntary unloading by about 250 ms. The decrease in EMG amplitude reached its peak at less than -100 ms and returned to baseline level 50 ms after unloading. During imposed unloading, the inhibition of EMG activity in the biceps brachii started just before $t=0$.

EMG amplitude was at its minimum less than 100 ms after unloading and stabilized to a baseline afterwards.

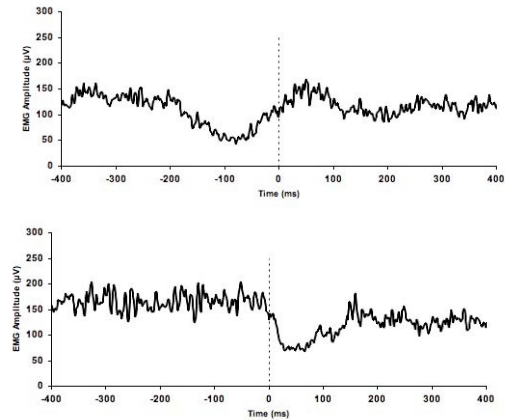


Figure 2: EMG amplitude of BB as a function of time relative to unloading ($t=0$) as indicated by the dotted lines. Averaged across trials ($n=80$), EMG amplitude of the BB started to decrease before voluntary (top) and after imposed (bottom) unloading respectively.

EEG / MEG data

To examine modulation of cortical oscillatory frequency before and after unloading, event-related spectral analyses in the range of 4 to 80 Hz were carried out. Since spectral analyses of EEG and MEG showed similar results, only results from MEG data are presented due to its superior resolution in source model solutions. The resulting TFR plots of all the sensors (not shown here) showed ERD and event-related synchronization (ERS) over the central-vertex, extending laterally to the motor area. Since the aim of the present study was to investigate the APA response, only TFR plots of one sensor over the right sensorimotor cortex is shown (Figure 3). During voluntary unloading, ERD in the 10 to 40 Hz frequency range started approximately 2 sec before onset. During imposed unloading, ERD in the same frequency band started immediately after $t=0$. Cortical oscillatory frequency returned to baseline about 2.5 sec after $t=0$ in both unloading conditions.

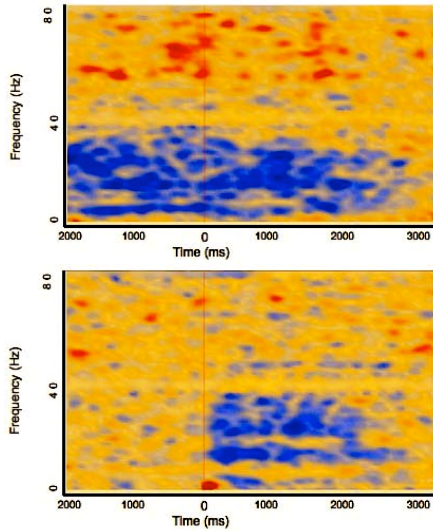


Figure 3: Mean cortical oscillatory frequency as a function of time from one sensor over the right sensorimotor cortex during voluntary (*top*) and imposed (*bottom*) unloading. The *red* lines represent the onset of unloading ($t=0$).

Averaged MEG waveform as a function of time plots from representative sensors over the left and right sensorimotor region showed differences in evoked magnetic fields during both unloading conditions (Figure 4). During voluntary unloading, amplitude of the evoked magnetic field over the left sensorimotor region was positive and reached a maximum of 313.5 fT 1305 ms before unloading. Subsequently, the amplitude started to decrease and reached a minimum of -214.5 fT 1194 ms after $t=0$, returning to baseline level 2496 ms after unloading. Amplitude of the evoked magnetic field over the right sensorimotor region was near baseline level before unloading and reached a maximum of 283.7 fT 1393 ms after $t=0$. The evoked field returned to baseline level 3900 ms after unloading. During imposed unloading, amplitude of the evoked magnetic field over the left sensorimotor region was near baseline level before and after unloading. In the right sensorimotor region, a transient maximum of amplitude 379.6 fT was measured 55 ms after $t=0$, returning to baseline level 133 ms after unloading.

Beamforming analyses in ERD in the beta frequency band (15 to 30 Hz) before (-300 to -100 ms) voluntary unloading localized brain activity to the right primary motor cortex, [27 -25 51], Brodmann Area (BA) 4. During imposed unloading, brain activity in the same frequency band immediately after (100 to 300 ms) unloading was localized to the right primary sensory cortex, [-32 -31 59], BA 3 (Figure 5).

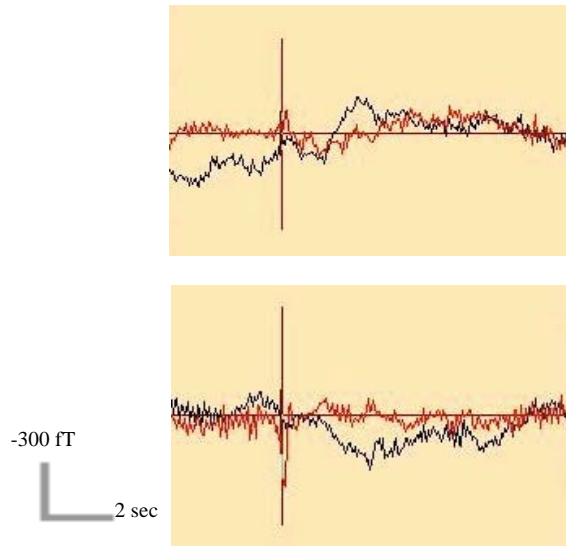


Figure 4: Averaged MEG waveform as a function of time. Waveforms from representative sensors over the left (*top*) and right (*bottom*) sensorimotor region indicate the amplitude of evoked magnetic fields during voluntary (*blue*) and imposed (*red*) unloading. *Vertical* lines represent onset of unloading ($t=0$).

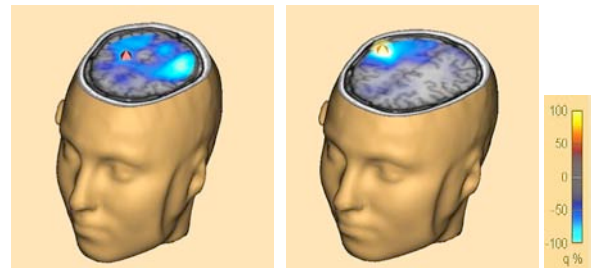


Figure 5: Source images superimposed on subject's anatomical MRI scan. Beamforming analyses in the beta frequency band (15 to 30 Hz) during a 200 ms time window before voluntary (*left*) and after imposed (*right*) unloading. Loci of sources are indicated by the *coloured* triangles.

Discussion

The present study investigated the systems of APA in a bimanual coordination task. EMG and MEG data suggested that during voluntary unloading, the brain anticipated the impending postural disturbance induced by the transient load change and responded by inhibiting EMG activity in the load-bearing muscle, i.e. BB to attenuate upward deflection of the arm. During imposed unloading, the inhibition of EMG activity in the BB started after the onset of unloading and corresponded to the classical unloading reflex reported by Struppler, Burg, & Erbel (1973), a feedback mechanism of sub-cortical origin.

EMG data

EMG data from previous studies (Hugon et al., 1982; Schmitz, Martin, & Assaiante, 2002; Taylor, 2005) showed that APA minimized deflection of the load-bearing arm during voluntary unloading. The anticipatory response was not a reflex but a command of supraspinal origin as the onset of EMG inhibition started 30 to 60 ms before unloading (Hugon et al., 1982; Massion, Alexandrov, & Vernazza, 1998; Diedrichsen et al., 2005). While this timing is different from the present study (see Figure 2), the difference can be accounted for by the different method used to assign the unloading latency in the current study. In most previous studies, a force-sensing device is used to detect the instance of transient load change and this latency is used as the onset of unloading. In the present experimental setup, the 1-kg weight had to be lifted off the photo-detector unit completely before unloading was registered. The averaged onset of the first EMG burst in the unloading arm was approximately 170 – 220 ms prior to the photo-detector register. If the trigger were shifted forward in time by 200 ms to account for the delay between the onset of unloading and complete unloading, the onset of APA would have occurred approximately 50 ms before unloading: a latency in agreement with earlier studies. For the purpose of comparing our results with findings from previous studies, the first EMG burst in the unloading arm was used as the onset of unloading. This is in agreement with previous studies that show the mean movement time to lift a 1-kg weight completely off a force-sensing device is about 200 ms (Viallet et al., 1987, 1992) and that the onsets of EMG activity in the unloading arm occurred quasi-synchronously with the instance of transient load change (Massion, Ioffe, Schmitz, Vialett, & Gantcheva, 1999; Forget & Lamarre, 1989; Massion et al., 1998). However, the same shift would be inappropriate for the imposed condition because the latency of the unloading reflex in the arm is in the order of 50 ms (Hugon et al., 1987; Schmitz et al., 2002; Forget & Lamarre, 1989). For imposed unloading, the correction advanced the trigger time by 50 ms bringing the current unloading reflex latency into agreement with the aforementioned studies.

EEG/MEG data

ERD represents the electrophysiological correlate of brain regions involved in processing of sensory or cognitive information or production of motor behaviour (Pfurtscheller, 1977, 1992). ERD in the alpha and beta frequency bands have been shown to precede and follow voluntary self-paced movements including button presses (Pfurtscheller & Aranibar, 1979) and rhythmic finger taps (Cheyne et al., 2006). In the present study, a broad band (10-40 Hz) ERD was observed bilaterally over the central-vertex region, extending laterally to the sensorimotor cortex during voluntary unloading. This finding concurred with studies that showed bilateral ERD during unilateral self-paced movement using surface EEG (Pfurtscheller & Aranibar, 1979; Cheyne et al., 2006; Babiloni, Carducci,

Cincotti, Rossini, Neuper, Pfurtscheller, & Babiloni, 1999) and intracerebral EEG (Rektor, Louvel, & Lamarche, 1998; Sockurkova, Rektor, Jurak, & Stancak, 2006). Given these findings, it was not clear whether ERD observed over the right sensorimotor cortex in the present study was related to the APA response in the left arm. However, during imposed unloading, the preponderance of ERD over the right sensorimotor cortex after unloading suggested that the observed ERD in the same frequency band was related to the inhibition of EMG activity in the load-bearing arm.

Prior to voluntary unloading, the subject had to reach across the body to lift the weight with the right hand. Neural activity responsible for this movement might have evoked a positive magnetic field that was observed over the contralateral sensorimotor region. During imposed unloading, where the right hand was completely at rest, amplitude of the evoked field over this region did not deviate significantly from the baseline level. In the present study, the left arm supported the weight regardless of the unloading condition. Neural activity responsible for supporting the load should not differ significantly between conditions. This was shown by the overlapping waveforms near baseline level over the right sensorimotor region prior to unloading. After $t=0$, imposed unloading resulted in a transient increase in evoked field amplitude, whereas a slow buildup was observed during voluntary unloading. We suggest that this difference might be related to the speed of arm deflection during unloading.

Results from previous studies showed that a slow magnetic field shift emerges 1 to 2 sec prior to onset of voluntary movements (Deecke, Weinberg, & Brickett, 1982; Deecke, Lang, Beisteiner, Uhl, Lindinger, & Cui, 1998; Cheyne et al., 2006) reaching peak amplitude 50-60 ms before movement onset. In these studies, the subjects flexed their index finger at regular intervals of 8-10 sec, resting completely between trials. In the present study, no obvious field shift was found over brain regions that corresponded to the sensorimotor cortex. We speculate that the complexity of the bimanual movement contributed to the absence of such shifts in the present task. Although the inter-trial interval was comparable, if not longer than those mentioned in previous studies, the right hand was not at rest prior to unloading. Due to the supine body position and arm configuration, the right hand had to reach across before it could grasp the weight. The reach and grasp movement of the right hand 1-2 sec prior to unloading could have confounded the resting baseline of the evoked cortical field, thus obscuring the profile of the slow shift over the left sensorimotor region. Whether a similar slow shift of the magnetic field exists over the right sensorimotor region was not clear because inhibition of EMG activity in the load-bearing muscle was not volitional per se, but mediated subconsciously by a timing signal sent from the hemisphere contralateral to the movement arm (Viallet et al., 1992; Massion et al., 1998; Massion et al., 1999).

Beamforming analyses in the beta frequency band (15-30 Hz) pre- voluntary unloading localized brain activity to the

right primary motor cortex (M1). During imposed unloading, beamforming analyses localized post-unloading brain activity to the right primary sensory cortex (S1). Martineau et al. (2004) found pre and post-unloading ERD over the right M1 in children during voluntary and imposed unloading respectively. Our results for the latter condition is different from those reported previously. In that study, only data from specific electrodes placed above M1 was reported. It is possible that other electrodes adjacent to M1, such as those over S1, could have demonstrated similar desynchronisation of cortical rhythms. Furthermore, S1 activation is more likely to be associated with cortical processing of afferent signals and should correspond to the long-latency component of the stretch reflex in human muscles (Corden, Lippold, Buchanan, & Norrington, 2000; Lewis, Polych, & Byblow, 2004). Taken together, pre-unloading ERD in M1 suggests that the brain anticipates postural disturbance induced by the transient load change and codes preemptive inhibition of EMG activity in the loaded arm, i.e. APA to minimize arm deflection. This was not the case in imposed unloading as compensatory response to regain arm posture occurred only after the onset of unloading.

APA and motor problems in ASD

Kanner (1943) and Asperger (1944) described early observations of motor behaviour anomalies in ASD. Using movement analysis techniques, Teitelbaum, Teitelbaum, Nye, Fryman, & Maurer (1998) showed that motor-related deficits are observable during infancy, years before social and linguistic dysfunctions become apparent. Leary and Hill (1996) speculated that motor dysfunction might underlie some of the core features of ASD such as speech production or social interaction. One line of research has been to establish motor-related deficits in ASD. Results from previous studies that utilized standard tests of motor functions including physical and neurologic examination of subtle signs, movement assessment battery for children, and Bruininks-Osteretsky test of motor proficiency showed that individuals with ASD performed less optimally than normal controls in motor test items (Green, Baird, Barnett, Henderson, Huber, & Henderson, 2002; Jansiewicz, Goldberg, Newschaffer, Denckla, Landa, & Mostofsky, 2006; Dewey, Cantell, & Crawford, 2007). Results from other studies that examined mechanisms of motor dysfunctions suggest that individuals with ASD were impaired in processes related to movement planning (Rinehart et al., 2001; Rinehart, Bellgrove, Tonge, Brereton, Howells-Rankin, & Bradshaw, 2006), movement reprogramming (Nazarali, Glazebrook, & Elliot, 2009), and motor skill generalization (Haswell et al., 2009). Up till now, few studies had directly examined the neural bases of motor-related problems in ASD (Martineau et al., 2004; Rinehart, Tonge, Bradshaw, Iankse, Enticott, & Johnson, 2006). Results from these studies demonstrated APA anomalies and impaired Bereitschaftspotential in autistic individuals during volitional motor actions. More research

is needed to better understand the association between neural abnormalities and motor dysfunctions.

To this end, the bimanual load-lifting task involving high-level neuro-motor coordination might be a good model for studying motor-related problems in ASD. During imposed unloading, reflexive inhibition of EMG activity in the loaded arm should not differ between ASD and normal controls since the response is primarily mediated via short-latency spinal circuitries (see Martineau et al., 2004). Conversely, the quasi-synchronous activation of excitatory EMG for voluntary unloading and inhibitory EMG for APA in anticipation of the sudden load change must be controlled at a supra-spinal level. According to Massion et al. (1998, 1999), execution of the motor program for voluntary unloading triggers a timing signal that traverses the hemispheres via sub-cortical collaterals to activate the motor program for APA. This high level neural coordination may be impaired in autistic people. Brock, Brown, Boucher, & Rippon (2002) suggest that ASD is associated with abnormalities of information integration and processing caused by disordered neuronal wiring between functional brain regions. Within this ‘temporal binding’ model framework we will in future test the hypothesis that motor dysfunctions in ASD is in part caused by disordered connectivity between activated brain regions. Furthermore, we speculate that any significant difference in APA in terms of brain activation, oscillatory frequency, muscle activation, and cortico-cortical coherence should help to elucidate the neural bases of neuro-motor incoordination in ASD. We will also use cortico-muscular coherence measures to determine whether abnormalities of motor output drive are associated with coordination deficits in autistic people.

Conclusion

The aim of this pilot study was to replicate the findings of Martineau and colleagues (2004). During voluntary unloading, anticipatory cortical ERD and inhibitory EMG in the contralateral loaded arm mediated the APA response thereby minimizing arm deflection. This phenomenon was not observed during imposed unloading. Results from the present study show that by increasing the number of trials per condition, ERD of cortical activity can be localized to the primary motor cortex in single subject. This suggests that the brain region was involved in coding inhibitory signals to the loaded muscle even before the onset of unloading to counteract the impending load change. Further testing of normal subjects up to $n=15$ should increase the power of the preliminary results reported here. In future, the data will be used as a reference for comparison with an autistic sample.

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