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Hyperactive action monitoring during motor-initiation in conversion paralysis: An event-related potential study

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Abstract

Conversion paralysis (CP) is featured by a stress-induced tonic immobility. Although the neural correlates of this psychiatric condition remain largely unexplored, previous reports showed CP to be associated with anterior cingulate cortex (ACC) hyperactivity. We examined the ACC action monitoring function by recording event-related potentials (ERPs) when conversion patients ($n = 6$) with unilateral arm paresis made speeded responses with their affected and healthy arms on a flankers task. During this task, pre-response ACC action monitoring is reflected in the N2 ERP component, which is increased when incongruent stimuli lead to simultaneously activated competing response tendencies. The results showed that the N2 congruency effects were significantly increased for responses with affected hands compared to healthy hands. There were no such results for post-response monitoring. This study is the first to present electrophysiological correlates of action monitoring in CP and suggests ACC to be hyperactive when movements with affected arms are to be initiated.

Keywords: Conversion paralysis; Action monitoring; Event-related potentials; Flankers task; Hysterical paralysis

1. Introduction

Conversion paralysis (CP) refers to a non-organic, stress-induced tonic immobility (American Psychiatric Association, 1994). In CP, the failure to initiate voluntary movement was previously found to be associated with hyperactivity of the anterior cingulate cortex (ACC). Using positron emission tomography (PET), Marshall et al. (1997) studied brain activity when a patient with unilateral CP attempted to move her affected versus her unaffected leg. When attempting to move the unaffected (right) leg, there was a normal pattern of activation including activation in the primary motor cortex (M1). However, when attempting to move the affected (left) leg, there was no activation of the right M1 but a relative increase in activation of the right ACC and orbitofrontal cortex (OFC). The authors concluded that the ACC and the OFC inhibited the prefrontal effects on the right M1 when the patient tried to move her affected left leg. These findings are in agreement with the results of several reaction time studies indicating that CP is associated with a failure in the intentional, prefrontal generated, motor control (Roelofs et al., 2001, 2002, 2003). Patients with conversion paresis, for example, showed impaired performance on a voluntary spatial attention task that strongly appeals to the ACC. This impairment was particularly pronounced when the task primed responses with affected arms, compared to unaffected arms (Roelofs et al., 2003). When contrasting motor-initiation and execution, CP was found to predominantly involve problems in the explicit motor-initiation and not in motor execution per see (Roelofs et al., 2002).

The identification of the ACC as a crucial structure involved in stress-related motor inhibition is not new. Reports on 'anterior cingulate cataplexy' date from half a century ago and refer to a loss of muscle tonus commonly induced by strong emotion (Ethelberg, 1950; Fuster, 1955; Levin, 1953). More recent investigations indeed showed that negative emotional stimulation induced hyperactivation in orbitofrontal cortex and a shift of main activation to ACC

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and medial prefrontal cortex in catatonic patients (Northoff et al., 2004). Also in animals the role of the ACC in the modulation of motor inhibition and freezing reactions has been well established (e.g. Lacroix et al., 2000). The ACC has direct connections with limbic structures, such as the amygdala and hypothalamus, which are known to be important in the expression of fear and anxiety (Gray, 1987; Ledoux, 1996). Chronic behavioural stress (Radley et al., 2004), as well as administration of the stress hormone cortisol (Wellman, 2001) were found to induce dendritic reorganization in pyramidal neurons of the ACC in rats. Moreover, lesions to the ACC were shown to result in behavioural changes to stress, such as reduced freezing reactions to novel situations (Lacroix et al., 1998). In contrast, increased behavioural inhibition in patients with anxiety disorders has been associated with hyperactivity in ACC action monitoring functions (Gehring et al., 2000; Hajcak et al., 2003; Hajcak and Simons, 2002; Ursu et al., 2003).

Although these findings support the hypothesis that ACC hyperactivity may play a role in the stress-induced immobility observed in conversion paralysis, it remains largely unexplored how the ACC may exert this role. Recent insights from neuro-imaging and ERP studies have led to new theories on how the ACC affects prefrontal motor control and offer methods for a more detailed, temporal investigation of the role of the ACC. There is increasing evidence that the ACC is involved in the monitoring of action, both before (pre-response) and after movement has been initiated (post-response). As far as pre-response monitoring, neuro-imaging studies in both healthy volunteers and patients with psychiatric disorders have shown that the ACC is specifically activated during tasks in which concurrent response tendencies are in conflict (Botvinick et al., 1999; Carter et al., 1998; Ursu et al., 2003; Van Veen et al., 2001). Event-related potential (ERP) recordings during similar conflict tasks have led to the identification of the stimulus-locked N2 ERP component, which is suggested to reflect pre-response conflict evoked by incongruent stimuli (Van Veen and Carter, 2002a,b). Besides pre-response monitoring, the ACC is proposed to exert post-response evaluation by error detection (Holroyd and Coles, 2002; Kiehl et al., 2000). Several ERP studies showed erroneous responses to be associated with a negative deflection in the ERP observed around 60–100 ms after an erroneous response, the so-called error negativity or error-related negativity (ERN) (Falkenstein et al., 1991; Gehring et al., 1993). Source localization studies suggest that the ERN and the N2 have a common neural source in the ACC (Van Veen and Carter, 2002b; Nieuwenhuis et al., 2003; Yeung et al., 2004).

In sum, the ACC hyperactivity observed in CP (Marshall et al., 1997) may result from overactive ACC action monitoring as reflected in increased N2 and ERN amplitudes. Testing this hypothesis is important to gain insight into the neural mechanisms underlying this age-old but poorly understood psychiatric condition of CP. The present study was designed to investigate ACC response monitoring by measuring N2 and ERN amplitudes in patients with conversion paresis (muscular weakness) of one arm during performance of the Eriksen Flankers Task (Eriksen and Eriksen, 1974). This speeded two choice reaction task requires motor responses to congruent and incongruent visual stimuli and is known to evoke response conflict (e.g. Coles et al., 1985; Kopp et al., 1996). Only patients that were unilaterally affected and still able to handle a response button with their affected arm were selected to facilitate powerful within-subject comparisons of the response monitoring for the movements with the affected and healthy arm. Patients could therefore serve as their own controls and no additional control group was needed. Because CP predominantly involves problems in the initiation of movements (Roelofs et al., 2001, 2002) we expected to find overactivity in pre-response monitoring (as reflected in increased N2 amplitudes) in specific.

2. Methods and materials

2.1. Participants

A total of six female patients (all right-handed with a mean age of 30.72 years, range = 15–49, S.D. = 14.98) diagnosed with conversion disorder according to the DSM-IV criteria (American Psychiatric Association, 1994) and showing paresis to one arm as a major symptom, participated in the study. The patients had been referred for either in- or outpatient treatment to a general psychiatric hospital specializing in the treatment of conversion disorders. A psychiatrist assessed whether the patients met the DSM-IV criteria for conversion disorder. A trained psychologist checked for other axis-I diagnoses using the Structured Clinical Interview for DSM-IV Axis-I Disorders (SCID-1/p: First et al., 1996). A neurologist screened all patients for somatic and neurological disorders. Whenever the somatic screening revealed any deviations that might explain the symptoms, the patients were not diagnosed with conversion disorder and were excluded from the study. Other exclusion criteria were symptoms involving pseudo-epileptic insults, tremors, sudden movements and deteriorated speech or vision. Moreover, only patients who could still handle a response button with their affected arm and whose other arm was unaffected were selected. Three patients showed conversion paresis to the right arm and the other three to the left arm. Table 1 shows relevant information with respect to the patients' age, the duration of complaints, psychiatric comorbidity, life-events and symptom severity. The study was approved by the local medical ethical committee and all patients gave their informed consent before participation.

Table 1
Patient information

Patient	Age	Duration of complaints[a]	Axis-I comorbidity (SCID-I)	Medication	History of traumatic events[b]	Events preceding symptom onset	MVC[c] healthy arm	MVC affected arm
1	32	3	Generalized anxiety disorder	Oxazepam 10 mg[d]	Incest	Loss of friend	5.35	2.77
2	17	6	–	–	–	Car crash	2.99	1.02
3	49	33	Depressive disorder PTSD	Paroxetine 20 mg/d	Incest	Loss of job	2.99	0.31
4	47	17	–	Sertraline 50 mg/d	Sexual abuse	Family conflict	2.55	2.50[e]
5	15	2	–	–	–	Hospital admission	3.37	0.43
6	21	13	–	Paroxetine 20 mg/d	–	Car accident	4.85	2.17

[a] In months.
[b] Assessed by means of the structured trauma interview (Draijer, 1989).
[c] MVC, maximum voluntary contraction in Newtons assessed by a force application that involved the same movement as the responses required on the Eriksen Flankers Task.
[d] Patient used medication on an irregular basis and refrained from taking the drug 10 h prior to the experiment.
[e] The relatively small difference in the force levels of patient 4 may be attributable to the fact that this patient had intermitted loss of force in her affected arm.

2.2. Design and procedure

Before the start of the experiment, isometric force measurements of maximum voluntary contractions (MVC) were obtained by means of a load cell transducer (type BC302, DS Europa s.r.l., Italy). Participants were seated with their forearms resting on a flat desktop. The position of the hand and forearm were adjusted so that both fingertips of the index fingers rested on the load cell. After being familiarized with how to exert forces by using the push buttons, the MVCs for the left and right index finger were assessed separately (Table 1). Participants were asked to briefly press the push button as hard as they could for seven consecutive times with one index finger. The maximum and minimum force values of these seven measurements were discarded. An average was computed of the remaining five values. This average became the MVC value, which was determined for both index fingers of each participant. A standard push button box replaced the load cell transducers for the remainder of the experiment.

All patients performed a speeded two-choice reaction task, the so-called 'flankers task' (Eriksen and Eriksen, 1974). Visual stimuli in this task are letter strings of five letters (HHHHH, SSSSS, HHSHH or SSHSS). During this task, participants are instructed to respond as fast as possible to the central letter of the target string while ignoring the surrounding or flanking letters, by pushing a button with their left or right index finger. The stimulus-response mappings are known to the participants beforehand. Thus, instructions may, for example, state that a left hand response is required when the H is the central letter and a right hand response when the central letter is an S. Four strings of letters are presented, two with five identical letters (congruent stimuli: HHHHH or SSSSS) and two including deviating flanking letters (incongruent stimuli: HHSHH or SSHSS). When congruent stimuli are presented, responding is relatively easy, as all visual information primes the correct response. In the case of incongruent stimuli, responding is more difficult as the visual information elicits conflict during response selection between the responses afforded by the target stimulus and the distractor stimuli that are associated with the opposite, conflicting response (Gratton et al., 1988). Stimulus-response mappings were counterbalanced across participants. Emphasis in the written and verbal instructions was placed on fast responding to avoid feedback indicating that the given response was too late according to a preset reaction time (RT) deadline. This individual RT deadline was used, as previous ERN studies had demonstrated that accuracy could affect ERN amplitude (see, e.g. Gehring et al., 1993). This procedure, which had already been employed successfully in previous studies (see, e.g. De Bruijn et al., 2004), ensures that performance levels are kept similar across participants. The RT deadline was calculated on the basis of a practice block of 60 trials during which the RT deadline was set to a liberal limit of 1000 ms. After completion of the practice trials, the participant's average RTs and standard deviations (S.D.) for the correct responses were computed. Subsequently, the RT deadline for each individual participant was determined by adding 0.5 S.D. to this mean RT.

The experimental phase consisted of six blocks of 100 trials each, i.e. 50 congruent and 50 incongruent stimuli, with a self-paced pause halfway each block. After each block, participants were informed on the number of incorrect and late responses and, additionally, verbal encouragements were given to keep performance accuracy around 90%. Each trial started by the presentation of a 100 ms lasting fixation point. After 300 ms, the stimulus appeared, again lasting 100 ms. During the next 900 ms, the screen remained blank after which a visual feedback stimulus (a 1000 ms lasting rectangle in yellow, blue or red) indicated correct, incorrect or late responses, respectively. After another 100 ms the next trial started. The experimental phase lasted about 40 min including breaks.

2.3. EEG recording

The electroencephalogram (EEG) was recorded from 27 tin electrodes mounted in an elastic electrode cap (Electrocap International). Electrodes were placed at seven midline.

(FPz/AFz/Fz/FCz/Cz/Pz/Oz) and twenty lateral (FP1-2/F7-8/F3-4/FC5-6/T3-4/C3-4/CP5-6/T5-6/P3-4/O1-2) locations in accordance with the international 10–20 system. All electrodes were referenced to the left mastoid, but were offline re-referenced to the average of the left and right mastoid. The vertical electro-oculogram (EOG) was recorded bipolarly from electrodes placed above and below the right eye. The horizontal EOG was also recorded bipolarly from electrodes lateral to both eyes. All electrode impedances were kept below 5 kΩ. The EEG and EOG signals were amplified using a time constant of 8 s and were filtered offline low-pass at 15 Hz. All signals were digitized with a sampling rate of 200 Hz using a 16-bit A/D converter.

2.4. Data analyses

EOG artefact correction was carried out using the procedure proposed by Gratton et al. (1983). For both behavioural and ERP analyses all responses with reaction times faster than 150 ms or slower than the set RT deadline, were removed from the data sets (6.59%). For each participant, epochs associated with correct responses were averaged separately for stimulus type (congruent, incongruent) and response hand (healthy, affected), time-locked to stimulus onset. The epochs ranged from 200 ms before until 500 ms after stimulus onset and were baseline corrected relative to a 200 ms pre-stimulus time window, before averaging. In the resulting stimulus-locked subject averages, N2 amplitude was defined as the most negative peak in the 200–400 ms time window after stimulus onset at electrodes Fz and FCz, where maximal N2 effects were expected. For each participant, epochs were also averaged time-locked to response onset, for correct and incorrect responses given with the healthy or the affected hand separately. These response-locked epochs, ranged from 100 ms before until 400 ms after response onset and were baseline corrected, relative to a 100 ms pre-response baseline, before averaging. Epochs containing movement or recording artefacts (8.21%) were removed from the dataset and were thus not included in the averages or analyses. In the resulting subject averages, ERN amplitude was determined on incorrect trials by subtracting the most negative peak in the 0–150 ms time window after response onset from the most positive peak in the time window starting 80 ms before and ending 80 ms after response onset at electrode FCz and Cz, where maximal ERN amplitudes were expected.

To investigate whether there are differential effects for response hand in pre-response and post-response action monitoring, average peak amplitudes of the ERN and the N2 congruency effects were entered into two separate analyses of variance (ANOVAs) with repeated measures on the electrode site and response hand (affected, healthy). To analyze the behavioural data, reaction times were entered into an ANOVA with repeated measures on stimulus type (congruent, incongruent), response (correct, incorrect) and response hand (affected, healthy). All statistical analyses employed a two-tailed alpha of 0.05.

Table 2
Mean reaction times and error rates on the Eriksen Flankers Task

Reaction time (ms)	Healthy hands		Affected hands	
	Congruent	Incongruent	Congruent	Incongruent
Correct responses	429 (75)	483 (87)	555 (259)	585 (267)
Incorrect responses[a]	356 (126)	381 (99)	395 (130)	408 (135)
Incorrect trials (%)	3.8 (1.7)	8.8 (5.9)	1.94 (1.4)	5.64 (2.5)

Standard deviations are given in parentheses.
[a] Note that patient 3 made no errors with her affected hand.

3. Results

3.1. Behavioural results

The error rates and reaction times for congruent and incongruent stimuli on the flankers task are presented in Table 2. When examining correct responses only, RTs (mean [S.D.]) were longer for incongruent stimuli (534 [164] ms) than for congruent stimuli (492 [158] ms) [$F(1, 5) = 77.04$, $p < 0.001$]. There were no main effects for response hand (affected: 570 [263]; healthy: 456 [80] ms) [$F(1, 5) = 1.67$, $p = 0.25$] and no interaction effects between response hand and stimulus type (congruent, incongruent) [$F(1, 5) = 1.50$, $p = 0.28$]. One patient made no erroneous responses with her affected hand that fell within the preset time limits. Due to empty cells, her data were excluded from the behavioural analyses for incorrect responses. In general, more errors were made to incongruent stimuli (7.2%) than to congruent (2.8%) ones [$F(1, 4) = 21.67$, $p < 0.01$] and RTs were shorter for incorrect responses (385 [120] ms) compared to correct responses (462 [114] ms) [$F(1, 4) = 63.31$, $p < 0.001$].

3.2. Pre-response monitoring

The N2 congruency effects for responses with affected and healthy hands are displayed in Fig. 1. An ANOVA of the average peak amplitudes of the N2 with repeated measures on the electrode site (two levels: Fz versus FCz), congruency (two levels: congruent versus incongruent), and hand (two levels: healthy versus affected) yielded a main effect for congruency [$F(1, 5) = 6.75$, $p < 0.05$], indicating that N2 amplitudes were more negative after incongruent stimuli (−0.07 [2.73] μV) than after congruent stimuli (1.39 [2.10] μV). There were no main effects for electrode or hand [both Fs < 1]. However, the interaction between congruency and hand was significant [$F(1, 5) = 12.19$, $p < 0.05$]. Post hoc t-tests demonstrated that this interaction was caused by an increase in N2 amplitudes after incongruent trials responded to with the affected hand [$t = 3.12$, $p < 0.05$], but not after congruent trials [$t = -0.87$, $p = 0.43$] (see, also Fig. 1, lower panel).

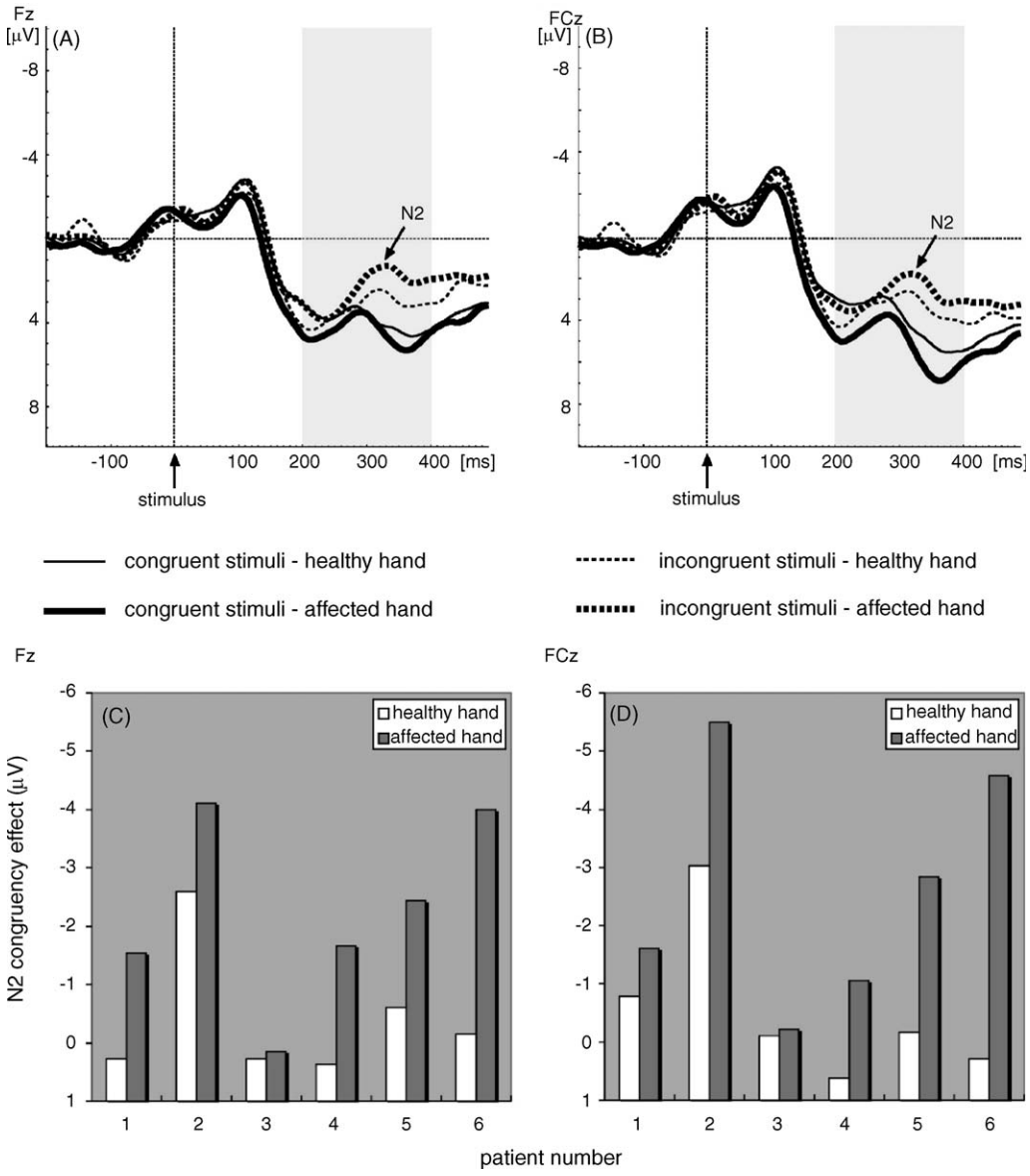

Fig. 1. ERPs measured from stimulus onset (0 ms). The upper panel (A and B) shows the ERPs for congruent (solid lines) and incongruent (dashed lines) stimuli followed by a correct response with the healthy (thin lines) or with the affected hand (thicker lines) at electrodes Fz (A) and FCz (B). The grey areas in the upper panel indicate the time window in which the N2 peak, identified by the arrow, was defined. The lower panel (C and D) shows the individual N2 congruency effects (N2 amplitude incongruent minus N2 amplitude congruent) for healthy and affected hands at electrodes Fz (C) and FCz (D).

Finally, to rule out that the N2 effects may be attributed to possible differential effects in the P2 time range, an additional three-way ANOVA (electrode side × congruency × hand) for the P2 component (P2 amplitude defined as most positive peak preceding the N2) was conducted. The results showed that the critical hand × congruency effect found for the N2 amplitudes was not manifested by the P2 component [$F(1, 5) = 1.27$, $p = 0.31$].

3.3. Post-response monitoring

To check whether there might also be differential effects for response hand on post-response monitoring (Fig. 2), ERN peak amplitudes of the subjects' averages were analyzed using an ANOVA with repeated measures on electrode site (two levels: FCz and Cz) and hand (two levels: healthy versus affected). This analysis yielded only a main effect for electrode site [$F(1, 4) = 7.83, p < 0.05$], indicating that ERN amplitude was larger at FCz (9.47 [6.26] μV) than at Cz (8.57 [6.41] μV). There was no main effect for hand and also the interaction between electrode site and hand was not significant [both Fs < 1], indicating that post-response ERN amplitude was not modulated by response hand. Note that patient 3 made no errors with her affected hand and was therefore excluded from the response-locked analyses. See Fig. 3 for current source density maps of the scalp topography of the N2 and ERN.

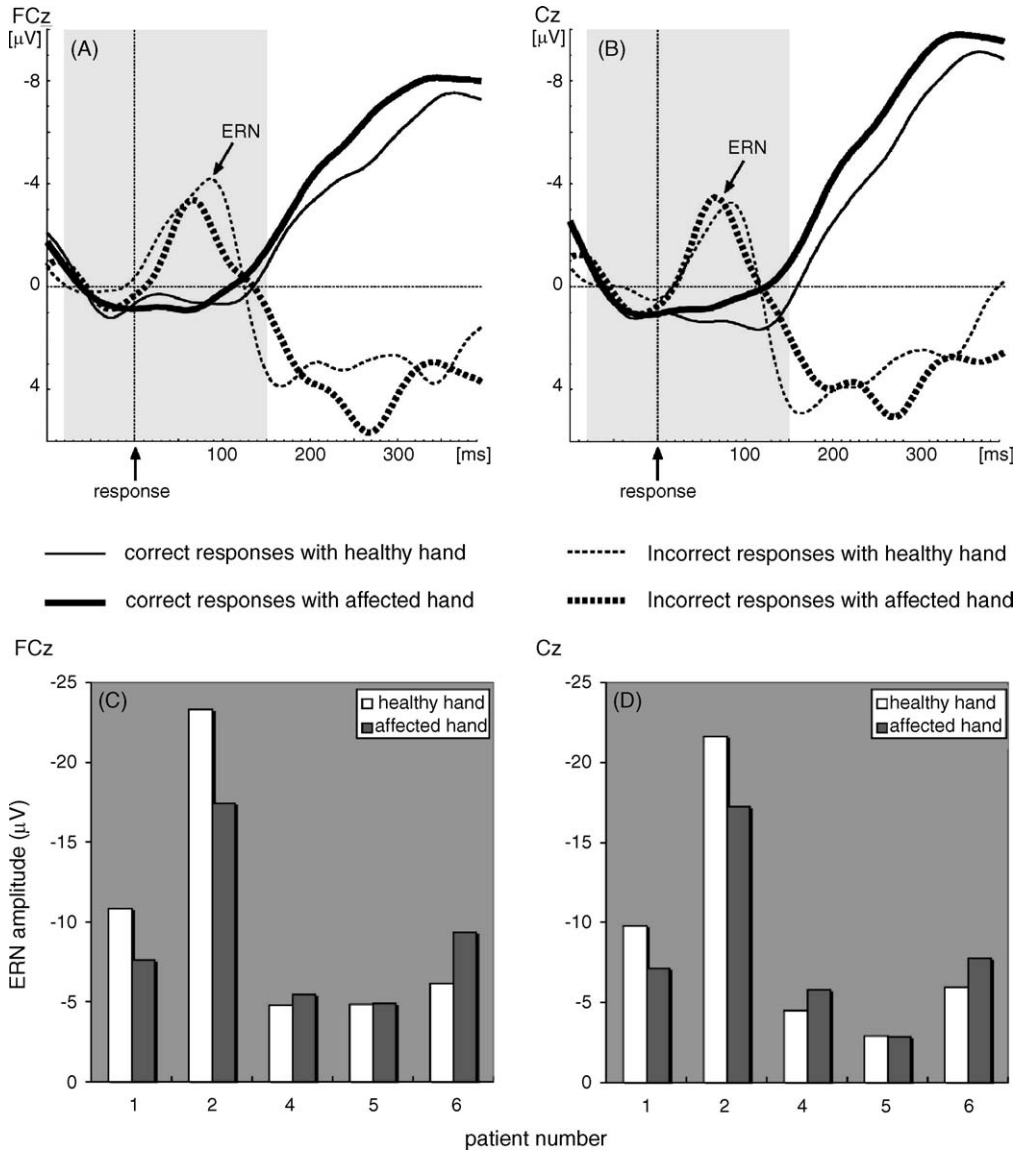

Fig. 2. ERPs measured from response onset (0 ms). The upper panel (A and B) shows the ERPs for correct (solid lines) and incorrect (dashed lines) responses given with the healthy (thin lines) or with the affected hand (thicker lines) at electrodes FCz (A) and Cz (B). The grey areas in the upper panel indicate the time window in which the ERN, identified by the arrow, was defined. The lower panel (C and D) shows individual ERN amplitudes for errors made with the healthy or the affected hand at electrodes FCz (C) and Cz (D). Note that patient 3 made no errors with her affected hand and was therefore excluded from the response-locked analyses.

4. Discussion

The present study aimed to investigate electrophysiological correlates of action monitoring in patients with conversion paralysis. Conversion paralysis was previously shown to be associated with ACC hyperactivity (Marshall et al., 1997). We hypothesised this increased ACC activity to reflect a hyperactive action-monitoring system. In line with this theory, the results indeed showed the N2 congruency effect, thought to reflect the monitoring of pre-response conflict, to be enhanced when patients responded with their affected hand compared to the healthy hand. These findings indicate that on trials that evoked simultaneous activation of competing response tendencies, CP patients showed hyperactive action monitoring when they had to choose and initiate a response with the affected hand.

Significant differences between responses with the affected and healthy hands were only found for the pre-response N2 congruency effects and not for the post-response ERN. The specific increases in N2 congruency effects indicate that the altered motor control is already manifested at an early stage of motor processing, before the actual motor response takes place. This result fits in with previous findings indicating that CP is associated with impairments in the initiation of movements and not the execution of movements per se (Roelofs et al., 2001, 2002, 2003).

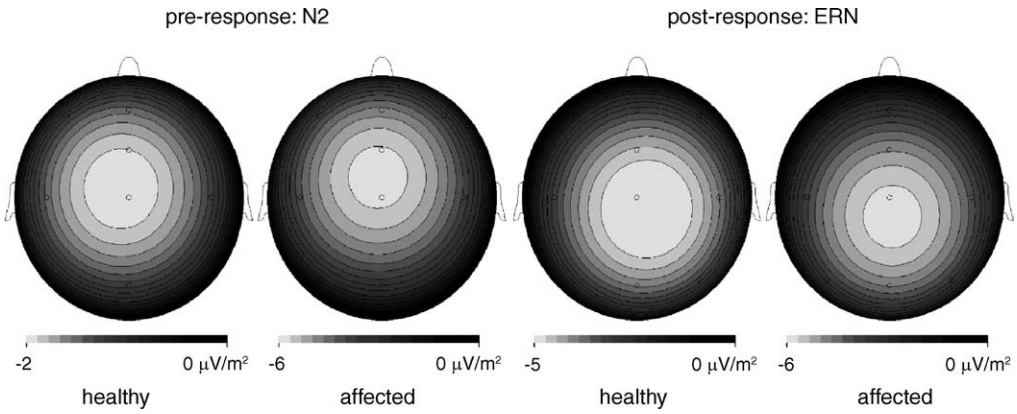

Fig. 3. Current source density maps of the stimulus-locked difference waveforms (incongruent minus congruent) representing the scalp topography of the N2 at peak amplitude (345 ms) and response-locked difference waveforms (incorrect minus correct) representing the scalp topography of the ERN at peak amplitude (60 ms). Lighter areas correspond to larger differences. Please note that, for comparison purposes only, different scales are used.

The fact that the increased N2 congruency effects were observed for responses with affected limbs only is in agreement with previous findings of the PET study by Marshall et al. (1997). In this latter study, ACC hyperactivity was also only present when the CP patient attempted to move her affected limb and was not observed for her healthy limb. These findings, together with the results from brain-imaging and ERP studies indicating that pre-response conflict monitoring, as reflected by the N2, stems from the ACC (Botvinick et al., 1999; Carter et al., 1998; Nieuwenhuis et al., 2003; Van Veen et al., 2001; Van Veen and Carter, 2002a; Yeung et al., 2004) support the evidence for the involvement of ACC in the failing motor control in CP. Moreover, and for the first time, the use of ERP measurements in the present study offered a further insight into the temporal features of the ACC action monitoring, showing the detection of pre-response conflict in particular to be altered. According to conflict monitoring theory, upon detection of response conflict by the ACC, requests for extra cognitive control are sent to the lateral PFC (Botvinick et al., 2001; Carter et al., 1998; Van Veen and Carter, 2002b). Consequently, the increased ACC and decreased M1 activity found in the case study by Marshall et al. (1997) could also be explained by this control network. An attempt to move the affected leg resulted in higher response conflict (increased ACC activation) and, consequently, in more inhibitory cognitive control over M1. Although there is increasing evidence for the proposed role of the ACC in performance monitoring (Ridderinkhof et al., 2004) it should be noted that the ACC may also exert a more executive role in implementing control directly during response selection (Posner and DiGirolamo, 1998; Matsumoto and Tanaka, 2004). Detailed investigation of the communication between ACC and its projection area's is needed to determine the exact role of the ACC in response monitoring.

The present findings offer new insights into the electrophysiological correlates of the failing motor control in conversion paralysis. We cannot, however, derive a definitive causal explanation from the findings because it is still unclear whether our findings reflect a cause, or merely a consequence of CP. One may, for example, argue that the increased N2 congruency effects for movements with affected limbs simply reflect a general limb preference because patients are likely to favour responses with their healthy limb over responses with their affected limb. However, if the effects would reflect an unwillingness to move their affected hand one would rather expect to find increased N2 amplitudes on congruent stimuli because these stimuli contain no distracting flankers and unambiguously inform the subject that a movement with the affected arm is required. The fact that we found increased N2 amplitudes on incongruent trials and not on congruent trials indicates that the action monitoring system for affected hand movements is hyperactive in case of conflicting task demands only. Apparently more conflict needs to be resolved when selection for action with the healthy hand by the distracting flankers needs to be inhibited. That these increased N2 congruency effects for affected hand responses go beyond a possible conflict elicited by hand preference is, moreover, indicated by the absence of any such effects in healthy right-handed subjects responding with their left hand. When examining 12 healthy right-handed subjects performing the same task, we found no differences in N2 congruency effects for responses with the left and right hand ($F < 1$), indicating that our findings cannot be attributed to preference (De Bruijn, 2003). Another argument to exclude any effect of a conscious intention to repress actions with affected limbs is the time course of the manifestation of the increased N2. The differentiation of N2 for congruent and incongruent stimuli already starts 200 ms after stimulus presentation, which is too quick for any intentional intervention. We therefore conclude that the results are not simply the result of an explicit unwillingness to move the affected hand. On the contrary, the results indicate that action monitoring is overactive when responses with the affected arm are to be initiated and selection for action with the healthy hand needs to be inhibited in a context of conflicting task demands.

To our knowledge, the present study is the first to show electrophysiological correlates of ACC action monitoring in patients with conversion paralysis. Although the findings offer sound support for the role of the ACC in conversion paralysis there are some limitations to the study that need to be addressed. Although our sample size is relatively high compared to those of other neurophysiological studies in CP, six patients remains a small amount. We should therefore consider that the null results for the ERN data may be due to lack of power. Closer inspection of the absolute ERN effects does, however, not point into this direction. Whereas the N2 effects for all patients demonstrate an increase for affected arm performance, there is no single sign for such trend with respect to the ERN effects. Another point of discussion is that the differential N2 effects for affected and healthy hands were not accompanied by significant differential RT effects for hands. Although the absolute RTs pointed at a relative slowing for affected hand responses, there were large variations in the RTs for the affected hands. This was probably due to motor fluency problems and large variations in muscle tone for the affected limbs. To minimize the efforts required for a motor response the patients' index fingers continuously rested on the response buttons and only minimal effort was needed to press the buttons. Nevertheless, the standard deviations for affected hand responses were extremely high. Together with the low N this may have resulted in a lack of power to reach significance on the behavioural data. Examination of the congruency effects for the affected hand responses separately, however, demonstrated a significant slowing for incongruent trials, supporting the N2 effects for affected hands. Another question to address is whether the patients' performance with the healthy hand serves as a good control for the affected hand because it may not be representative for performance of healthy controls. However, the problem with a direct comparison between patients and healthy controls is that a possible hyperactivity in ACC action monitoring in patients can also be attributable to the effects of comorbid mood and anxiety disorders and not to the direct effects of CP. Several studies so far have shown that patients with anxiety disorders demonstrate hyperactive action monitoring (e.g. Gehring et al., 2000). The purpose of our study was to identify neural correlates specific for movements with the affected hands of the patients. To make sure that possible differential effects in action monitoring could be directly related to the specific symptoms, we used a within-subject design, controlling for eventual effects of comorbid psychiatry, use of medication and personality factors. A final point of discussion addresses the generalizability of the findings. To enable direct comparison of performance with affected and healthy limbs we selected patients with some mobility in their affected hands, raising limitations with respect to the generalizability of the results to cases of full paralysis.

Despite these limitations, the results offer important implications for future research into the neurobiological and neurobehavioral mechanisms underlying conversion paralysis. At least two currently predominant hypotheses on the aetiology of CP should be tested in the light of the present results. One hypothesis is that CP is the result of aversive conditioning of movements with affected limbs during exposure to preceding traumatic or stressful events (e.g. Ludwig, 1972; Nijenhuis et al., 1998). According to this theory, the sensory-motor components of defensive motor reactions during traumatic or stressful events are coded into memory and serve as a cue for the activation of negative emotions associated with the trauma (Brown, 2004). Future studies using aversive conditioning of movements with one hand should test whether such mechanisms can result in increased ACC action monitoring. Another predominant theory is based on animal research, showing increased activity of the hypothalamic-pituitary-adrenal (HPA) axis (i.e. increased cortisol levels) to be associated with prolonged freezing responses and increased motor inhibition (e.g. Kalin et al., 1998a,b; Nunez et al., 1996). Deregulations of the HPA-axis were also found in patients with conversion disorder on a dexamethasone suppression test (Tunca et al., 1996). Compared to controls, patients with conversion disorder showed increased post-dexamethasone cortisol levels, which is indicative of impaired suppression of cortisol. Previous research in healthy subjects (Hsu et al., 2003) showed that cortisol administration resulted in altered ACC action monitoring associated with decreased performance on a Stroop task. Future research is needed to investigate whether hyperactivity of the HPA-axis plays a role in the altered ACC action monitoring functions in conversion patients.

In conclusion, the present study is the first to show electrophysiological correlates of ACC action monitoring in patients with conversion paralysis. The findings of increased N2 congruency effects for affected limbs, demonstrate that conversion paralysis is associated with hyperactive pre-response monitoring and support the role of the ACC in CP.

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