



Deficit in late-stage contingent negative variation provides evidence for disrupted movement preparation in patients with conversion paresis



Rebekah L. Blakemore^{a,b,*}, Brian I. Hyland^{c,b}, Graeme D. Hammond-Tooke^{d,b}, J. Greg Anson^e

^a School of Physical Education, Sport and Exercise Sciences, University of Otago, Dunedin 9016, New Zealand

^b Brain Health Research Centre, University of Otago, Dunedin 9016, New Zealand

^c Department of Physiology, University of Otago, Dunedin 9016, New Zealand

^d Department of Medicine, University of Otago, Dunedin 9016, New Zealand

^e Department of Sport and Exercise Science, and Centre for Brain Research, University of Auckland, Auckland 1142, New Zealand

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ABSTRACT

Conversion paresis is the presence of unexplained weakness without detectable neuropathology that is not feigned. To examine the ‘abnormal preparation’ and ‘disrupted execution’ hypotheses proposed to explain the movement deficits in conversion paresis, electroencephalographic, electromyographic and kinematic measures were recorded during motor preparation and execution. Six patients with unilateral upper limb conversion weakness, 24 participants feigning weakness and 12 control participants performed a 2-choice precued reaction time task. Precues provided advance information about the responding hand or finger. Patients and feigners demonstrated similar diminished force, longer movement time and extended duration of muscle activity in their symptomatic limb. Patients showed significantly suppressed contingent negative variation (CNV) amplitudes, but only when the symptomatic limb was precued. Despite the similarity in performance measures, this CNV suppression was not seen in feigners. Diminished CNV for symptomatic hand precues may reflect engagement of an inhibitory mechanism suppressing cortical activity related to preparatory processes.

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1. Introduction

Motor conversion disorder is characterised by impaired movement that cannot be explained by an organic neurological cause, inconsistent symptoms (e.g., the motor impairment diminishes with distraction) and reflexes and muscle tone that remain normal (American Psychiatric Association, 1994). It is distinguished from other “non-organic” movement disorders by a lack of conscious intention to deceive (Bass, 2001). Symptoms are thought to arise from underlying psychological stressors such as trauma or conflict,

but the neural mechanisms remain unknown (Scott & Anson, 2009; Vuilleumier, 2005).

The neurobiology underpinning conversion paresis and other conversion disorders remains mysterious. There are two particular aspects that need to be understood: first, what is the brain mechanism underlying the generation of abnormal activity in output of the motor control system that leads to, in the case of conversion paresis, weakened movement; and second, how is it that the patient is unconscious of the origin of their symptoms? In a recent paper, we provided evidence of disrupted early components in the sensory evoked potential in a simple reaction time (RT) task in a group of conversion paresis patients. Such changes in evoked potential amplitude did not occur in participants consciously feigning the same level of movement deficit (Blakemore, Hyland, Hammond-Tooke, & Anson, 2013). We proposed that these evoked potential changes may reflect processes associated with suppressing conscious awareness of self-agency.

In the present report, we address two proposed hypotheses that account for the generation of the abnormal motor command in

* Corresponding author. Present address: Department of Neuroscience, University Medical Centre, University of Geneva, 1 rue Michel-Servet, 1211 Geneva, Switzerland. Tel.: +41 22 379 5326; fax: +41 22 379 5402.

E-mail addresses: rebekah.blakemore@otago.ac.nz (R.L. Blakemore), brian.hyland@otago.ac.nz (B.I. Hyland), graeme.hammond-tooke@southernhb.govt.nz (G.D. Hammond-Tooke), g.anson@auckland.ac.nz (J.G. Anson).

motor conversion disorder in a choice reaction time task. The 'disrupted execution' hypothesis proposes that the intent to move and ability to generate motor programs is intact but 'lower' cortical areas, such as the motor cortex are inhibited so that the signal for motor execution from motor cortex is disrupted or delayed (Marshall, Halligan, Fink, Wade, & Frackowiak, 1997; Tiihonen, Kuikka, Viinamäki, Lehtonen, & Partanen, 1995). In contrast, the second hypothesis attributes impaired motor output to 'abnormal preparation' resulting from deficits in the genesis of a motor program during motor preparation (Spence, Crimlisk, Cope, Ron, & Grasby, 2000; Vuilleumier et al., 2001). The 'abnormal preparation' and 'disrupted execution' hypotheses have been derived from neuroimaging studies, based on whether activity was altered in motor preparatory or execution cortical regions. However, in most of these studies there was no requirement for overt movement, so determining whether the patients were actually in 'preparation' or 'attempted execution' phases is difficult. Preparation and execution are sequential events during movement performance, thus accurately resolving whether neural deficits affect preparation and/or execution requires investigation that is able to define the time-course of these components.

Evidence from previous behavioural studies has provided conflicting results regarding delays in preparing and/or executing movement in conversion paresis. Longer RTs without changes in movement duration were found in motor imagery tasks (Roelofs et al., 2001; Roelofs, van Galen, Keijsers, & Hoogduin, 2002), yet impairments in both RT and movement time have been reported in a simple RT task (Blakemore et al., 2013). One reason for these discrepant findings is that Roelofs et al. measured RT and movement time from verbal responses. Although verbal responses are commonly used in RT paradigms, they involve various complex processing stages for the initiation and production of speech (Levelt, 2001) typically resulting in less precise measures (with longer RT and larger standard deviations) than those taken from manual measuring instruments (e.g., Feyereisen, 1997). But even with more precise methods of RT measurement, such behavioural data alone cannot completely resolve the preparation versus execution debate because RT cannot be ascribed to one or the other process – RT as measured from imperative stimulus presentation to movement initiation includes processes of muscle activation that can be considered purely related to execution (Weiss, 1965).

To address this we have investigated motor preparation and execution, using electroencephalography (EEG) and a precued RT task (Rosenbaum, 1980), in a group of patients diagnosed with unilateral conversion paresis. Such patients are able to perform voluntary movement tasks with their symptomatic limb albeit weakly, unlike those with conversion paralysis in whom movement is abolished (as studied for example in de Lange, Roelofs, & Toni, 2007; Marshall et al., 1997; Schönfeldt-Lecuona, Connemann, Viviani, Spitzer, & Herwig, 2006; Tiihonen et al., 1995). We contrasted the patient data with healthy control participants, and a group of healthy volunteers feigning paresis.

In the present study we report data from choice RT conditions, in which the precue provided *partial* information about parameters defining the upcoming movement (hand or finger). This experimental manipulation allowed us to specifically address the 'preparation versus execution' debate that underlies explanations for impaired movement in conversion paresis. To further probe movement deficits in conversion paresis we examined concurrent changes in brain activity just prior to movement initiation (i.e., at the end of the preparatory period) by analysing the amplitude of the contingent negative variation. The CNV is a slow surface negativity that develops during the interval between the precue and imperative stimulus of RT tasks (Walter, Cooper, Aldridge, McCallum, & Winter, 1964), and is related to motor preparation. The use of CNV in psychiatry and neurology is well-established

(Tecce & Cattanach, 1987) and measurement of CNV amplitude is well suited to investigate deficits in voluntary movement in conversion disorder. Surprisingly, little empirical research has examined modulation of CNV in patients clinically diagnosed with conversion or somatoform disorders. In one study, Timsit-Berthier, Delaunoy, Koninckx, and Rousseau (1973) measured CNV amplitude in psychotic and neurotic patients and in a control group. The 'neurotic group' consisted of patients with symptoms of depression, phobias, obsessions and "mechanisms of conversion", though whether any of these patients were diagnosed with conversion disorder is unclear. Smaller CNV amplitudes were found in the neurotic patients than in the psychotic patients and controls, however explanations for the disrupted CNV development were lacking.

For the first time in studies of conversion disorder, we simultaneously recorded the CNV with the electromyogram (EMG) to fractionate RT into premotor (preparation) and motor (execution) phases of movement initiation. The results indicate temporally specific modulation of task performance and altered electrophysiological measures in patients compared to healthy controls and healthy controls instructed to feign paresis. Specifically, we report a novel finding that change in cortical preparatory activity was only observed when patients had prior knowledge about movement with their symptomatic limb.

2. Materials and methods

This study was approved by the Lower South Regional Ethics Committee (NAF-2005 v1). All participants provided informed consent.

2.1. Participants

Six patients (4 female; mean age 57 ± 7 years; mean symptom duration 18 ± 14 months) diagnosed with Conversion Disorder according to the criteria in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1994) were investigated. Patients were referred by a Consultant Physician in Neurology at the local hospital following full neurological exam including neuroimaging and offered the opportunity to participate if they met the following inclusion criteria: absence of intracranial abnormalities, pain-free unilateral upper limb paresis, and no evidence or history of neurological disease. Table 1 shows the clinical details for each patient. All but one of the patients had left hand paresis (i.e., affecting their non-preferred hand). Because differences in preferred versus non-preferred hand could link to differences in hemispheric functions and might confound CNV measures, data from the one patient with a right hand paresis were excluded from CNV analyses. All patients refrained from taking medication (listed in Table 1) 10 h prior to the experimental session.

For each patient, six sex- and aged-matched healthy volunteers were recruited (36 in total; mean age 54 ± 3 years, 24 female). Healthy volunteers were included if they had no diagnosis of a psychiatric disorder in the past 12 months, no upper limb pain or injuries, and no prior or current neurological disorder. The 36 healthy participants were randomly assigned to one of three groups ($n = 12$, 8 females in each): a control group, a 'Feign_{effort}' or a 'Feign_{resist}' group. These two feigning groups were included to investigate whether different instructions or strategies used to consciously generate weakness give rise to different neural and/or behavioural activity. Thus the feigning groups were differentiated on the basis of specific instructions given to mimic weakness. The Feign_{effort} group were instructed to imagine that their left arm, hand and fingers had become so weak, such as following a severe injury to the limb, that their muscles would be unable to exert a lot of force. The Feign_{resist} group participants were instructed to imagine that their left fingers were moving against a resistance that however hard they tried, they would find it difficult to depress the keys. Because there were no significant differences between the two feigning groups on any dependent measure, including CNV amplitude ($p > .05$), the results from the Feign_{effort} and Feign_{resist} groups were pooled to form one group, referred to as 'feigners' (grand mean CNV waveforms for each feigning group for responses made by the symptomatic and asymptomatic hands are illustrated in Figs. S1 and S2 respectively). For all subsequent analyses, the Group factor therefore compared three groups: patients, controls, and feigners. The difference in average age among the participant groups was not significant ($p = .33$). All participants had normal speech and hearing, normal or corrected-to-normal vision, and were right-handed, as assessed by the Edinburgh Handedness Inventory (Oldfield, 1971), with a mean laterality quotient of 88% for the conversion paresis patients and 81% for the non-patient participants.

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