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Response Inhibition in Motor Conversion Disorder

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Abstract

Conversion disorders (CDs) are unexplained neurological symptoms presumed to be related to a psychological issue. Studies focusing on conversion paralysis have suggested potential impairments in motor initiation or execution. Here we studied CD patients with aberrant or excessive motor movements and focused on motor response inhibition. We also assessed cognitive measures in multiple domains. We compared 30 CD patients and 30 age-, sex-, and education-matched healthy volunteers on a motor response inhibition task (go/no go), along with verbal motor response inhibition (color-word interference) and measures of attention, sustained attention, processing speed, language, memory, visuospatial processing, and executive function including planning and verbal fluency. CD patients had greater impairments in commission errors on the go/no go task ($P < .001$) compared with healthy volunteers, which remained significant after Bonferroni correction for multiple comparisons and after controlling for attention, sustained attention, depression, and anxiety. There were no significant differences in other cognitive measures. We highlight a specific deficit in motor response inhibition that may play a role in impaired inhibition of unwanted movement such as the excessive and aberrant movements seen in motor conversion. Patients with nonepileptic seizures, a different form of conversion disorder, are commonly reported to have lower IQ and multiple cognitive deficits. Our results point toward potential differences between conversion disorder subgroups.

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Keywords

conversion disorder; psychogenic movement disorder; response inhibition; IQ; cognition

Conversion disorders (CDs) are unexplained neurological symptoms presumed to be related to underlying psychological issues. The neurobiological basis of these disorders is not well understood. Most studies have focused on conversion paralysis or the absence of movement, hypothesizing impairments in the generation of motor intention or conceptualization¹⁻³ or that motor intention is intact but execution is disrupted.^{4,5} A recent study suggests that in conversion paralysis, the neural correlates associated with motor inhibition are not impaired.⁵ In the current study, we focused on patients with positive motor phenomena or aberrant or excessive movements such as tremor, dystonia, tics, or chorea.⁶ We asked whether the ability to inhibit unwanted motor responses is impaired. In a recent behavioral study, motor CD patients demonstrated differences in the subjective sense of timing surrounding voluntary movement compared with healthy controls.⁷ These differences in the sense of when movement was willed and when it occurred were shown for normal voluntary movements rather than the patient's abnormal conversion movements, suggesting that the underlying abnormality in motor CD affects all movements, voluntary and involuntary. Thus, we elected to study voluntary motor response inhibition in motor conversion disorder, assuming that the demonstration of abnormalities in voluntary motor inhibition may reflect abnormalities in conversion movements. We hypothesized that motor CD patients might have specific abnormalities in motor response inhibition, which was tested using a go/no go task. A common neural region implicated in motor response inhibition tasks including both the go/no go and stop signal tasks is the presupplementary motor area.⁸ We have previously shown abnormal activation in the supplementary motor complex (SMC) during an affective task⁹ and a motor initiation task,¹⁰ thus providing further rationale for a possible involvement of motor response inhibition.

The go/no go task has been shown to be widely distributed throughout the brain, with contributions from nonmotor areas.^{11,12} As such, poor performance may result from attentional or other cognitive deficits. In another subtype of conversion disorder, nonepileptic seizure (NES), patients have levels of cognitive impairment similar to those with epileptic seizures and have greater impairment relative to healthy controls.¹³⁻¹⁵ Such assessments have not yet been conducted in motor conversion disorder patients. Thus, we also assessed other general cognitive domains to determine if differences in motor response inhibition might be part of a greater network of cognitive deficits in patients with motor CDs. We have focused on tasks consistent with the major domains studied in NES patients, which show a low-average or borderline IQ^{13,16-18} along with impairments in multiple cognitive domains in processing speed, language, verbal and nonverbal memory, executive function (planning and verbal fluency), and visuospatial processing.^{13,15,18-22}

Patients and Methods

Subjects

Patients with CD were recruited from the outpatient Human Motor Control Section clinic at the National Institute of Neurological Disorders and Stroke, National Institutes of Health (NIH). Inclusion criteria for the behavioral study included diagnostic confirmation of “clinically definite” psychogenic movement disorder by a movement disorders neurologist (M.H.) and of conversion disorder by a psychiatrist (V.V.); no current major depression of moderate severity (BDI>20) or serious psychiatric, medical or neurological illness; no history of traumatic brain injury; and being at least 19 years old. The diagnosis of “clinically definite” psychogenic movement disorder was made after a detailed history, extensive neurological examination, and the performance of all necessary and reasonable tests, including but not limited to magnetic resonance imaging (MRI), electroencephalogram, and electromyogram testing, to rule out other diagnoses for the motor signs.²³ Age- (± 5 years) and sex-matched healthy volunteers were recruited from the NIH healthy volunteer database. All subjects had been referred from a general neurologist to the Human Motor Control Section specialty clinic. Psychiatric comorbidity was screened using the Structured Clinical Interview for DSM-IV Axis I Disorders (by V.V. and R.A.),²⁴ and neuropsychological testing was conducted (by E.W.). Subjects also completed the Beck Depression and Anxiety Inventories.^{25,26} The study was approved by the Institutional Review Board of the NIH, and all subjects signed informed consent.

Behavioral Tasks

Motor response inhibition was measured using Conner’s Continuous Performance Test II task, a computerized 14-minute visual performance task in which the subject responds to rapidly presented nontarget letters and inhibits motor responding to an infrequently shown target letter.²⁷ The 360 trials are divided into 20 blocks with interstimulus intervals varying between 1, 2, and 4 seconds. The intervals are counterbalanced between blocks. The task duration is 14 minutes. Response inhibition is measured by the number of commission errors. The task also measures motor reaction time, motor perseveration (defined as responding within 100 ms of letter onset), and sustained attention or vigilance (as measured by change in hit reaction time and standard error over blocks).

Because these patients had generalized abnormal movements, such as myoclonus or tremor involving multiple limbs and/or the neck and trunk, no attempt was made to have patients use an “unaffected” hand for key presses. They were instructed to use their dominant hands and to stop if they felt that they could not participate.

Neuropsychological Battery

General intellectual functioning was estimated using the Weschler Test of Adult Reading (WTAR), which assesses visual, performance, and full-scale intelligence quotient (IQ) using a reading test of 50 words.²⁸ Processing speed was assessed using the Weschler Adult Intelligence Scale Symbol Search test, which assesses the speed of symbol matching appearing in different groups, and the Digit Symbol test, which presents pairs of digits and symbols and requires pairings of additional digits and symbols.²⁹ Tests for memory function

included the Hopkins Verbal Learning Test,³⁰ which measures verbal memory assessing immediate recall, delayed recall, and delayed recognition, and the Brief Visuospatial Memory Test,³¹ which measures visuospatial memory assessing both immediate and delayed recall of 6 geometric figures. Planning and problem solving was assessed using the Delis–Kaplan Executive Function System (D-KEFS) Tower test, which measures spatial planning, rule learning, inhibition, and establishing and maintaining cognitive set.³² Verbal fluency was assessed using the D-KEFS verbal fluency test, which assesses the ability to produce verbal responses fluently in accordance with set rules in a 1-minute period and tests both phonemic (letter fluency) and concepts (category fluency). The D-KEFS color-word interference test assesses the ability to inhibit an overlearned prepotent verbal response in accordance with set rules (eg, inhibit reading the colored word rather than naming the color of the word) and tests verbal inhibition, simultaneous processing, and cognitive flexibility. The Boston Naming Test measures object naming based on line drawings.³³ To assess visuospatial processing and parietal function, we used the judgment of line orientation from the Repeatable Battery for the Assessment of Neuropsychological Status, in which subjects select from a series of lines the one that corresponds to the same orientation as the target stimulus.³⁴

Statistical Analysis

Tests of normality were conducted using the Shapiro–Wilks test; variables of $P < .05$ were normalized with log transformation. Data of subjects scoring greater than 3 standard deviations from the mean were excluded from analysis. Variables were compared using the independent t test, and $P < .002$ was considered significant following Bonferroni correction for multiple comparisons. Significant variables were tested using univariate analysis with the Beck Depression Inventory and Beck Anxiety Inventory and CPT omission and hit reaction time and standard error block change scores as covariates of no interest to control for depression, anxiety, attention, and vigilance as confounders.

Results

Thirty CD patients were compared with 30 age- and sex-matched healthy volunteers (HVs; Table 1). The mean WTAR full-scale IQ in CD patients was 107.55 (SD, 10.2). The duration of abnormal movements in CD patients was 6.13 years (SD, 5.87 years). Movement presentations were as follows: tremor, 73%; dystonia not fixed, 27%; gait difficulty, 35%; chorea, 4%; and myoclonus, 12%. Medication use was as follows: antidepressants, 31%; benzodiazepines, 35%; levodopa or dopamine agonist, 8%; anticonvulsant, 23%; antipsychotic, 4%; and muscle relaxant, 4%. Psychiatric diagnoses were as follows: mild major depressive episode (BDI < 20), $n = 2$; dysthymia, $n = 1$; generalized anxiety disorder, $n = 4$; phobia, $n = 2$.

One CD subject scored greater than 4 standard deviations above the mean on the CPT measures and was excluded from analysis (omission errors: mean, 51.20; SD, 22.3; subject 1: 187.55). CD patients made more commission errors (errors in withholding responding) on the go/no go task relative to HVs ($P = .001$; Table 2). There were no differences in hit reaction time, perseveration, measures of attention or fatigue such as omission errors, or

measures of sustained vigilance such as differences in hit reaction time or standard error between blocks, suggesting the finding was specific to motor response inhibition. The level of significance for commission errors did not change with inclusion of attention and vigilance scores as covariates of no interest ($P=.001$). The level of significance for commission errors also did not change with inclusion of depression or anxiety scores as covariates of interest ($P=.002$). There were no significant differences in measures of attention, vigilance, verbal and visual memory, language, visuospatial processing, or planning and verbal fluency following correction for multiple comparisons (Table 3). The association with impaired motor response inhibition remained significant in a subanalysis performed only with patients with tremor and matched HVs ($P=.001$).

Discussion

Motor CD patients have a specific impairment in the ability to inhibit motor responses relative to healthy volunteers, suggesting a potential mechanism underlying the expression of aberrant conversion motor phenomena. There were no significant differences in general cognitive function between outpatient CD patients and healthy volunteers including in a task measuring inhibition of verbal prepotent responses and the domains of attention, vigilance, processing speed, memory, language, visuospatial processing, and executive processes such as planning and fluency. Studies in NES populations frequently show a lowaverage or borderline IQ^{13,16–18} along with impairments in multiple cognitive domains.^{13,15,18–22} That we did not demonstrate abnormalities in other cognitive domains stands in marked contrast to the findings of multiple cognitive deficits in patients with NES and suggests that motor response inhibition impairment in motor CD patients is not a result of other cognitive deficits.

Motor Response Inhibition

The SMC, including the pre-supplementary motor area (pre-SMA) with connections to prefrontal regions and the SMA proper, has been implicated in motor inhibition.³⁵ We have previously shown that arousing stimuli increases functional connectivity between the amygdala and SMA in motor conversion patients compared with healthy controls³⁶ and that these patients show relative SMC hypoactivity compared with controls during motor initiation.¹⁰ Although studies of response inhibition as measured using the stop signal task have often focused on the right inferior frontal gyrus (rIFG) as the “seat” of inhibition, recent data have challenged this model. By using a version of the stop signal task that allowed for differentiation of brain activation associated with appropriate response inhibition from that associated with the need to pay attention to changing stimuli, Sharp et al showed that only pre-SMA area activation was associated with inhibition alone.³⁷ Similarly, in an fMRI study of the stop signal task in which the responses to the stop signal cue were varied, the rIFG was shown to be more relevant to the detection of salient, or task-relevant, cues irrespective of any form of motor response.³⁸ In a patient with subdural electrodes covering the pre-SMA and rIFG, pre-SMA activity was found to precede rIFG activity when preparing to stop and during stopping in the go/no go task, suggesting a potential role for proactive stopping.³⁹ Although the networks underlying the go/no go and stop signal tasks differ, the pre-SMA is an area of overlap in studies directly comparing the 2 tasks.⁸ Here we

have shown specific impairment of response inhibition of prepotent motor responses in a patient group shown to have qualitative differences in SMC activation for movement. Although our results suggest a mechanism by which motor CD patients might be less able to inhibit unwanted movements during movement selection, this does not explain how such movements become repetitive and debilitating for these patients. Investigators have posited the role of “top-down” processes in these patients as inappropriately interpreting random movement as meaningful.⁴⁰ In this way, a system of beliefs regarding volition is created and constantly updated, both misinterpreting movements as involuntary and bringing about further movement via greater attention.⁴¹ Further research is needed to demonstrate how these systems of motor control interact in motor CD patients.

Although we controlled for depressive and anxiety differences, these findings may also represent premorbid differences in the patient population in characteristics such as underlying impulsivity, personality differences, and precipitants such as stressors. A recent study demonstrated impulsive decision making in psychogenic movement disorder with a tendency to “jump to conclusions,” or make rapid decisions without adequate consideration of the evidence.⁴² This is a form of decisional impulsivity known as reflection impulsivity, whereas the go/no go task measures motor impulsivity. Whether our findings might reflect impairments in impulsivity rather than reflect mechanisms underlying the functional motor signs remains to be established. Further studies to explore these influences are indicated.

We tested both motor response inhibition and the Stroop to test measures of response inhibition. The Stroop interference task tests the ability to inhibit a competing prepotent word and provides a measure of response inhibition to conflict, response selection, selective attention, and cognitive flexibility. That we demonstrated specific abnormalities on motor response inhibition suggests any possible inhibitory deficit is not generalized and might be specific to the motor domain. The capacity to inhibit a motor response compared with inhibiting a response in the context of conflict might differ in motor CD patients. The go/no go task measures externally cued motor response inhibition of a response that has not yet started. Other tasks assessing motor inhibitory responses are indicated including the stop signal task, which measures externally cued inhibition of an ongoing response and tasks assessing externally cued nonconscious motor inhibition.³⁵ Internally cued motor response inhibition would also be of interest but may be more difficult to study. Other studies focusing on inhibition of prepotent responses outside the motor domain such as random number generation or the Hayling sentence completion test are also indicated.

General Cognitive Function and IQ

Our study showed that motor CD patients have a full-scale IQ in the average range, and we did not demonstrate differences in multiple cognitive domains between motor CD patients and matched healthy volunteers. In contrast, studies in NES populations have frequently shown a low-average or borderline IQ.^{13,16–18} Studies of cognitive function in patients with NES compared with those with epileptic seizure have had mixed results, with many studies reporting similar impairments in multiple domains, although some studies report better performance in NES patients.^{13,15,18–22} These cognitive studies in the NES population are confounded by a high prevalence of comorbid neurological insults. For instance, in 1 of

these studies, 58% of the NES patients had a history of closed-head injury,¹³ and in another study, 80% of the NES patients had a history of major birth trauma, head injury, or central nervous system infections.⁴³ Possible explanations such as decreased motivation or effort have also been put forward to explain these findings.^{13,14,44} A recent study of female NES patients demonstrated greater impairments in patients with NES compared with patients with epileptic seizure in attention, working memory, and information-processing speed, with mean scores below the average range (9th to 24th percentiles). The authors attribute these impairments to differences in mood and anxiety scores.⁴⁵ The current study focused on an outpatient population with primary motor complaints and controlled for a history of traumatic brain injury, attention, sustained attention, depression, and anxiety. The lack of differences in general cognitive function in the current study may be related to differences in the sample population, as previous studies may have focused on nonepileptic seizure patients and more severe inpatient populations and may have included patients with traumatic brain injuries and did not necessarily control for differences in IQ scores, attention, depression, or anxiety. Alternatively, the mechanisms underlying motor conversion disorders may also differ from that of NES.

Limitations

There are several limitations in the current study. We included a range of conversion motor phenomena in this study, as our hypothesis was that patients with hyperkinetic motor conversion would have impaired motor response inhibition even for normal voluntary movements. The combination of patients with multiple manifestations of hyperkinetic motor CD may limit generalizability of the findings to patients with hypokinetic motor conversion or other types of conversion. A subanalysis of patients with tremor confirmed the finding of impaired motor inhibition, but sample size limited further differentiation of data by movement type. Stringent correction for multiple comparisons was performed. Findings such as lower category fluency ($P=.04$) and judgment of line orientation ($P=.11$) might be considered a trend but were not considered significant as they might represent type I errors. Larger sample sizes are indicated to evaluate these measures and particularly to address more subtle differences. There were significant differences between groups in depression and anxiety scores, which was previously observed with motor CD.³⁶ However, we controlled for both depression and anxiety and covariates of no interest, and furthermore, the pattern did not fit that of cognitive deficits in depression, which emphasize processing speed deficits and, more variably, cognitive inflexibility, working memory deficits, and attentional and vigilance deficits.⁴⁶ Deficits in attention are likely given the greater attentional bias observed in patients with motor CD.⁴¹ The sample size was also limited; larger sample sizes may be indicated to detect more subtle deficits in these domains. Patients were on multiple kinds of centrally acting medications, but sample size prohibited analyzing the data with regards to medication effects. Although the difficulty associated with performing this task in the context of an ongoing involuntary movement disorder also could be a potential confound, very few trials were actually affected by involuntary movement and had to be excluded. The relative success of coordinating movement for this task points toward the distractibility often demonstrated in CD patients.

Conclusions

There appears to be a specific deficit in motor inhibition in the selection of motor responses in patients with motor conversion disorder that may underlie the development of excessive or aberrant conversion movements. We have further shown that patients with motor CD have IQ levels in the average range and intact general cognitive functioning, suggesting possible mechanistic differences from patients with NES.

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TABLE 1

Characteristics of subjects with conversion disorder and of healthy volunteers

	CD	HV	<i>t</i>	<i>P</i>
Number	30	30		
Sex (F), n (%)	20 (67)	20 (67)		
Age	47.98 (13.61)	50.62 (12.80)	-0.89	0.38
Depression BDI	9.11 (5.36)	3.12 (3.23)	4.88	<.001
Anxiety BAI	8.65 (8.28)	1.88 (1.92)	4.06	<.001
IQ WTAR VIQ ^a	107.24 (9.99)	108.65 (10.76)	-0.49	0.63
WTAR PIQ ^a	106.07 (7.71)	106.97 (8.71)	-0.38	0.71
WTAR FSIQ ^a	107.55 (10.20)	108.61 (10.86)	-0.35	0.73

^aData were log10-transformed prior to parametric analyses. CD, conversion disorder; HV, healthy volunteers; F, female; BDI, Beck Depression Inventory; BAI, Beck Anxiety Inventory; WTAR VIQ, Wechsler Test of Adult Reading Verbal Intelligence Quotient; PIQ, performance IQ; FIQ, full-scale IQ.

TABLE 2

Continuous performance task scores

	CD	HV	t	P
CPT (n)	30	30		
Motor inhibition	Commission errors ^a 50.22 (8.61)	43.15 (7.91)	3.31	0.001
Reaction time	Hit reaction time 49.32 (10.15)	51.44 (8.32)	0.88	0.38
Perseveration	Perseveration ^a 50.44 (13.99)	47.32 (7.11)	1.09	0.28
Attention	Omission errors ^a 49.10 (10.83)	47.26 (9.16)	0.71	0.48
Vigilance	Hit RT block change 48.22 (11.41)	46.93 (10.23)	0.46	0.65
	Hit SE block change 53.71 (10.25)	54.33 (8.94)	0.25	0.80

^aData were log10-transformed prior to parametric analyses. The direction of abnormal measures in the Connor's Continuous Performance Task are indicated: omission errors, higher; commission errors, higher; hit reaction time, perseveration, higher; hit reaction time, higher, standard error block change, higher, CD, conversion disorder; HV, healthy volunteers; CPT, Connor's Continuous Performance Test; RT, reaction time; SE, standard error.

TABLE 3

General cognitive measures

	CD	HV	t	P
Number	30	30		
Processing speed				
WAIS-III symbol search ^a	10.13 (2.08)	10.69 (3.21)	-0.17	0.87
WAIS-III Digit-Symbol	10.10 (2.73)	10.45 (3.56)	-0.48	0.94
Planning				
D-K Tower test	10.15 (4.18)	9.82 (2.94)	0.41	0.69
Verbal inhibition				
D-K Color-Word Interference Inhib ^a	9.36 (3.57)	9.64 (3.52)	-0.11	0.91
Inhib/Switch ^a	9.51 (3.86)	9.97 (3.70)	-0.48	0.63
Color naming ^a	8.82 (3.37)	8.67 (3.32)	0.07	0.95
Word reading ^a	8.71 (3.91)	9.54 (3.28)	-0.96	0.34
Verbal fluency				
D-K letter fluency	10.79 (3.13)	11.31 (4.56)	-0.59	0.56
D-K category fluency	10.26 (2.84)	12.02 (4.45)	-2.08	0.04
Language				
Boston Naming Test	45.39 (11.64)	43.87 (10.64)	0.58	0.57
Visual memory				
BVMt learning	52.74 (13.49)	52.50 (14.19)	0.07	0.94
BVMt recall ^a	41.75 (13.59)	45.21 (16.19)	-0.65	0.5
Verbal memory				
HVLt retention ^a	46.37 (10.71)	45.59 (13.04)	0.45	0.66
HVLt delayed recall ^a	47.44 (8.73)	44.92 (13.55)	1.24	0.22
HVLt recognition ^a	49.71 (9.47)	48.72 (9.63)	0.36	0.72
Visuospatial function				
WAIS-III Digit-Symbol	10.10 (2.73)	10.45 (3.56)	-0.48	0.94
RBANS JLO, total score	16.11 (3.24)	14.68 (3.59)	1.56	0.11
RBANS JLO, z score	28.20 (18.24)	28.56 (19.90)	0.1	0.92

^aData were log10-transformed prior to parametric analyses. CD, conversion disorder; HV, healthy volunteers; WAIS, Wechsler Adult Intelligence Scale; D-K, Delis-Kaplan Executive Function System; BVMt, Brief Visuospatial Memory Test; HVLt, Hopkins Verbal Learning Test; RBANS, Repeatable Battery for the Assessment of Neuropsychological Status; JLO, judgment of line orientation.