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## CLINICAL PSYCHOLOGY & NEUROPSYCHOLOGY | RESEARCH ARTICLE

# Cognitive rehabilitation treatment for mental slowness in conversion disorder: A case report

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**Abstract:** Cognitive rehabilitation treatment (CRT) has been described in patients with brain injury, but it has not been attempted in cases of cognitive dysfunction without organic cause. This case report describes CRT of neurocognitive impairment in a 54-year-old female patient with conversion disorder (CD). She experienced difficulties with regard to speaking, motor function, and pain symptoms, which developed after stressful life circumstances. Baseline neuropsychological assessment (NPA) showed mental slowness and impaired (working) memory. Time Pressure Management (TPM) was used as CRT to teach the patient a compensatory strategy to overcome mental slowness in 12 sessions. During treatment, physical symptoms were monitored with the Physical Symptom Questionnaire (LKV), and mental slowness with the Mental Slowness Questionnaire (MSQ). After treatment, the LKV score dropped from 85 to 47, indicating 54% treatment response. Mental slowness showed improvement based on the MSQ and was confirmed by an NPA after treatment. Other neurocognitive functions improved as well and the motoric CD symptoms subsided. This case report suggests that improvement of mental slowness, as well as motor CD symptoms, can be achieved by TPM in non-organic neurocognitive impairment in CD. This finding has not been described in the literature. Further research is warranted to explore the efficacy of TPM in CD.

### ABOUT THE AUTHORS

Lars de Vroege works as a neuropsychologist at the Clinical Centre of Excellence for Body, Mind and Health situated in Tilburg, the Netherlands. He also works as a PhD student at the Tilburg School of Social and Behavioral Sciences at the Department Tranzo under the supervision of Prof. Christina M. van der Feltz-Cornelis, MD, PhD. He is interested in validation research, alexithymia, cognitive functions, complexity, and Type D personality in patients with somatic symptom and related disorders (SSRD). His dissertation is on characteristics of patients with SSRD, with a specific focus on alexithymia and cognitive functioning. Based upon the results of his validation studies, questionnaires developed for assessing somatic symptoms showed only moderate validity. Hence, the exploration of other characteristics of patients with somatic symptom and related disorders is clinically relevant and might be better suited to describing this specific patients group and might lead to tailor-made treatment options.

### PUBLIC INTEREST STATEMENT

Patients with conversion disorder suffer from significant distress and pseudoneurological symptoms on motor or sensory function that is incompatible with neurological disorder. These symptoms can occur either with or without a psychological stressor. Previous studies yielded only limited effectiveness of current treatment modes. As there is some evidence for cognitive impairments in conversion disorder, and they seem to play a role in enhancing the conversive symptoms and on treatment adherence in clinical practice, we report a case in which we explore outcome of a treatment option that is already used in traumatic head injuries to overcome these kinds of impairments, the so-called cognitive rehabilitation treatment. The results showed that cognitive impairment decreased substantially and the conversion disorder went into remission. In view of the high burden of disease, this is a highly relevant finding that opens a new avenue of possibilities for treatment of patients with conversion disorder. Further research in clinical settings is warranted.

**Subjects: Clinical Neuropsychology; Psychiatry & Clinical Psychology - Adult; Neuropsychiatry**

**Keywords: conversion disorder; neurocognitive impairment; cognitive rehabilitation treatment; time pressure management; pain**

### 1. Introduction

Conversion disorder (CD), as stated in the Diagnostic and Statistical Manual of Mental Disorder (DSM)-V (American Psychiatric Association, [APA], 2013), includes one or more deficits affecting either motor or sensory function, that are incompatible with clinically recognized neurological or medical conditions, that cannot be explained by any other medical or mental disorder and cause clinically significant distress or impairment in occupational, social, or other important areas of functioning. The symptoms or deficits can be a sense of weakness or paralysis, abnormal movement (e.g. tremor or myoclonus), swallowing or speech symptoms, seizure-like attacks, memory loss or anesthesia, and special sensory symptoms or mixed symptoms. CD can exist with or without a psychological stressor and is considered persistent if symptoms are present for over six months.

Treatment interventions for CD have been scarcely evaluated in Randomised Controlled Trials (RCT) so far, although a Cochrane Review in 2005 (Ruddy & House, 2005) that found insufficient evidence for psychosocial interventions in CD stated that randomized studies are possible in this field. Kroenke stated in 2007 that evidence for the effectiveness of interventions in CD was lacking, with two RCTs evaluating the effect of hypnosis showing limited results (Kroenke, 2007). A systematic review evaluating the effectiveness of a drug interview in the treatment of CD concluded that although there were some indications of a possible positive effect, especially in the use of suggestion and the occurrence of emotional catharsis during the interview, the research was of poor quality (Poole, Wuerz, & Agrawal, 2010). Two systematic reviews of the effects of physiotherapy found no RCT establishing its effectiveness in adults (Nielsen, Stone, & Edwards, 2013) or children with CD (FitzGerald, Southby, Haines, Hough, & Skinner, 2015). Despite this lack of evidence, clinical practice for CD has so far included multidisciplinary treatment, including psychosocial interventions and physiotherapy and sometimes suggestion or hypnosis. Therefore, there is certainly room for improvement and a need for new treatment modes for CD. A candidate for such an innovative approach might be focusing on the cognitive impairments in CD, as motor functions require adequate cognitive functioning, especially in terms of planning.

So far, studies exploring cognition in CD have reported impairment of executive function (EF) in combination with memory impairment (Brown, Nicholson, Aybek, Kanaan, & David, 2013; Kozłowska et al., 2015) or impaired visuospatial function, memory, and attention (Demir, Celikel, Taycan, & Etikan, 2013; Kozłowska et al., 2015), or standalone memory impairment (Fargo et al., 2004). There are a limited number of studies exploring cognition in CD. Moreover, the studies at hand examined a selection of neurocognitive functions, which precludes the possibility of drawing conclusions on whether impaired memory is caused by mental slowness or solely an impairment of verbal memory. This is a major limitation within the present literature. Furthermore, a variety of tests conducted by different studies result in ambiguous and inconsistent findings with regard to neurocognitive impairment in CD. The role of neurocognitive impairment in CD has received too little research attention, and this is of clinical relevance in view of developing possible treatment options for cognitive impairment in CD, such as cognitive rehabilitation treatment (CRT).

CRT is already used in patients who suffer from cognitive impairment due to stroke or traumatic brain injury. Common symptoms after stroke and traumatic brain injury include mental slowness (Ballard et al., 2003; Gerritsen, Berg, Deelman, Visser-Keizer, & Jong, 2003) that may lead to fatigue and altered mood (Hochstenbach, Mulder, van Limbeek, Donders, & Schoonderwaldt, 1998; Rasquin, Lodder, Ponds, Winkens, & Jolles, 2004). A CRT that has been proven beneficial for mental slowness in stroke patients (Winkens, van Heugten, Wade, Habets, & Fasotti, 2009) is Time Pressure Management (TPM) (Fasotti, Kovacs, Eling, & Brouwer, 2000).

TPM involves a cognitive strategy that compensates for the consequences of mental slowness in daily life (e.g. preparing a meal or during conversations). The essence of TPM is that patients allow themselves to take the time needed to deal with tasks. In that sense, TPM leads to retraining or acquiring compensatory strategies so that neurocognitive symptoms can be reduced (Ben-Yishay & Diller, 1993). However, the effect of CRT in general, or TPM in particular, as a treatment option for neurocognitive impairment in CD has not yet been explored.

The application of CRT to a specific neurocognitive symptom, as in TPM, enables a clinician to tailor treatment specifically to the patients' neuropsychological profile. Laatsch and Stress (2000) described how treatment outcomes can be maximized in patients with stroke or traumatic brain injury by taking into account and focusing on neurocognitive symptoms. Such an approach in patients with CD warrants a description of neurocognitive symptoms of patients with CD but studies exploring these symptoms are limited in number. However, patients do present themselves with neurocognitive impairment in clinical practice. One can thus argue that they might benefit from CRT tailored to resolve the specific impairment found by neuropsychological assessment (NPA). However, the applicability and effect of CRT in CD has not been studied. This case report aims to do so.

In this case report, we describe a 54-year-old woman with severe neurocognitive symptoms who presented herself at our clinic in 2013. Her symptoms had developed since an episode in 2010 in which she had presented herself at a neurology department with Transient Ischemic Attack (TIA) like symptoms that were deemed psychogenic after neurological assessment, including magnetic resonance imaging (MRI). She was diagnosed with CD and received treatment for her neurocognitive symptoms in our outpatient clinic with good results. This case report (a) supports the use of NPA in patients with CD for assessing neurocognitive impairment, followed by selecting a fitting CRT, (b) presents preliminary evidence that the severity of mental disorders dissolves after TPM, and (c) shows that the opportunity to evaluate and treat neurocognitive symptoms in CD should be further explored in future controlled research.

## 2. Case report

### 2.1. History

A 54-year-old woman (JH), living alone and working in a health care facility under stressful conditions which led to fatigue and heart palpitations, was threatened by a patient during her work. The day after that, JH started to experience blurred vision. She thought this blurry vision was a result of the stress she experienced at work. The next morning, she experienced tingling feelings in the left side of her face, left arm, and leg. She also noticed her face was drooping to the left, the left side of her tongue felt numb, and she had started to slur her speech and stutter. She presented herself with these symptoms to a neurological clinic. JH reported that she had already experienced daily headaches and pain in the neck before the event, but that this pain had increased. Subjective neurocognitive symptoms, mainly memory problems, trouble concentrating, and participating in conversations, which resulted in the experience of increased headache, were present at the neurologic assessment as well. Neurological assessment for TIA showed a normal brain computerized tomography (CT) scan, brain nerves, and electrocardiogram (sinus rhythm). Doppler of carotid arteries revealed no abnormalities. An MRI to rule out stroke showed no underlying pathological explanation for the symptoms. The neurologist concluded that the symptoms were psychogenic and referred JH to a psychologist and physiotherapist for treatment. After check-up, two months later, speaking had improved. The psychologist concluded that JH's symptoms could be explained by prolonged exposure to stress at work, which had led to overburdening. The neurocognitive symptoms were still present, for which JH requested further examination. The MRI was repeated but again showed no abnormalities. NPA was suggested by the neurologist, but JH rejected it. She was referred by the neurologist to a psychologist for group therapy. After a few sessions, JH quit her therapy because she felt her symptoms did not decrease.

## 2.2. Presentation at our centre

JH presented herself at the Clinical Centre of excellence for Body, Mind and Health, GGz Breburg (CLGG), Tilburg, the Netherlands in 2013. She reported severe subjective neurocognitive symptoms in both memory and attention, leading to inactivity and fatigue, which became obvious by her missing appointments that she had failed to remember. She experienced continuous pain in the occipital region of her head and in the entire left part of her body. The pain worsened when she became more fatigued. At presentation, the patient stuttered and talked with slurred speech. Physical examination did not show any neurological or other abnormalities. After psychiatric examination, the patient was diagnosed with CD.

## 2.3. Lab results

Lab results of patient JH, exploring hemoglobin level, leukocytes, electrolytes, kidney function, cholesterol, glucose, and Thyroid Stimulating Hormone, showed no abnormalities.

## 2.4. Neuropsychological assessment

At intake, a NPA was done to assess JH's neurocognitive symptoms. The following domains were tested using the accompanying tests. Language was assessed using the Boston Naming Task (Heesbeen & van Loon-Vervoorn, 2001), verbal fluency (Animal naming, 2 min), and letter fluency ("N" and "A", 1 min; Deelman, Koning-Haanstra, Liebrand, & van der Burg, 1981). Working memory was assessed by using the Digit Span of the Wechsler Adult Intelligence Scale—fourth edition (WAIS-IV; Wechsler, 2008), verbal memory by the Rey Auditory Verbal Learning Test (RAVLT; Saan & Deelman, 1986), and the Rivermead Behavioural Memory Test (RBMT; Wilson, Cockburn, & Baddely, 1985) and visual memory with the Rey Osterrieth Complex Figure Test (ROCFT; Osterrieth, 1944). Information processing speed was measured by the Symbol Substitution subtest of the WAIS-IV (Wechsler, 2008), Trail Making Test part A (TMT-A; Reitan, 1992), and the Stroop Color-Word test part I and II (Jensen, 1965; Stroop, 1935). Within the domain of attention, inhibition was assessed using part III of the Stroop Color-Word test (Stroop, 1935) (while controlling for the scores on part I and II of the Stroop), divided attention using the TMT-B (while controlling for performance on TMT-A) and sustained attention using the d2-test (Brickenkamp, 2002). EF was explored using three subtests of the Behavioural Assessment of the Dysexecutive Syndrome (BADS; Wilson, Alderman, Burgess, Emslie, & Evans, 1996), namely the Key search, Zoo map, and Rule learning Task for assessing, respectively, planning, (complex) planning, and rule learning. Malingering was assessed using the Test of Memory Malingering (TOMM; Tombaugh, 1996).

A selection of tests was repeated after treatment with TPM (referred to as T4; the time between T0 and T4 was six months). Parallel tests were used in this NPA when possible, namely for the RAVLT and the ROCFT. With regard to the ROCFT, the parallel Modified Taylor Complex Figure Test (MTCF) (Lezak, Howieson, & Loring, 2004) version was used. All test scores were compared to available normative scores, taking into account age, gender, and educational level.

In order to make use of TPM, Winkens and Fasotti (2010) suggest the use of at least one measurement of mental slowness to assess neurocognitive impairment in this domain during NPA. In this study, we used three measures of mental slowness to assess mental slowness during the NPA: the Symbol substitution subtest of the WAIS-IV, TMT-A, and parts I and II of the Stroop Color-Word Test. The results for the NPA prior and after treatment are shown in Table 1.

NPA at intake showed impaired scores (scores below 6th percentile compared to healthy norms) within the domains of sustained attention, working memory, divided attention, and information processing speed. Furthermore, difficulties (scores below 16th percentile compared to healthy norms) were found in the domains of visual and verbal memory and planning. No signs of malingering were found. During the anamnesis, patient JH reported that she needed more time to achieve subjective normal performances and that she "got lost" when she received a substantial amount of information. These symptoms and neurocognitive impairments resulted in significant fatigue, which led to

**Table 1. JH's performance on the NPA**

Neurocognitive domain	Test	Score at T0	Score at T4 <sup>a</sup>
Language	BNT	166/175	
	Animal naming	37	
	Letter fluency (N + A)	16	
Working memory	Digit span	17**	22
Memory	RAVLT		
	Immediate recall	37*	38
	Delayed recall	6/15**	7/15
	Recognition	28/30	29/30
	RBMT		
	Immediate recall	12.5*	
	Delayed recall	9.5**	
	ROCFT/MTCF <sup>b</sup>		
	Immediate recall	10*	29
	Delayed recall	9**	29
Information processing speed	TMT-A	34	46
	Symbol substitution	37**	47
	Stroop		
	Part I	86**	64**
	Part II	120*	79**
Attention	TMT-B	129**	65
	Stroop		
	Part III	176	154*
	d2	26**	
Executive functioning	Rule learning	4	
	Key search test	3	
	Zoo map test	2	
Malingering	TOMM	50/50	50/50

\*≤16th percentile.

\*\*≤6th percentile compared with normative data.

<sup>a</sup>Parallel versions for the RAVLT and ROCFT are used.

<sup>b</sup>ROCFT was used at T0, MTCF was used at T4.

inactivity. Patient JH told that she was unable to perform household activities or practice sports, something she enjoyed doing before.

After TPM, impairment of information processing speed, working memory, visual/verbal memory, and divided attention dissolved. Patient JH was still impaired with regard to information processing speed, based on her verbal reaction during parts I and II of the Stroop Color-Word Test. Based on part III of the Stroop Color-Word Test, she also experienced difficulties within the domain of attention (inhibition). No signs of malingering were found, as assessed by the TOMM.

### 2.5. Questionnaire assessments

At CLGG, patients are monitored during treatment using Routine Outcome Monitoring (ROM) embedded in a shared decision-making model (Van der Feltz-Cornelis, Andrea, et al., 2014). At intake, they fill in a complete assessment that includes the Patient Health Questionnaire 9 (PHQ-9) that measures depression and is a reliable questionnaire (coefficient alpha = .89) (Kroenke, Spitzer, & Williams, 2001), the General Anxiety Disorder-7 (GAD-7) that is considered a reliable questionnaire (coefficient

alpha = .92) for measuring anxiety (Spitzer, Kroenke, Williams, & Löwe, 2006), the Patient Health Questionnaire 15 (PHQ-15) a reliable questionnaire (coefficient alpha = .80) (Kroenke, Spitzer, Williams, & Löwe, 2010) that measures somatic symptoms (Kroenke, Spitzer, & Williams, 2002), the Physical Symptoms Questionnaire (Dutch abbreviation: LKV) that measures physical symptoms (coefficient alpha = .88) (Van Hemert, 2003), the Brief Pain Inventory (BPI) that reliably measures pain (coefficient alpha = .85) (Tan, Jensen, Thornby, & Shanti, 2004), and the Outcome Questionnaire (OQ; Lambert et al., 2004) that is designed for repeated measurements of client progress while in therapy (coefficient alpha = .91) (de Jong et al., 2007). A selection of questionnaires is based on the outcome of a shared decision-making process with the patient and the clinician and is established during a multidisciplinary meeting. Table 2 shows the scores on the ROM questionnaires at intake and after TPM.

At intake, the ROM scores show high scores on PHQ-15, PHQ-9, and LKV, which indicate, respectively, present somatic symptoms, depression, and physical symptoms. Furthermore, the GAD-7 score is indicative of the absence of anxiety. BPI score of 5 is indicative of the presence of pain during the past week. The OQ (Lambert et al., 2004) is used to measure the outcome of treatment and was 93 at intake. After TPM, the scores of OQ, PHQ-9, LKV, and BPI were substantially lower. In order to monitor improvement during treatment, the patient was asked to fill out selected questionnaires to monitor their improvement from the start of treatment (T1) until the end of TPM treatment (T4). In this case, because of her various physical symptoms, JH was asked to fill out the LKV (Van Hemert, 2003) to monitor physical symptomatology during the course of treatment.

Furthermore, to assess mental slowness subjectively, the Mental Slowness Questionnaire (MSQ; Winkens, Van Heugten, Fasotti, et al., 2009) was used as suggested in the protocol (Winkens & Fasotti, 2010). The MSQ consists of 21 items exploring problems with engaging in activities which are related to mental slowness. For instance, “I can’t follow a story when I listen to the radio or watch TV at the same time” or “It takes me more time to execute work, choir or household activities because I have to think about them more.” Each item is scored on a five-point scale ranging from 0 to 4. Answering options correspond to the frequency of experiencing the complaint (0: this never happens, 1: this happens occasionally, 2: this happens now and then, 3: this happens regularly, 4: this happens often). The maximum score of this subscale is 84 (21 × 4). A severity score is also given for each item on a three-point scale with answers ranging from “Not troublesome,” “A little bit troublesome” to “Very troublesome,” scored 0, 1, and 2, respectively. The maximum score of this subscale is 42 (21 × 2). To conclude, a weighted scale can be calculated by multiplying the score of the frequency scale with that of the severity scale. The maximum score of this weighted subscale is 168 (score on difficulty scale, multiplied by scale of severity, multiplied by the number of questions; that is, 4 × 2 × 21).

At the start of TPM, JH scored 85 on the LKV (physical symptoms were apparent at intake). At T2 and T3, the LKV scores were 84 and 87, respectively. At the end of treatment (T4), the LKV score was 47, which indicates a decrease of 15 in experienced physical symptoms.

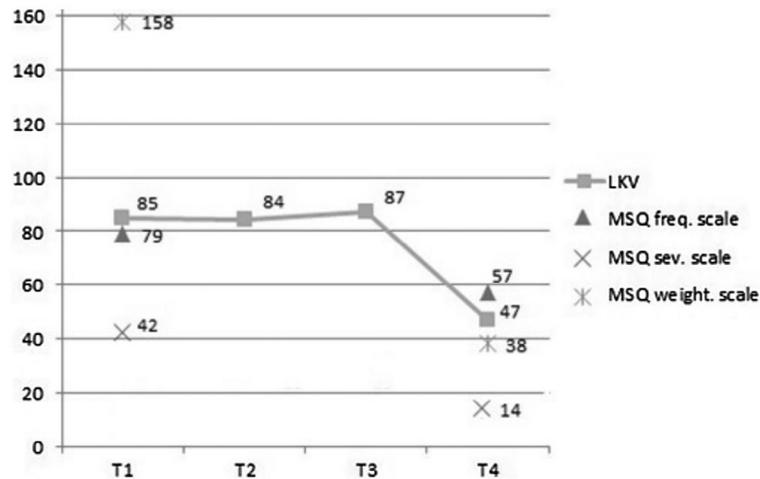
**Table 2. ROM questionnaires at intake and after TPM**

Questionnaire	Scores at intake	Scores after TPM
OQ	93	75
PHQ-15	8	
PHQ-9	11	7
GAD-7	5	
LKV	85	47
BPI	5	3

Abbreviations: OQ: Outcome Questionnaire; PHQ: Patient Health Questionnaire; GAD: General Anxiety Disorder; LKV (Dutch abbreviation): Physical Symptoms Questionnaire; BPI: Brief Pain Inventory.

**Figure 1. Scores on the LKV and MSQ over time during TPM.**

Abbreviations: LKV (Dutch abbreviation): Physical Symptom Questionnaire; MSQ: Mental Slowness Questionnaire; freq.: frequency; sev.: severity; weight.: weighted.  
 Note: MSQ is not assessed during T2 and T3.



At T1, the MSQ frequency score was 79, the MSQ severity score was 42, and the weighted scale of frequency and severity scale was 158. After treatment at T4, all scores decreased substantially to a score of 57 on the MSQ frequency, MSQ severity score dropped to 14 and the score on the MSQ weighted scale was 38. Figure 1 represents the scores on the LKV and MSQ over time during TPM.

**2.6. Intervention—Neuropsychological treatment**

The TPM strategy includes enhancement of awareness, optimization of organization, rehearsal of task requirement, or modification of task environment. These concrete tools are learnt by the patient in three stages. The first stage focuses primarily on increasing awareness of the deficits and the relationship between mental slowness and perceived problems in daily life. In the second stage, the main focus is on acceptance and acquisition of the strategy. Besides relating the (poor) performance of the patient and the concept of time pressure, the strategy was explained and taught to the patient. The final stage focuses on strategy application and maintenance. This stage mainly involves real-life application of the strategy by the patient, evaluating the results of its application and improving the strategy during the treatment session. TPM training in patients with closed head injuries and stroke already appeared to be more beneficial compared to other kinds of training and generalizes to measures of memory and speed function (Winkens, Van Heugten, Wade, Habets, & Fasotti, 2009). Table 3 describes the cognitive strategy used in TPM.

**Table 3. The cognitive strategy in TPM**

Questions to be asked	Main objective to be taught
1. Are there two or more things to be done at the same time for which there is not enough time? If yes: go to step 2, else just do the task	To recognize time pressure in the task at hand
2. Make a short plan of which things can be done before the actual task begins	To prevent as much time pressure as possible
3. Make an emergency plan describing what to do in case of overwhelming time pressure	Dealing with time pressure as quickly and effectively as possible
4. Plan and emergency plan ready? Then use it regularly	Urging the patient to monitor him/herself while using the TPM strategy

Source: Extracted with permission from Fasotti et al. (2000).

### **2.7. Questionnaire assessments—Treatment course and outcome**

After gaining awareness of the situation, during the first stage of TPM, JH learned that mental slowness influenced her life more than she had previously realized. After successfully learning TPM and applying the strategy in real life, JH experienced less frequent and less severe subjective neurocognitive symptoms.

After the last session of TPM, JH described experiencing pain one day every week. This pain was manageable and only limited her in activities that she still found more stressful, for instance, visiting friends in a crowded place. However, she explained being able to visit crowded parties, which she had not been capable of doing before, and stated “I can have control over my life once again.” JH also started to exercise again, although setting boundaries was sometimes still troublesome. Pushing boundaries still resulted in inactivity the next day, but these days decreased in number substantially and recovery speeded up compared to pre-treatment. With regard to the acquired TPM strategy, JH stated that she noticed when she had not optimally applied the strategy. JH reported being able to successfully adjust her behavior to a particular situation and to handle the situation with less mental slowness and stress.

JH also stated being able to cope with mental slowness and to apply the strategy in daily life to overcome hindrances caused by mental slowness, which was confirmed by the decreased MSQ scores after treatment. The improvement in aforementioned neurocognitive symptoms was confirmed with a NPA post-treatment that showed improvements in mental slowness, divided attention, and (working) memory. With regard to the information processing of verbal information (Stroop I and II), these did not improve. This can be explained by JH’s slurred speech and stuttering during conversations, which were still present post-treatment. Also, inhibition remained impaired at T4. With regard to the ROM, physical symptoms decreased after TPM. JH started to regain her life and was better able to participate in daily life activities compared to pre-treatment.

### **2.8. Evaluation of treatment and diagnosis**

JH told us during intake that she felt mistreated and misunderstood by medical professionals she had seen before during the assessment at the neurological clinic (with negative results on, for instance, the CT scan and MRI) and by the conclusion of the neurologist that her symptoms could be explained as psychogenic. Prior to the start of the neuropsychological treatment, the NPA results gave the clinician the opportunity to discuss neurocognitive impairment and thus confirm JH’s subjective neurocognitive complaints. Later on, in the first stage of the neuropsychological treatment, the present neurocognitive impairment without organic substrate gave the clinician the opportunity to discuss the negative results of the MRI in relation to the objectified neurocognitive impairment. We provided JH an explanation stating that since the MRI confirmed no organic substrate, yet the NPA had confirmed impairment was present, these neurocognitive functions could be used in treatment to retrain these cognitive brain functions and ultimately decrease the neurocognitive impairment she suffered from. This explanation also opened up the opportunity to discuss the diagnosis of CD. As a result, JH felt understood and taken seriously, as she explained afterward. Within our shared decision-making process, we then offered her the opportunity to see a psychologist for cognitive behavioral therapy (CBT) aimed at fatigue since she still reported suffering from fatigue.

## **3. Discussion**

### **3.1. Key results**

JH suffered from CD with mixed symptoms, including many physical symptoms (such as pain). She also suffered from mental slowness and impaired working memory and attention at a symptomatic level. These symptoms led to inactivity and she had great difficulty functioning in daily life. Furthermore, she experienced memory problems and fatigue after (mental) effort that severely interfered with her social life, for instance, when visiting friends. After TPM, she reported being able to participate in daily life and her physical symptoms, as well as neurocognitive impairment, improved. The CD went into remission.

### 3.2. Additional results

The use of a NPA enabled the clinicians to explain neurocognitive impairment without organic cause to a patient that has felt misunderstood for three years. The combination of the negative results on prior neurological assessments and the results of the NPA allowed the clinician to provide the patient with a framework and explanation for her symptoms. It also enabled the clinician to provide an outline of therapy in which rehabilitation of brain function was the central feature. In this way, the patient felt understood, leading to increased self-confidence and commitment. It also contributed to an improved relationship with the clinicians at our clinic, enabling them to discuss the diagnosis of CD.

### 3.3. Comparison to the literature

This is the first case report describing treatment of mental slowness in CD using TPM. Not only neurocognitive impairment but also somatic symptoms improved, which suggests an association between neurocognitive impairment, feeling ill, and the experience of somatic symptoms, stress, or pain in CD. A NPA can serve as a confirmation of these subjective neurocognitive symptoms. Therefore, clinicians are advised to use NPA to assess these symptoms in patients with CD. Furthermore, these neurocognitive impairments can serve as a framework for treatment. This opens an alternative avenue for treatment of CD that may be very welcome, especially because few successful treatment options are known for CD (Kroenke, 2007).

When memory problems are present, CRT designed for memory problems can be used to overcome these symptoms in the context of a neuropsychological treatment (Hendriks, Kessels, Gorissen, Schmand, & Duits, 2014; Ponds, van Heugten, Fasotti, & Wekking, 2010). One can argue that CBT, the treatment of choice (Kroenke, 2007), can be negatively influenced by such neurocognitive problems. In particular, memory problems that are followed by impairment in information processing speed might interfere with therapy since these patients might forget appointments and homework. In this way, memory problems interfere with CBT and the adaptation of a cognitive strategy to overcome these impairments can be of added value before and during CBT sessions. Furthermore, one can argue that through using TPM, the planning capacity of patients is also improved since the cognitive strategy incorporates a restructuring of actions (see Table 3) in situations where mental speed is necessary. If this is the case, planning problems may also play a role in the pathogenesis of CD. In this way, the need to explore and implement personalized types of treatments has been stressed (Schumann et al., 2014; Van der Feltz-Cornelis, Van Os, et al., 2014). This case report shows that a tailor-made treatment can be selected based on a specific patient's neurocognitive profile, even if this is not based on brain injury but on psychological causes, as with CD, and that such a tailor-made treatment for CD can be beneficial.

### 3.4. Strengths and limitations

This is the first study exploring the use of TPM in patients with CD, and this innovative approach is a strength of the study. Another strength of this study is the inclusion of a malingering test in the NPA. Since the results of this test did not indicate malingering, it can be stated that cognition does appear to be genuinely impaired, despite the lack of organic substrate. The use of ROM is also a strength of this study, since it enabled us to monitor physical symptoms during the course of treatment. It showed us that physical symptomatology decreased as treatment continued, something which JH affirmed during the sessions. The use of the MSQ before and after treatment is also a strength of this study, as this enabled us to explore the consequences of mental slowness in daily life (rather than only in idealized circumstances, as is the case with the NPA) (Winkens, van Heugten, Fasotti, & Wade, 2011) and to assess the treatment effects at T4 in an objective manner. Finally, the use of a NPA after treatment allows us to confirm improvement of JH's subjective neurocognitive symptoms and therefore can be considered as a strength of this study as well.

One limitation of this study is the use of the same neuropsychological tests at T4 and T0. Although parallel versions of neuropsychological tests were used as much as possible, the neuropsychological measures at T4 might be influenced by practice effects, since JH was already familiar with some of the test structures used at T0. Furthermore, the NPA at T4 did not include parallel tests related to planning, so conclusions about improved planning capacities could not be drawn.

Since this is a single case report study, caution should also be exercised with regard to the conclusion of this study. The improvement might have reflected the natural course of her condition; however, this seems improbable, as the patient had suffered from her condition for three years when she visited our center, despite previous treatments. Also, as should be obvious, the positive results of a case study cannot be considered generalizable per se. Nevertheless, this study does provide clinically relevant outcomes that warrant further research.

Although our patient's fatigue and cognitive impairment could have been related to myalgic encephalomyelitis (Carruthers et al., 2011), our patient did not fulfill the other criteria for this condition. Therefore, we can conclude with certainty that our patient had a CD. One might argue that our patient suffered from neural inflammation, apoptosis, or other related issues consistent with brain infection of some types, but since our patient improved during treatment these options do not apply. Therefore, we had no rationale for including additional tests such as electroencephalogram or magnetoencephalography.

### 3.5. Implications for research

Future studies should focus on adapting CRT for treatment of a specific neurocognitive symptom in patients with CD, based on the results of a NPA. This enables clinicians to tailor a treatment based on patient profiles which can maximize improvement (Laatsch & Stress, 2000). Also, future studies should explore the efficacy of tailor-made CRTs in patients with CD compared to usual treatments (medication and/or cognitive behavioral therapy for instance) in RCTs. Researchers should include parallel versions for each neurocognitive domain that is tested before and after treatment. This allows researchers to draw conclusions for all domains and not just a selection of domains.

The etiology of CD remains unclear. Neuroimaging studies might shed new light on the origin of CD. The results of this case study show that impairment in neurocognitive functioning improves after treatment, which suggest alterations on a neurobiological level and altered brain activity after treatment. Brain alterations have been found by several studies (de Lange, Toni, & Roelofs, 2010; Ejareh dar & Kanaan, 2016; Feinstein, 2011; Voon et al., 2010; Vuilleumier, 2005). Although these studies are diverse in terms of methodology, they are included relatively small samples and showed different results a recent review concludes that the majority of these studies showed abnormalities in emotion processing and emotion-motor processing (Ejareh dar & Kanaan, 2016). Although in its infancy, fMRI studies provide insight into the etiology of CD and allow the construction of a model for CD. Future studies should therefore include pre-treatment fMRI, pre-treatment NPA, followed by tailor-made CRT, and post-treatment NPA and fMRI. In this way, a theoretical model for CD can be explored and the effectiveness of CRT in CD patients can be determined.

## 4. Conclusion

This case report describes a patient with CD with mixed symptoms, mental slowness and other neurocognitive symptoms as well as inactivity. After CRT for mental slowness, the severity of both neurocognitive impairment and the somatic symptoms improved, the CD dissolved, and the patient could resume her daily activities. This suggests that CRT can be a treatment option for CD with neurocognitive symptoms. Further research into the efficacy of tailor-made CRTs by RCTs in patients with CD should be conducted. Additionally, fMRI studies should be continued to explore the role of brain activity in improved neurocognitive functioning in CD.

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### Competing Interests

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