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Social cognition in cervical dystonia: A case-control study

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ABSTRACT

Background: Although considered a motor disorder, adult onset isolated focal dystonia has many non-motor symptoms. There is a paucity of neuropsychological research on cognitive processing in adult onset focal dystonia.
Methods: We employed a battery of clinical and cognitive assessments, including basic and complex social cognition, and assessed 46 patients with adult-onset cervical dystonia, compared to 46 age-, sex–, education-, and premorbid IQ-matched healthy controls.
Results: Significant between-group differences were observed in relation to measures of memory encoding, recall and recognition, as well as multimodal measures of basic Social Cognition (emotion recognition: face and prosody), but not complex Social Cognition (mentalising). There were no deficits observed in multimodal measures of executive function. Controlling for mood did not affect performance.
Conclusion: In this multi-dimensional assessment of cognition in cervical dystonia, we report deficits in memory encoding, and in social cognition. Further investigation of social cognitive processes, memory, and sustained attention are required. Longitudinal studies are also needed to further delineate the role of psychological distress on cognitive outcomes and document the cognitive profile over time.

1. Introduction

Cervical Dystonia (CD) is the most common adult onset idiopathic isolated focal dystonia (AOIFD) [1]. Cervical dystonia is a hyperkinetic movement disorder characterized by irregular, involuntary, spasmodic neck movements and postures, with or without head tremor [2]. There is growing evidence that the basal ganglia, specifically cortico-striatal-thalamo-cortical networks, play a role in the clinical presentation of dystonia, with non-motor brain regions negatively implicated in CD [3], which may result in non-motor symptoms. In AOFID, GABAergic mechanisms are affected at all levels of the central nervous system [4], specifically in basolateral regions of the amygdala [5]. While traditionally considered a 'pure motor' disorder, patients with CD have non-motor features such as anxiety and depression (40–60%) [6], abnormal sensory processing [7], sleep difficulties, and cognitive deficits [8]. Recently, it has been shown that people with CD experience increased levels of perceived stigmatisation [9], neuropsychiatric comorbidities [10], and a range of non-motor symptoms recently summarised [11]. Temporal discrimination deficits in CD would suggest dysfunctional subcortical mechanisms for covert orienting of attention, specifically that involving salient environmental sensory stimuli, through the superior colliculus [12]. The superior colliculus is also involved in visual processing of emotional facial recognition [13] with anterior signalling through the pulvinar to the amygdala [14]. As a result, some individuals with CD are postulated to have impaired salient emotional face processing due to collicular-pulvinar-amygdala pathway dysfunction.

1.1. Cognitive dysfunction in AOIFD

There is a paucity of cognitive assessment research in AOIFD, especially in relation to CD; nine patients with cranial dystonia were reported to have sustained attention deficits compared to matched controls, despite intact global intelligence [15]. Similarly, a study of people with primary dystonia (n = 10; torticollis [50%]; arm dystonia [20%]; and generalised [30%]) and matched controls (n = 12) compared outcomes on a battery of measures, with only categorical fluency being impaired in patients [16]. In

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contrast, Balas and colleagues [17] found better performance on semantic fluency when comparing 20 symptomatic DYT1 dystonia patients to a healthy cohort ($n = 20$). In terms of impairment, however, they did find poorer performance on a verbal memory task (list). In a study by Scott and colleagues, attention and executive deficits were reported, though their findings might be confounded by concomitant dopaminergic and anti-cholinergic medication use by the participants [18]. Alemán and colleagues reported, in 20 patients with blepharospasm, deficits on measures of attention and a decreased capacity of performing complex motor tasks compared to matched controls, independent from depression, anxiety, and premorbid intelligence [19]. When compared to healthy controls, a heterogeneous group of 45 patients with various phenotypes of AOIFD performed worse on measures of executive function i.e., set maintenance and set shifting, such as the Wisconsin Card Sorting Task [20], similar to the findings of Lange and colleagues [21]. In a cohort of 38 patients with cervical and generalised dystonia, 7 of 12 impaired patients had CD, and were impaired on measures of speed and attentional shifting [22], with the authors controlling for medication, disease severity, and onset as co-variates, suggesting that cognition was independent of medication effects. A larger study of 45 people with primary adult-onset dystonia (16 B.P. 15CD; 14 segmental dystonia) found working memory deficits, impaired mental control, visual memory (visual reproduction task), processing speed, and set-shifting [23].

1.2. Social cognition in cervical dystonia

Social cognition incorporates the ability to represent and attribute affective and cognitive mental states to others, non-humans, and/or non-living things [24]. It integrates cognitive processes such as the ability to follow eye-gaze, share attention, recognize emotion, to distinguish between self and others' intentions [25], and to construct and judge a social narrative. Some elements of social cognition have been examined in patients with AOIFD. In 26 non-depressed people with cervical dystonia, significant impairments were reported on a measure of cognitive theory of mind, the Social Faux Pas Recognition Test, when compared to controls [26]. Two studies have examined emotion recognition in AOIFD; using visual stimuli, 32 patients (20 with cervical dystonia and 12 with blepharospasm) had difficulty identifying disgust, compared to age-matched controls [27]. Using auditory stimuli, significant deficits were also found in the recognition of angrily intonated words in 30 patients with cervical dystonia when compared to controls [28].

The literature on cognitive function in CD specifically, is scarce. Limited research suggests that patients with CD, specifically, may have deficits in attention and executive function, as well as social cognition. While aspects of cognitive deficits may relate to illness-specific features or mood, cognitive dysfunction may also represent a core feature of CD.

1.3. Study objective

The objective of this study was to assess the cognitive profile of CD patients in comparison to matched healthy controls. Based on the literature, we hypothesised that people with CD would perform less well than healthy controls on measures of social cognition, attentional-based tasks, and measures of executive function.

2. Methods

2.1. Study Population

Patients with adult-onset CD, satisfying standard diagnostic criteria [1], were approached consecutively and invited to take part at the botulinum toxin injection clinic at the host institution, with an uptake of approximately 90%. Following informed consent, participants were recruited and assessed from September 2018 to April 2019. Exclusion criteria included other neurological disorders; comorbidities precluding completion of questionnaires or cognitive assessments i.e., physical, psychiatric disorders, or substance misuse disorders. All participants were native English speakers.

2.2. Healthy Controls (HC)

HCs were recruited from a network of volunteers as part of a separate study protocol. HCs were matched to CD patients by age, sex, years of education, and estimated premorbid intelligence. In line with the patient exclusion criteria, healthy controls were excluded if they presented with a neurological disorder; comorbidities precluding completion of questionnaires or cognitive assessments i.e., physical, psychiatric disorders, or substance misuse disorders, and/or were not native English speakers.

2.3. Clinical and cognitive measures

Demographic data were collected from all participants. For patients, disease severity, disability, and pain were measured by clinical neurologists using the Toronto Western Spasmodic Torticollis Scale-2 (TWSTRS-2) [29]. All data were collected during clinic visits, which lasted approximately two-hours.

2.4. Cognitive measures

Predicted premorbid full scale intelligence (pFSIQ), on which groups were matched, was estimated using the Test of Premorbid Function-UK Edition (TOPF-UK) [30]. Mood was assessed using the Hospital Anxiety and Depression Scale (HADS), which provides an Anxiety subscale, Depression subscale, and Total (combined Anxiety and Depression) score [31]. The subscales are categorically scored as normal (0–7), borderline (8–10), and clinically elevated ($11-21$). The neuropsychological battery employed a range of standardised cognitive instruments, focusing on executive function, memory, and social cognition predominantly, as reported in Supplementary Table 1 [32–39].

2.5. Cognitive composites

In line with standard practice, composite scores were created a priori for cognitive processes i.e., Encoding, Recall, and Recognition and cognitive domains e.g., basic/complex Social Cognition, to reduce the likelihood of making Type 1 error through multiple comparisons on individual tests. Supplementary Table 1 reports the tests used to create the composite, as each measure was exclusive to a single composite. Composite scores showed satisfactory internal consistency ($\alpha > 0.70$). In line with the literature on emotional processing and social cognition in CD [40], our composites included *basic social cognition*, and *complex social cognition*, rather than a single *Social Cognition* composite.

2.6. Analysis

Means, standard deviation, and percentages are reported for continuous and categorical variables. Comparisons were made using χ^2 test for categorical variables and multiple analysis of variance (MANOVA) for continuous variables. The HADS total score was used as a co-variate to control for the effect of mood on cognitive outcomes using MANCOVA. Pearson correlations were conducted to investigate the relationship between clinical variables and cognitive composites, with pairwise exclusion. To determine an appropriate sample size, an a priori power analysis was conducted with G*Power 3.1 [41], indicating a minimum sample size of 26 cases per group (control group and patients group) would be required. Multiple comparisons were controlled for using Holm-Bonferroni sequential deletion. IBM SPSS Version 26 (SPSS Inc.) was used.

2.7. Ethical approval

The study was reviewed and approved by the St Vincent's Healthcare Group Ethics and Medical Research Committee. All participants gave written informed consent prior to their participation in the study.

3. Results

3.1. Demographics

A total of 46 people with cervical dystonia (CD) and 46 healthy controls (HCs) participants were enrolled. The groups were matched in age (CD: 58.79 ± 10.37 years; HC: 59.86 ± 5.82, $p = 0.543$), years of education (16.25 ± 3.70 years and 15.56 ± 3.90, respectively; $p = 0.395$), and estimated premorbid IQ (z = −0.01 ± 0.98, $p = 0.944$). There was a similar sex distribution between groups (CD: 67% women; $n = 31$; HC: 69% women; $n = 32$).

3.2. Cervical dystonia patients

Average age of CD onset was 41.3 ± 11.2, with an average illness duration of 17 years. The mean TWSTRS-2 Severity scale score was 10.52 (±4.40), TWSTRS-2 Disability was 5.43 (±3.77) and TWSTRS-2 Pain was 11.20 (±8.07).

In relation to the Anxiety, 41% were in the normal range; 15% borderline; and 26% were above cut-off for clinical caseness. Considering Depression, 65% were within the normal range; 10% considered borderline; with 6% above clinical cut-off. Each participant with borderline depression had borderline anxiety; the cohort with clinically elevated depression had comorbid clinically abnormal anxiety. Of the total cohort, 26% ($n = 12$) were actively engaged in treatment for psychological distress at the time of cognitive assessment.

3.3. Between-group comparisons

3.3.1. Anxiety and depression

CD patients reported significantly higher HADS-Anxiety (CD: 7.89 ± 4.48 v HC: 4.22 ± 2.81; $p < 0.001$), HADS-Depression (CD: 4.60 ± 3.66 v HC: 2.81 ± 2.57; $p = 0.033$) and HADS-Total (CD: 12.5 ± 7.56 v HC: 7.03 ± 5.12, $p = 0.002$).

3.3.2. Cognitive composite and task comparisons

CD patients were compared to controls on the a priori cognitive composites, reported in Table 1.

In the Executive Composite, there was no significant between-group difference ($p = 0.647$). There were no significant differences on individual measures: Fluency: $p = 0.757$; Forward Digit Span: $p = 0.750$; Reverse Digit Span: $p = 0.065$; Stroop Trial 3: $p = 0.501$. There was no significant difference on the Speed composite ($p = 0.539$), which considered Trial 1 (Colour Naming: $p = 0.614$) and Trial 2 (Word Reading: $p = 0.549$) of the Stroop test.

No between group difference was found on the Complex Social Cognition composite ($p = 0.407$) which included the Reading the Mind in the Eyes test ($p = 0.403$), Conflicting Emotional Prosody ($p = 0.171$), and cross-modal matching of emotional faces to emotional prosody ($p = 0.892$). However, there was a significant difference in the Basic Social Cognition composite ($p = 0.007$); performance on both the *Name Facial Affect* (Picture), and *Name Emotional Prosody* (Audio) tasks were significantly lower for CD participants than HC ($p = 0.033$; $p = 0.045$, respectively). A detailed breakdown of between-group comparisons per test can be seen in Table 2.

Each individual memory composite i.e., Encoding ($p = 0.028$), Recall ($p < 0.001$), and Recognition ($p = 0.006$), was significantly different between CD patients and controls. The Encoding composite contained the RAVLT Total Score ($p = 0.926$), Logical Memory I ($p = 0.038$), and the Immediate production of the Complex Figure Test ($p < 0.001$). Of note, performance on the Complex Figure Copy trial, a measure of visual-spatial constructional abilities was also significantly lower in CD patients than controls. A MANCOVA using the HADS total as a covariate did not affect performance on the encoding composite. The Recall Composite contained the RAVLT Delayed Recall ($p = 0.509$), Logical Memory II ($p < 0.001$), and the Complex Figure Recall ($p < 0.001$). The Recognition composite contained the recognition paradigms from the RAVLT ($p = 0.007$), Logical Memory ($p = 0.01$), and Complex Figure ($p = 0.001$). Considering Retention percentages of recalled information compared to encoded material, no significant difference was observed on the RAVLT ($p = 0.151$) or on the Complex Figure ($p = 0.878$). The Logical Memory percentage retention was significantly lower than the control cohort ($p < 0.001$).

3.4. Correlates: cognitive composites and clinical measures

3.4.1. Clinical measures

In CD patients, there were no significant correlations between performance on the Copy Trial of the Complex Figure Test and the TWSTRS-2 Pain ($r = 0.141$; $p = 0.367$), Severity ($r = -0.188$; $p = 0.226$), or Disability ($r = 0.141$; $p = 0.367$) sub-scales. None of the cognitive composite scores correlated with the TWSTRS-2 subscales, however, the TWSTR-Disability correlated with the HADS-D ($r = 0.397$; $p < 0.05$).

3.4.2. Cognitive composites

As reported in Table 3, the Encoding composite significantly correlated with the Recall Composite ($r = 0.858$; $p < 0.001$), the Executive composite ($r = 0.622$; $p < 0.001$), Speed Composite ($r = 0.365$; $p < 0.05$), and Complex Social Cognition ($r = 0.417$; $p < 0.05$). The Recall composite correlated with the Recognition Composite ($r = 0.433$; $p < 0.001$), the Executive composite ($r = 0.573$; $p < 0.001$), and Basic Social Cognition ($r = 0.367$; $p < 0.05$). The Executive composite correlated with the Speed Composite ($r = 0.539$; $p < 0.001$), Basic Social Cognition ($r = 0.411$; $p < 0.05$), and Complex Social Cognition ($r = 0.429$; $p < 0.05$). The Speed Composite correlated with Basic Social Cognition ($r = 0.351$; $p < 0.05$), and Complex Social Cognition ($r = 0.372$; $p < 0.05$). Basic Social Cognition and Complex Social Cognition correlated ($r = 0.558$; $p < 0.001$).

4. Discussion

In this study we investigated the cognitive profile of 46 people with cervical dystonia (CD), compared to 46 healthy controls matched by age,

Table 1
mean and standard deviation of cognitive composite z-scores and between group comparisons.

Composite Scores		z-Score	*p*-Value
Encoding	RAVLT Total; Logical Memory Encoding; ROCFT Immediate.	−0.40 ± 0.86	**0.028***
Delayed Recall	RAVLT Delay; Logical Memory Delay; ROCFT Delay.	−0.58 ± 0.79	**≤0.001*****
Recognition	RAVLT, Logical Memory, and ROCFT Recognition Paradigms.	−0.44 ± 0.80	**0.006****
Executive Function	Lexical Fluency; Digit Span (Forward and Reverse); Stroop Trial 3	−0.08 ± 0.70	0.647
Speed	Stroop Trial 1 (Colour Naming); Stroop Trial 2 (Word Reading)	−0.14 ± 1.31	0.539
Basic Social Cognition	Naming Facial Affect; Naming Emotional Prosody	−0.57 ± 0.77	**0.007****
Complex Social Cognition	RMET; Conflicting Emotional Prosody; Matching Face to Prosody.	−07. ±0.74	0.407

Values in **bold** highlight significant results between CD and healthy controls. RAVLT: Rey Auditory Verbal Learning Task; ROCFT: Rey-Osterrieth Complex Figure Test; RMET: Reading the Mind in the Eyes Test.

* <0.05.
** ≤0.01.
*** ≤0.001.

Table 2
z-scores of patient performance and between-group comparison for individual test items.

Cognitive domain	Cognitive measure	Subdomain/score	z-Score	p-Value
Memory	RAVLT	A1	−0.03 ± 0.98	0.834
		A5	0.05 ± 0.94	0.793
		Total Encoding	0.05 ± 1.14	0.926
		A6	0.03 ± 0.81	0.855
		Delayed Recall	0.17 ± 0.70	0.509
		Recognition	0.48 ± 0.74	**0.007****
	Logical Memory	Encoding	−0.43 ± 1.16	**0.038***
		Delayed Recall	−0.87 ± 0.99	**<0.001*****
		Recognition	−0.50 ± 1.02	**0.01****
	ROCFT	Immediate Recall	−0.85 ± 1.34	**≤0.001*****
		Delayed Recall	−0.99 ± 1.36	**<0.001*****
		Recognition	−1.47 ± 2.39	**≤0.001*****
Visuo-spatial	ROCFT	Copy Figure	−1.18 ± 2.24	**0.001**
Social cognition	RMET	Total Score	−0.10 ± 1.40	**0.403**
	Florida Affect Battery	Name Face Affect	−0.53 ± 1.01	**0.033***
		Name Prosody Affect	−0.61 ± 1.07	**0.045***
		Conflicting Prosody (Total: $i = 36$)	−0.32 ± 1.13	0.171
		Subscale: Conflicting ($i = 12$)	−0.18 ± 0.83	0.394
		Subscale: Inconsistent ($i = 12$)	−0.20 ± 0.99	0.364
		Subscale: Congruent ($i = 12$)	−0.21 ± 0.91	0.335
		Matching Face to Prosody	−0.01 ± 0.95	0.892
Executive function	Lexical Fluency	Total Score (F, A, S)	−0.14 ± 0.96	0.757
	Digit Span	Forward	0.04 ± 1.09	0.750
		Reverse	−0.38 ± 1.06	0.065
	Stroop CWI Test	Stroop – 3: Inhibition	0.13 ± 0.94	0.501
Speed	Stroop CWI Test	Stroop – 1: Colour	−0.14 ± 1.57	0.614
	Stroop CWI Test	Stroop – 2: Word	−0.15 ± 1.37	0.549

Values in **bold** highlight significant results between CD and healthy controls. RAVLT: Rey Auditory Verbal Learning Task; A1; First time hearing the list of words; A5: final trial of encoding; A6: Proactive interference task; Stroop CWI: Colour Word Interference Test; ROCFT: Rey-Osterrieth Complex Figure Test; RMET: Reading the Mind in the Eyes Test.
* <0.05.
** ≤ 0.01.
*** ≤ 0.001.

sex, years of education, and estimated premorbid IQ on a detailed battery of neuropsychological measures. CD patients performed significantly worse on encoding, recall, and recognition aspects of memory, as well as in basic social cognition (naming facial affect, and naming emotional prosody). CD patients did not perform lower than controls on measures of speed, executive function, or complex social cognition. CD patients reported significantly higher levels of psychological distress through the HADS, though controlling for this did not affect between-group performance.

The lower performance on the encoding composite is likely to have reduced the potential volume of information which could be recalled or recognised after the standardised delay. We computed retention percentages and found that, relative to what was encoded, patients did not differ to controls on the list learning (RAVLT), or the Complex Figure (RCFT). There was, however, a significant difference on the standardised story. Patients with CD were not significantly impaired when compared to controls on the list learning task, nor were they impaired on the digit span forward (attention) or reverse digit span (working memory). This deficit is likely to reflect shallow levels of encoding due to large quantities of novel material, as CD patients were impaired on the encoding of the story, requiring sustained attention and orientation without the benefit of repetition, rehearsal, or an opportunity for recurrent exposure to the stimuli, as is the case on list learning (RAVLT Trials A1–A5). These data would suggest that patients had an inability to attend to the stimuli for a sufficient time to allow for semantic organisation or deeper encoding, which may be due to the cognitive load and temporal gradient of the story. This may also reflect the incidental learning required for the Complex Figure. This pattern of shallow encoding, for both auditory information as well as incidental visual learning, is likely to be why there were no significant deficits in

Table 3
Correlations of the cognitive composite scores, mood measure, and measure of severity for people with CD.

	Variable	1	2	3	4	5	6	7	8	9	10	11	12	13
1	TWSTRS Pain	1	–	–	–	–	–	–	–	–	–	–	–	–
2	TWSTRS Disability	**0.447****	1	–	–	–	–	–	–	–	–	–	–	–
3	TWSTRS Severity	0.164	0.151	1	–	–	–	–	–	–	–	–	–	–
4	Anxiety	0.265	0.213	0.006	1	–	–	–	–	–	–	–	–	–
5	Depression	0.320	**0.397***	−0.015	**0.723****	1	–	–	–	–	–	–	–	–
6	HADS Total	0.310	0.317	−0.004	**0.942****	**0.912****	1	–	–	–	–	–	–	–
7	Encoding	0.041	0.094	−0.088	−0.215	−0.096	−0.174	1	–	–	–	–	–	–
8	Recall	0.199	0.288	−0.135	−0.040	0.034	−0.007	**0.858****	1	–	–	–	–	–
9	Recognition	0.032	0.009	−0.004	−0.068	−0.071	−0.075	0.266	**0.433****	1	–	–	–	–
10	Executive	−0.116	0.044	−0.211	−0.240	−0.070	−0.175	**0.622****	**0.573****	0.149	1	–	–	–
11	Speed	0.065	0.064	−0.024	−0.185	−0.217	−0.215	**0.365***	0.297	−0.090	**0.539****	1	–	–
12	Basic Social Cognition	−0.060	0.081	−0.088	0.105	0.158	0.136	0.328	**0.367***	0.067	**0.411***	**0.351***	1	–
13	Complex Social Cognition	−0.241	−0.037	0.083	0.004	0.097	0.045	**0.417***	0.190	−0.155	**0.429***	**0.372***	**0.558****	1

Values in **bold** highlight significant correlations within CD cohort.
* Correlation is significant at the 0.05 level (2-tailed).
** Correlation is significant at the 0.01 level (2-tailed).

executive function found, as goal-orientated task-specific shorter tasks can be completed due to a relatively intact executive system. As CD patients performed significantly worse than controls when required to copy the complex figure, one could interpret this as either being due to executive dysfunction (planning, or organisation), or a deficit in visual attention (i.e., sustained attention to visual stimuli). To distinguish between attentional processes which may underlie the observed discrepancy, as Posner and colleagues describe [42–44], a deficit in alerting and orientating attention, rather than executive attention, may be present, hence the intact performance on executive tests. We propose that the pattern of deficits in the complex figure represents a visual analogue to the attentional deficits observed on the story encoding trial. Of note, controlling for elevated anxiety and depression did not account for this finding, suggesting that this performance pattern is independent of mood.

This study further provides evidence for impaired basic social cognition in people cervical dystonia i.e., emotion recognition. Our patient cohort performed significantly lower than controls when requested to correctly label affect (happy, sad, angry, fearful, or neutral) for both visual (face) and auditory (prosody) stimuli.

Studies show the bidirectional modulation of ventromedial prefrontal cortico-amygdala circuitry (whereby ventral amygdalo-fugal pathways projects anteriorly to the prefrontal cortex and anterior cingulate, [45], though our results may be better contextualised by a postulated focal dysfunction in the collicular-pulvinar-amygdala pathway. Many of the tasks linked to the former were intact i.e., mentalising, working memory, and executive functions etc. More focal dysfunction in the collicular-pulvinar-amygdala pathway may explain why neither measures of executive function nor complex social cognition tasks were significantly impaired in CD patients compared to controls. Notwithstanding, aspects of visual orientation, visual attention, automatic/unconscious visual and auditory orientation, and visual and auditory attention are evident on measures with a high temporal gradient i.e., large volumes of information in the story encoding, and high visual orientation and attention required for the incidental learning trial on the Complex Figure.

In essence, we hypothesise that disruption in this network (collicular-pulvinar-amygdala) may contextualise the cognitive findings in relation to differences in social cognitive performance on basic processing, and also encoding aspects of memory performance requiring sustained attention due to reduced cerebral GABA levels both in the superior colliculus and in the amygdala. We also suggest that this postulated collicular-pulvinar-amygdala dysfunction causes the other observable features of cervical dystonia i.e., abnormal temporal discrimination and a predisposition to anxiety and depression.

Our findings are congruent with previous reports of lower performance on measures of naming affect [27] and identification of emotional prosody [28], as well as elevated anxiety and low mood [6]. Considering tasks that specifically assess executive function, unlike other studies in the AOIFD literature [15,18–20], our CD patient cohort did not differ significantly from our healthy controls on measures of executive function.

A limitation of our study is that our test battery did not specifically include measures of sustained attention such as the Sustained Attention to Response Task (SART), N-Back test, or Attention Network Test (ANT), unlike Allam and colleagues [23], who found a deficit comparing patients to controls; nor did our test battery include a serial test of executive function, such as the Paced Auditory Serial Attention Task (PASAT). Our battery was also not comprehensive from a language perspective. Despite that, a strength of the battery is the large social cognitive component from both a basic (emotional recognition) and complex (mentalising and cross-modal integration) perspective, as well as many measures of executive function.

There are many avenues for future research following this study. A large-scale case-matched longitudinal study of people with CD could allow for greater interpretation of the interaction effects of disease duration on cognitive outcomes, as well as the natural history of the cognitive profile in CD. The subcortical pathway for emotional face recognition and attentional networks should be assessed in patients with cervical dystonia, using complementary ancillary methodologies i.e., functional and/or structural neuroimaging.

In conclusion, this study adds to the known literature in relation to basic emotional processing in patients with cervical dystonia, and finds that patients may also have impaired encoding, recall, and recognition capacity as well as impaired basic social cognition (naming facial affect and emotional prosody); further research and therapeutic intervention trials are warranted.

Supplementary data to this article can be found online at https://doi.org/10.1016/j.prdoa.2020.100072.

Declaration of competing interest

There are no other potential conflicts of interest for research relating to this article, for all authors.

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